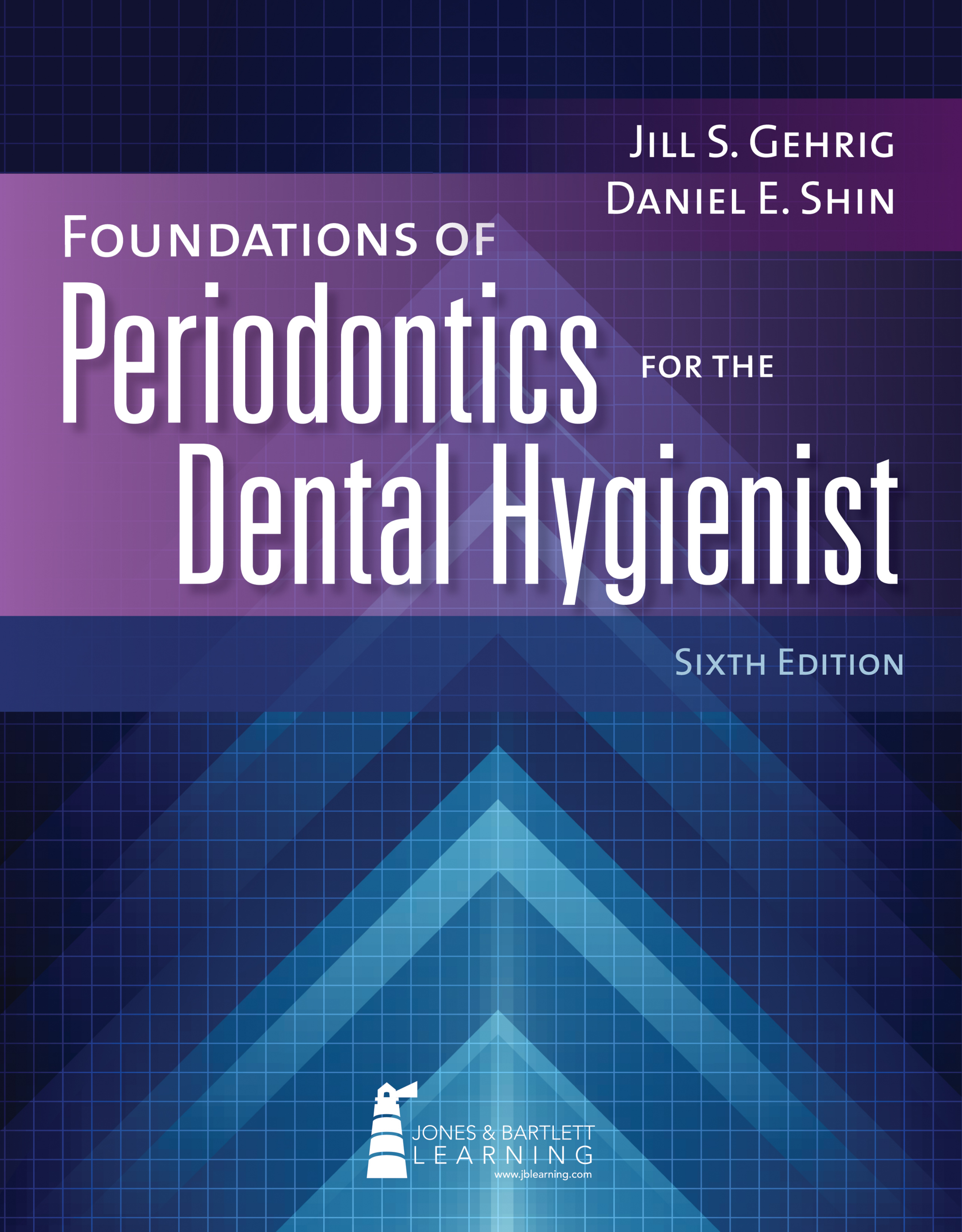

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DANIEL E. SHIN

FOUNDATIONS OF Periodontics FOR THE Dental Hygienist

SIXTH EDITION

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FOUNDATIONS OF Periodontics FOR THE Dental Hygienist

SIXTH EDITION

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Preface

This textbook endeavors to present an evidence-based discussion of periodontology based on information from recent research. Periodontology, however, is a rapidly changing science. The authors, editors, and publisher have made every effort to confirm the accuracy of the information presented and to describe generally accepted practices at the time of publication. However, as new information becomes available, changes in treatment may become necessary. The reader is encouraged to keep up with dental and medical research through the many peer-reviewed journals available to verify information found here and to determine the best treatment for each individual patient. The authors, contributors, editors, and publisher are not responsible for errors or omissions or for any consequences from application of the information in this book and make no warranty, express or implied, with respect to the contents of this publication.

The ancient Greek philosopher Heraclitus famously stated: "*There is nothing permanent except change.*" This quotation serves as the basis for the evolution of each successive edition of our textbook. And, while some may view any semblance of change with a sense of apprehension, the authors of the sixth edition of *Foundations of Periodontics for the Dental Hygienist* see this as an opportunity to grow upon previous editions so as to improve the preparation of students for productive functioning in the rapidly evolving and highly demanding environment of clinical periodontology.

The *Sixth Edition* is written with three primary goals in mind. First and foremost, this textbook focuses on the dental hygienist's role in periodontics. Our second goal is to develop a book with an instructional design that facilitates the teaching and learning of the complex subject of periodontics—as it relates to dental hygiene practice—without omitting salient concepts or "watering down" the material. Third, we hope to help guide you, the teacher, in transmitting your expertise and knowledge in periodontology to your students. To accomplish all our goals, many of the complex theoretical concepts that are covered in this textbook are reinforced with clinical photographs/radiographs, illustrations, flowcharts, and hypothetical case scenarios that, we hope, may provide your students with visualization aids that can assist in the learning process. Written primarily for dental hygiene students, *Foundations of Periodontics for the Dental Hygienist* also would be a valuable resource on current concepts in periodontics for the practicing dental hygienist or general dentist.

Content Sequencing

The textbook is divided into eight major content areas:

Part 1: The Periodontium in Health

Part 2: Diseases Affecting the Periodontium and Peri-Implant Tissues

Part 3: Etiologic Factors for Periodontal Diseases and Peri-Implant Diseases/Conditions

Part 4: Assessment and Planning for Patients with Periodontal Diseases and Peri-Implant Diseases/Conditions

Part 5: Implementation of Therapy for Patients with Periodontal Diseases and Peri-Implant Diseases/Conditions

Part 6: Other Aspects of the Management of Patients with Periodontal Diseases and Peri-Implant Diseases/Conditions

Part 7: Comprehensive Patient Cases

Part 8: Online Resources

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New to This Edition

The sixth edition of *Foundation of Periodontics for the Dental Hygienist* has been extensively updated.

- The content section “Diseases of the Periodontium and Peri-Implant Tissues” aligns with the 2017 AAP/EFP Classifications of Periodontal and Peri-Implant Diseases and Conditions in Chapters 3–9.
- The content section “Etiologic Factors for Periodontal Diseases and Peri-Implant Diseases/Conditions” has been updated to include Chapters 10–18.
- **Chapter 1** contains expanded content on the position of the gingival margin in health as related to age and history of previous periodontitis, as well as new content on the vascular supply to the periodontium.
- **Chapter 3** contains additional content on transseptal fibers and pathologic tooth migration, as well as new content on the changes in alveolar bone height in disease, patterns of bone loss, and osseous defects.
- **Chapter 4** contains new content in the category of periodontal health, as well as new content on intact periodontium versus reduced periodontium.
- **Chapter 5** is a NEW! chapter on periodontal health and gingival diseases and addresses the new 2017 AAP/EFP Classification category of periodontal health and gingival diseases/conditions.
- **Chapter 6** contains new content to align with the 2017 AAP/EFP Classification of Periodontal and Peri-Implant Diseases and Conditions.
- **Chapter 7** is a NEW! chapter focusing on mucogingival deformities around the teeth and aligns with the 2017 AAP/EFP Classification of Periodontal and Peri-implant Diseases and Conditions.
- **Chapter 8** contains new content on titanium implants versus zirconia implants, hard and soft tissue deficiencies, biomechanical overload, and the range of probing depths compatible with peri-implant health.
- **Chapter 9** contains new content on the implications of an untreated periodontal abscess, the differentiation of periodontal versus pulpal abscesses, the signs and symptoms of the periocoronal abscess, recurrent abscesses, endodontic–periodontal lesions, necrotizing periodontal diseases, and typical treatment steps of necrotizing periodontal diseases.
- **Chapter 10** contains new content on the balance between periodontal health and disease.
- **Chapter 11** contains new content on the transmission of biofilm bacteria.
- **Chapter 12** contains new content on innate and adaptive immunity, components of the immune system, and the subsets of T-lymphocytes.
- **Chapter 13** contains new content on the role the host immunoinflammatory response plays in the pathogenesis of periodontal disease, as well as new content on the bone remodeling cycle.
- **Chapter 14** and **Chapter 15** are NEW! chapters designed to assist students in understanding the effect of systemic disorders/conditions on oral health versus the effect of oral diseases on systemic health.
- **Chapter 16** contains new content on the detrimental impact contributing factors can have in the development and progression of periodontal disease.
- **Chapter 17** contains new content on tobacco and nicotine delivery systems, the biochemical basis underlying nicotine dependence and addiction, the impacts of combustible smoking and noncombustible tobacco on the oral microbial biofilms and periodontitis, smoking and bone metabolism, oral jewelry, cannabis as a risk factor for periodontal disease, and the effects of smoking on periodontal therapy.
- **Chapter 18** contains updated content on nutrition and the periodontium.
- **Chapter 19** contains a comparison of the terms *clinical attachment level* and *clinical attachment loss*.
- **Chapter 20** contains new content on the radiographic appearance of cortical and cancellous bone, as well as new content on the importance of accurate three-dimensional images (CBCT) for treatment planning for surgical placement of dental implants.
- **Chapter 26** contains a new section on the types of manual and power toothbrushes.
- **Chapter 27** contains new content on power-driven air floss.
- **Chapter 28** contains new content on the use of antibiotics, locally delivered controlled-released chemotherapeutic agents, non-controlled-release chemotherapeutic agents, and educating patients on the use of complementary and alternative dental products/remedies.
- **Chapter 29** contains new content on anticytokine medications, statins, probiotics, and specialized proresolution mediators.

- **Chapter 30** contains new content on flap reflection to provide better access for periodontal instrumentation of root surfaces, as well as updated information on the concepts of relative and absolute contraindications for periodontal surgery.
- **Chapter 33** contains new content on ultrasonic periodontal probes.
- **Chapter 37** is a NEW! chapter on oral malodor and xerostomia/hyposalivation.

Textbook Features

The sixth edition of *Foundations of Periodontics for the Dental Hygienist* has many features designed to facilitate learning and teaching.

1. **Chapter Overview and Outline.** Each chapter begins with a concise overview of the chapter content. The chapter outline makes it easier to locate material within the chapter. The outline provides the reader with an organizational framework with which to approach new material.
2. **Learning Objectives and Key Terms.** Learning objectives assist students in recognizing and studying important concepts in each chapter. Key terms are listed at the beginning of each chapter. One of the most challenging tasks for any student is learning a whole new dental vocabulary and gaining the confidence to use new terms with accuracy and ease. The "Key Terms" list assists students in this task by identifying important terminology and facilitating the study and review of terminology in each chapter. Terms are highlighted in bold type and clearly defined within the chapter.
3. **Instructional Design**
    - Each chapter is subdivided into sections to help the reader recognize major content areas.
    - Chapters are written in an expanded outline format that makes it easy for students to identify, learn, and review key concepts.
    - Material is presented in a manner that recognizes that students have different learning styles. Hundreds of illustrations and clinical photographs visually reinforce chapter content.
    - Chapter content is supplemented in a visual format with boxes, tables, and flow charts.
4. **Focus on Patients.** *Focus on Patients* items allow the reader to apply chapter content in the context of clinical practice. The cases provide opportunities for students to integrate knowledge into their clinical work. Three types of scenarios help students apply content to the real-world setting:
    - Clinical Patient Care scenarios
    - Evidence in Action scenarios
    - Ethical Dilemma scenarios
5. **Patient Case Studies**
    - Chapter 34 presents five hypothetical patient scenarios. Patient assessment data pertinent to the periodontium challenges the student to interpret and use the information in periodontal care planning for the patient.
    - Chapter 38 (an online chapter) provides radiographs for six cases. These cases are intended to give students the opportunity to develop their critical-thinking skills in analyzing and interpreting radiographs as it pertains to the hard tissues of the periodontium in health and disease.
6. **Glossary.** The glossary provides quick access to common periodontal terminology.
7. **Online Chapters**
    - Chapter 35: Periodontal Disease in the Pediatric Population
    - Chapter 36: Iatrosedation: Easing and Managing Pediatric Patient Fears
    - Chapter 37: Oral Malodor, Xerostomia, and Hyposalivation
    - Chapter 38: Patient Cases Radiographic Analysis
8. **Online Instructor and Student Resources**
    - Instructor Resources
        - Slides in PowerPoint format for each chapter
        - Lesson plans
        - Test bank
        - Answers to Chapter Review Exercises

  - Image bank
  - Discussion topics and classroom activities
- Student Resources
  - Animation: Anatomy of Periodontium in Disease
  - Chapter Review Questions
  - Learning Objectives

Foundations of Periodontics for the Dental Hygienist, Sixth Edition strives to present the complex subject of periodontics in a reader-friendly manner. The authors greatly appreciate the comments and suggestions from educators and students about previous editions of this book. It is our sincere hope that this textbook will assist students and practitioners alike to acquire knowledge that will serve as a foundation for the prevention and management of periodontal diseases.

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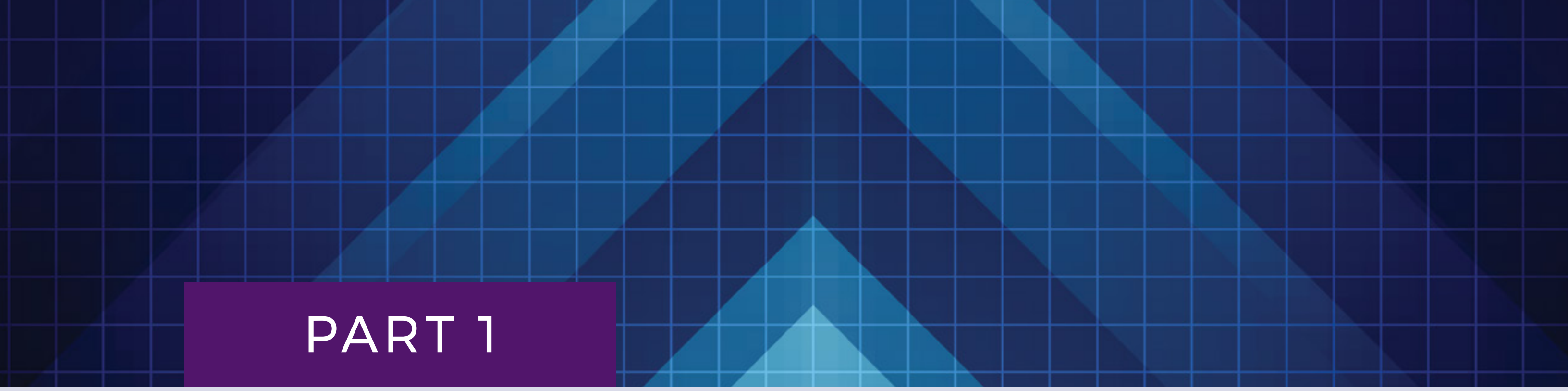

PART 1

The Periodontium in Health

CHAPTER 1 Periodontium: The Tooth Supporting Structures. 3

CHAPTER 2 Microscopic Anatomy of the Periodontium. 23

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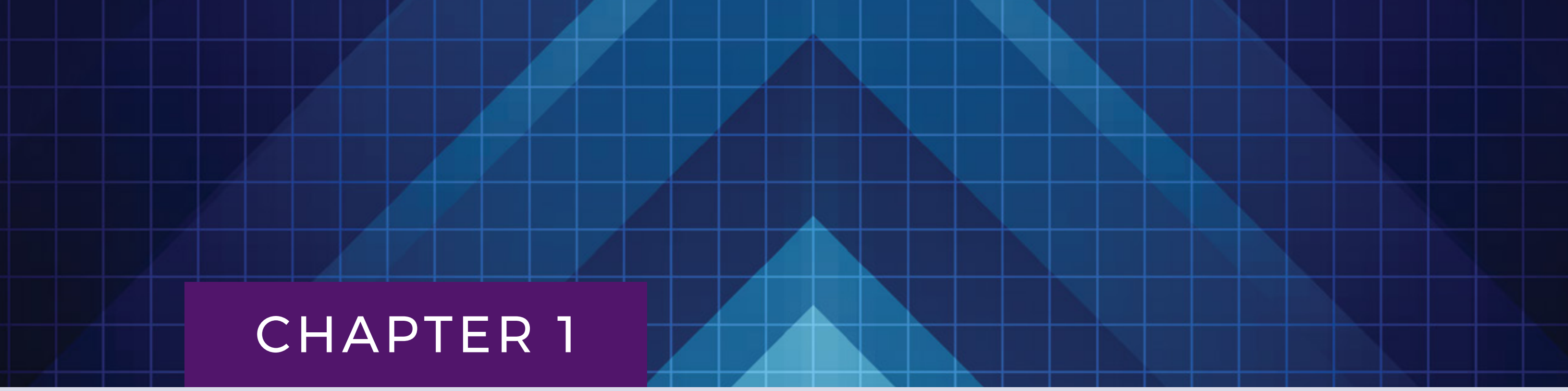

CHAPTER 1

Periodontium: The Tooth-Supporting Structures

- Section 1 Tissues of the Periodontium
  - The Gingiva
  - Periodontal Ligament
  - Root Cementum
  - Alveolar Bone
- Section 2 Nerve Supply, Blood Supply, and Lymphatic System
  - Nerve Supply to the Periodontium
  - Blood Supply to the Periodontium
  - Lymphatic System and the Periodontium
- Section 3 Focus on Patients
  - Clinical Patient Care

CLINICAL APPLICATION

Dental health care providers continuously interact with patients when making clinical decisions, performing clinical procedures, evaluating new techniques, and adapting to emerging treatment approaches. Nearly every action taken by a clinician requires a detailed knowledge of the anatomy of the tooth-supporting structures—the periodontium. Chapters 1 and 2 outline current knowledge about the anatomy of the periodontium. Chapter 1 deals with what is known about the fundamental structure of the complex system of tissues that support the teeth and can serve as a basis for organizing thoughts about additional anatomical information as it becomes available through additional research. Chapter 2 deals with the microscopic anatomy of these structures.

LEARNING OBJECTIVES

- Identify the tissues of the periodontium on an unlabeled diagram depicting the periodontium in cross section.
- Describe the function that each tissue serves in the periodontium, including the gingiva, periodontal ligament, cementum, and alveolar bone.
- In a clinical setting or on a color photograph, identify the following anatomical areas of the gingiva: free gingiva, gingival sulcus, interdental gingiva, and attached gingiva.
- In a clinical setting or on a color photograph, identify the following boundaries of the gingiva: gingival margin, free gingival groove, and mucogingival junction.
- In a clinical setting, identify the free gingiva on an anterior tooth by inserting a periodontal probe to the base of the sulcus.

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- In a clinical setting, compare and contrast the coral pink tissue of the attached gingiva with the darker, shiny tissue of the alveolar mucosa.
- In the clinical setting, use compressed air to detect the presence or absence of stippling of the attached gingiva.
- Identify the alveolar process (alveolar bone) on a human skull.
- Describe the shape and contour of the alveolar crest of the bone in health.
- Describe the nerve and blood supply to the periodontium.
- Explain the role of the lymphatic system in the health of the periodontium.

KEY TERMS

Periodontium
Gingiva
Periodontal ligament (PDL)
Cementum
Alveolar bone (alveolar process)
Gingival margin
Alveolar mucosa
Free gingival groove
Mucogingival junction
Free gingiva
Attached gingiva
Stippling
Interdental col
Interdental gingiva
Papillae
Gingival sulcus
Gingival crevicular fluid
Sharpey's fibers
Alveolar bone proper
Cribriform plate
Alveolus
Cortical bone
Alveolar crest
Cancellous bone (spongy bone)
Periosteum
Innervation
Trigeminal nerve
Anastomose
Lymphatic system
Lymph nodes

Section 1

Tissues of the Periodontium

The **periodontium** (*peri* = around and *odontos* = tooth) is the functional system of tissues that surrounds the teeth and attaches them to the jawbone (**Figures 1-1** and **1-2**). The periodontium is also called **the supporting tissues of the teeth** and the attachment apparatus. The tissues of the periodontium include the following:

1. **Gingiva**—the tissue that covers the cervical portions of the teeth and the alveolar processes of the jaws.
2. **Periodontal ligament (PDL)**—the fibers that surround the root of the tooth. These fibers attach to the bone of the socket on one side and to the cementum of the root on the other side.
3. **Cementum**—the thin layer of mineralized tissue that covers the root of the tooth.
4. **Alveolar bone** (also known as **alveolar process**)—the bone that surrounds the roots of the teeth. It forms the bony sockets that support and protect the roots of the teeth.

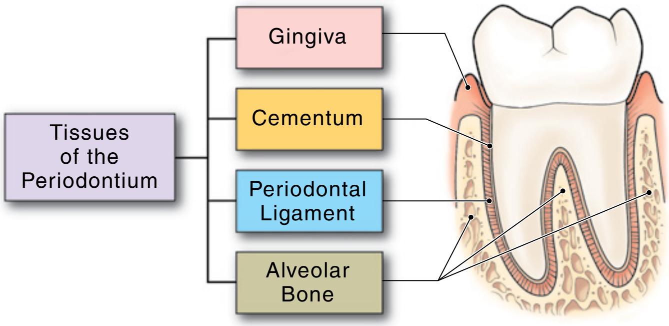

Figure 1-1 Tissues Comprising the Periodontium. A graphic representation of the periodontium in cross section.

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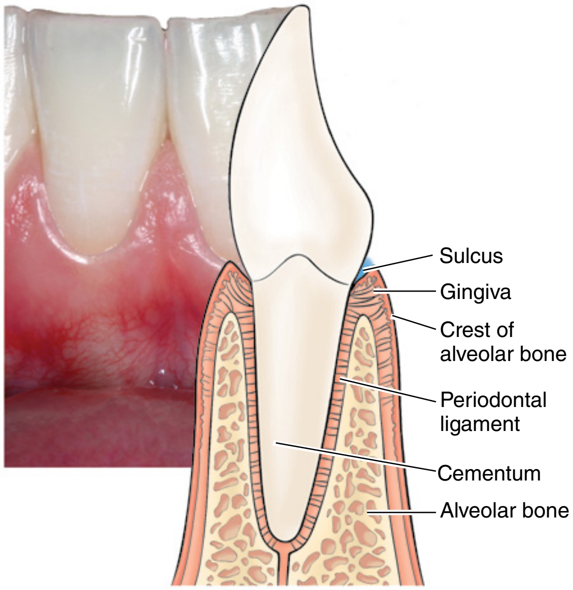

Figure 1-2 Healthy Periodontium. Clinical photograph and drawing depicting the structures of the periodontium.

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Each of the tissues of the periodontium plays a vital role in maintaining the health and function of the periodontium (**Table 1-1**). Knowledge of the periodontal tissues in health is a necessary foundation for understanding the concepts of (1) normal function of the periodontium, (2) disease prevention, and (3) the periodontal disease process.

Table 1-1 The Periodontium

Structure	Brief Description of Its Function
Gingiva	■ Provides a tissue seal around the cervical portion (neck) of the tooth ■ Covers the alveolar processes of the jaws ■ Holds the tissue against the tooth during mastication
Periodontal ligament	■ Suspends and maintains the tooth in its socket
Cementum	■ Anchors the ends of the periodontal ligament fibers to the tooth so that the tooth stays in its socket ■ Protects the dentin of the root
Alveolar bone	■ Surrounds and supports the roots of the tooth

© Jones & Bartlett Learning

Dental hygiene students usually are introduced to the tissues of the periodontium during the first semester or quarter of the dental hygiene curriculum. In the preclinical stages of the curriculum, mastering dental terminology and anatomy can sometimes be overwhelming and confusing. This chapter provides an opportunity to review this complex system of tissues known as the periodontium.

The Gingiva

1. **Overview of the Gingiva**
   a. **Description.** The gingiva is the part of the mucosa that surrounds the cervical portions of the teeth and covers the alveolar processes of the jaws (**Figure 1-3**).

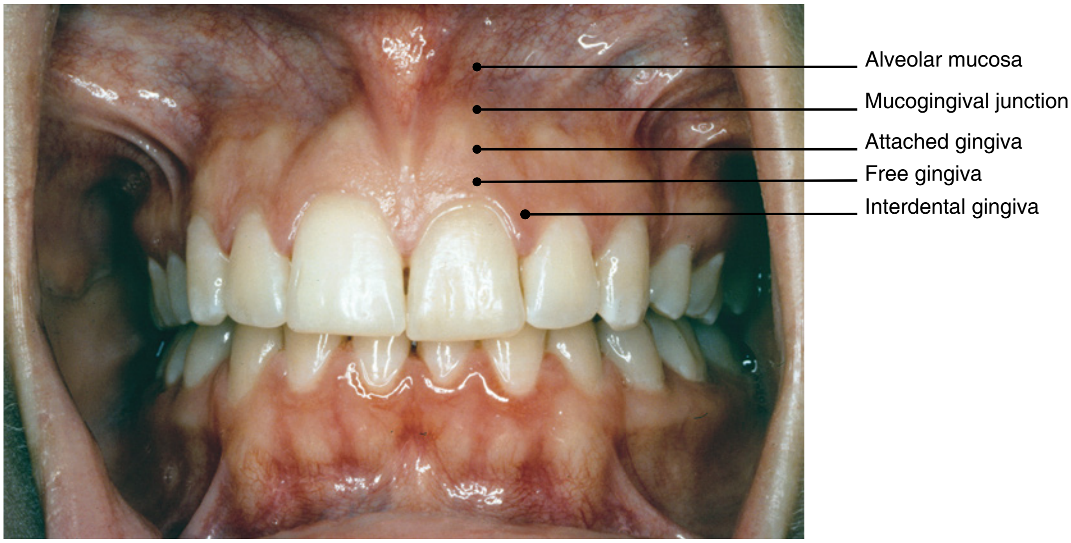
Alveolar mucosa
Mucogingival junction
Attached gingiva
Free gingiva
Interdental gingiva

Figure 1-3 **The Gingival Tissues.** Photograph of healthy gingival tissues showing the free, attached, and interdental gingiva.

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1. In health, the contour and shape of the gingival margin approximates the scalloped curvature of the cementoenamel junction (CEJ).
2. The position of the gingival margin in health may be age dependent. In a periodontally healthy young individual, the position of the gingival margin may be located at or slightly coronal to the CEJ of each tooth. In contrast, in a periodontally healthy older individual, the gingival margin may be positioned apical to the CEJ. Reasons for this may be attributed to a previous history of periodontitis (resulting in recession of the gingival margin) or due to non-periodontitis reasons (e.g., history of mechanical abrasion).
3. The gingiva attaches to the tooth by means of a specialized epithelial tissue (junctional epithelium).
4. The gingiva is composed of a thin outer layer of epithelium and an underlying layer of connective tissue.
5. The gingiva is divided into four anatomical areas:
   a. Free gingiva
   b. Gingival sulcus
   c. Interdental gingiva
   d. Attached gingiva

b. **Function.** The gingiva protects the underlying tooth-supporting structures of the periodontium from the oral environment. The oral environment is exposed to a wide range of temperatures in food and drink, mechanical forces, and many oral bacteria. To accomplish these functions, the gingiva has several defense mechanisms, including saliva and the immune system.

c. **Boundaries of the Gingiva**

1. The coronal boundary, or upper edge, of the gingiva is the **gingival margin** (**Figure 1-4**).

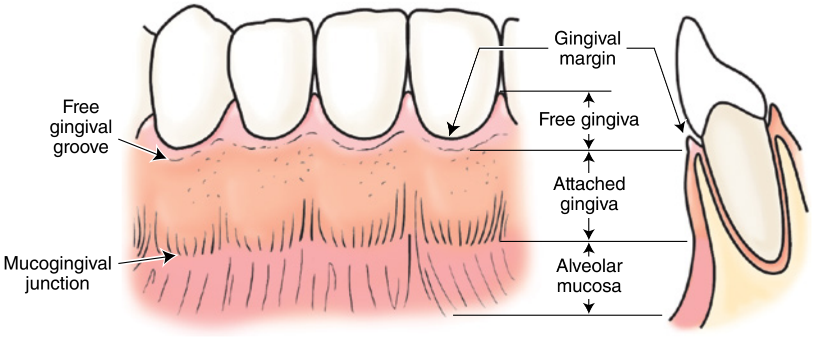
Gingival margin
Free gingival groove
Free gingiva
Attached gingiva
Mucogingival junction
Alveolar mucosa

Figure 1-4 **Boundaries of the Gingiva.** Illustration showing the boundaries and anatomical areas of the gingiva.

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        2. The apical boundary, or lower edge, of the gingiva is the alveolar mucosa. The **alveolar mucosa** can be distinguished easily from the gingiva by its dark red color and smooth, shiny surface.
    d. **Demarcations of the Gingiva**
        1. The **free gingival groove** is a shallow linear depression that separates the free and attached gingiva (this line is rarely visible to the naked eye).
        2. The **mucogingival junction** is the clinically visible boundary where the pink attached gingiva meets the red, shiny alveolar mucosa. (*Clinically visible* means that this landmark is readily seen by the naked eye.)

2. **Free Gingiva.** The **free gingiva** is the unattached portion of the gingiva that surrounds the tooth in the region of the CEJ. The free gingiva is also known as the *unattached gingiva* or the *marginal gingiva*.
    a. **Location of the Free Gingiva**
        1. The free gingiva is the most coronal boundary of the gingiva.
        2. It surrounds the tooth in a turtleneck or cuff-like manner.
        3. The free gingiva attaches to the tooth by means of a specialized epithelium, known as the junctional epithelium.
    b. **Characteristics of the Free Gingiva**
        1. The tissue of the free gingiva fits closely around the tooth, but is not directly attached to it.
        2. Because it is unattached, a periodontal probe can be used to slightly retract the free gingiva away from the tooth.
        3. The free gingiva also forms the soft tissue lateral wall of the gingival sulcus.
    c. **Contour of the Free Gingival Margin**
        1. The tissue of the free gingiva meets the tooth in a thin rounded edge called the gingival margin.
        2. In health, the gingival margin follows the scalloped, curved contours of the underlying CEJ.

3. **Attached Gingiva.** The **attached gingiva** is continuous with the free gingiva and is the part of the gingiva that is tightly bound to the underlying cementum on the cervical-third of the root and to the periosteum (connective tissue cover) of the alveolar bone.
    a. **Location of the Attached Gingiva.** The attached gingiva lies between the free gingiva and the alveolar mucosa (**Figure 1-5**).

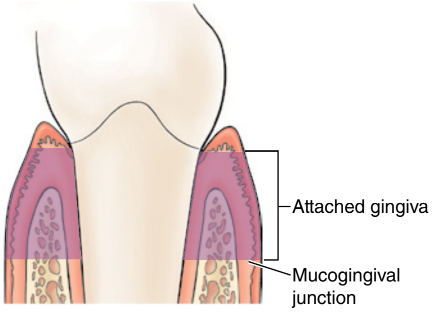

Figure 1-5 Location of the Attached Gingiva. The attached gingiva extends from the free gingival groove to the mucogingival junction.

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    b. **Width of the Attached Gingiva**
        1. The attached gingiva is widest in the incisor and molar regions, ranging from 3.3 to 3.9 mm on the mandible and 3.5 to 4.5 mm on the maxilla (**Figure 1-6**).

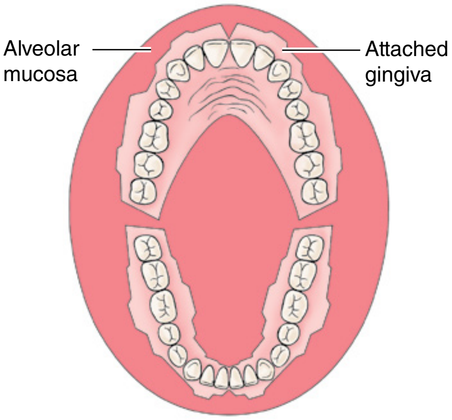

Figure 1-6 Mean Width of the Attached Gingiva. The attached gingiva is widest in the incisor and molar regions and narrowest in premolar regions.

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2. The attached gingiva is narrowest in premolar regions (1.8 mm on mandible and 1.9 mm on maxilla).
3. The width of the attached gingiva is not measured on the palate because clinically it is not possible to determine where the attached gingiva ends and the palatal mucosa begins (**Figure 1-7**).

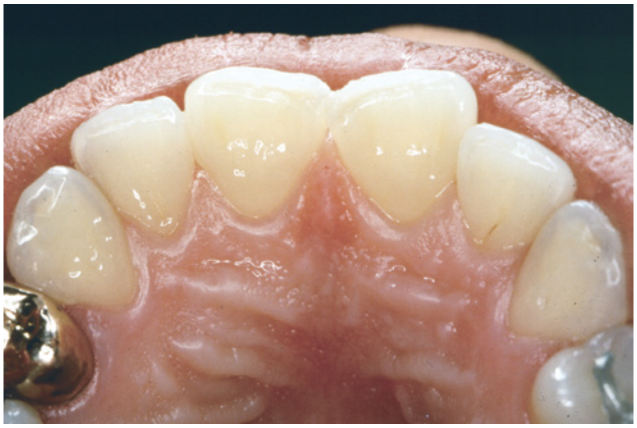

Figure 1-7 Gingival Tissue of the Palate. On the palate, the palatal gingiva is directly continuous with the keratinized masticatory mucosa.

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4. It was once believed that a minimum 2-mm width of keratinized gingiva (free gingiva plus attached gingiva) was necessary to maintain the health of the periodontium.[1] Currently, this concept is being revisited because the amount of keratinized tissue that is "necessary" has never been established.

c. **Color of the Attached Gingiva**
   1. In health, the attached gingiva is pale or light coral pink.
   2. The attached gingiva may be pigmented (**Figure 1-8**). This pigmented tissue is known as physiologic pigmentation.

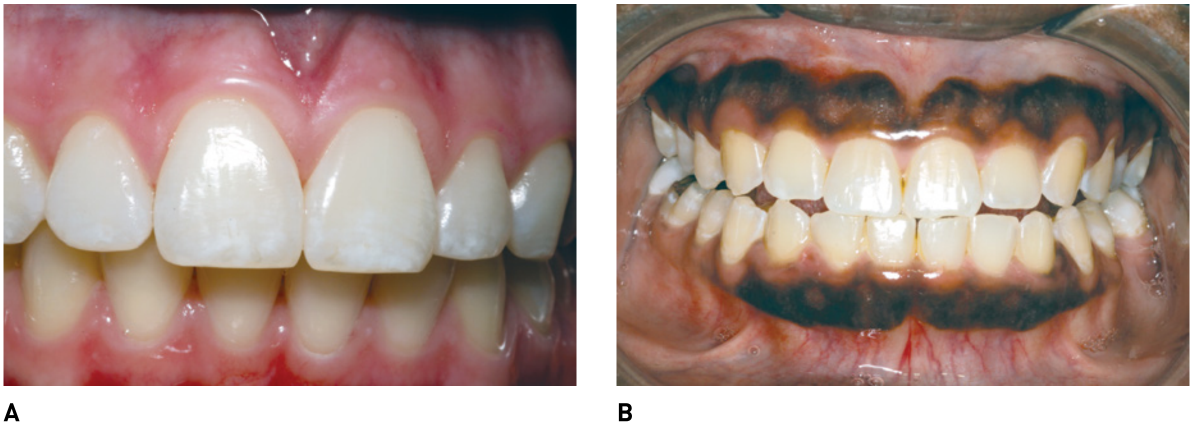

Figure 1-8 Color Variations of Normal Gingiva. The color of the normal gingiva varies among different individuals. **A.** The color is a lighter, coral pink in individuals with fair complexions. **B.** In individuals with dark skin and hair, the gingiva may be pigmented.

A and **B:** Courtesy of Elizabeth Carr, University of Mississippi Medical Center, Jackson, MS.

   a. Pigmentation occurs more frequently in dark-skinned individuals.[2]
   b. The pigmented areas of the attached gingiva may range from light brown to black.
   c. Pigmentation is due to an increased production of melanin (pigment) from melanocytes (melanin-producing cells found in the epidermal layer).

d. **Texture of the Attached Gingiva.** In health, the surface of the attached gingiva may have a dimpled appearance similar to the skin of an orange peel. This dimpled appearance is known as **stippling** (**Figure 1-9**). Stippling acts to provide mechanical reinforcement to the gingiva. Stippling is seen only on the attached and interdental gingiva, not the free gingiva. Healthy tissue, however, does not always exhibit a stippled appearance, and the presence of stippling varies greatly from individual to individual. In fact, stippling is only present in 40% of periodontally healthy adults. As such, stippling is absent in the majority of periodontally healthy individuals.

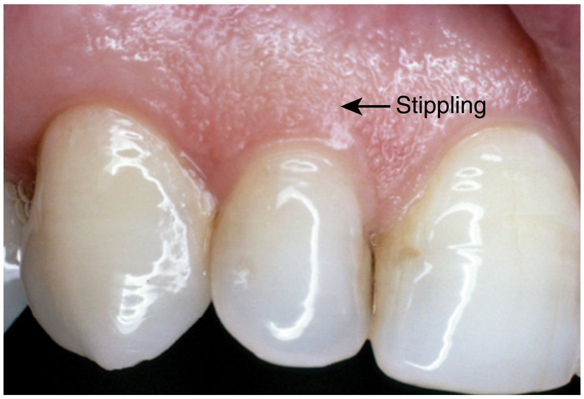

Figure 1-9 Gingival Stippling. In health, the surface of the attached gingiva and interdental gingiva may have a dimpled appearance known as gingival stippling.

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e. **Function of the Attached Gingiva**
   1. The attached gingiva allows the gingival tissue to withstand the mechanical forces created during activities such as mastication, speaking, and toothbrushing.
   2. The attached gingiva prevents the free gingiva from being pulled away (apically) from the tooth when tension is applied to the alveolar mucosa.

4. **Interdental Gingiva.** The **interdental gingiva** is the portion of the gingiva that fills the interdental embrasure between two adjacent teeth apical to the contact area (**Figure 1-10**).

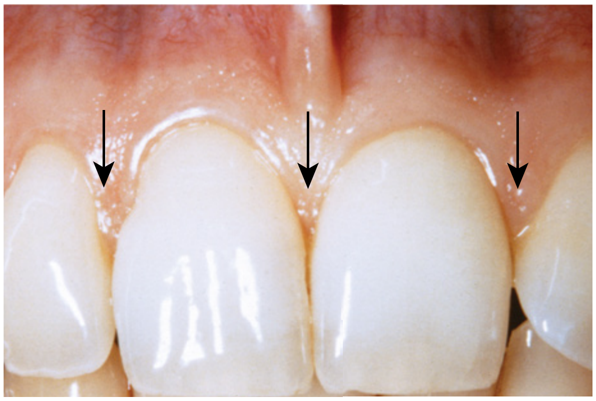

Figure 1-10 The Interdental Gingiva. The interdental tissue fills the area between two adjacent teeth.

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a. **Parts of Interdental Gingiva**
   1. The interdental gingiva consists of two interdental **papillae**—one facial papilla and one lingual papilla (*papilla* = singular noun; *papillae* = plural noun).
      a. The lateral borders and tip of an interdental papilla are formed by the free gingiva from the adjacent teeth.
      b. The center portion of the interdental papilla is formed by the gingival col.
   2. The **interdental col** is a valley-like depression in the portion of the interdental gingiva[2] that lies directly apical to the contact area of two adjacent (touching) teeth and connects the facial and lingual papillae (**Figure 1-11**). ***The col is absent if the adjacent teeth are not in contact*** (i.e., there is a space between two adjacent teeth), there is no adjacent tooth (i.e., the lingual surface of the posterior-most tooth in the arch), or if the interdental gingiva has receded.

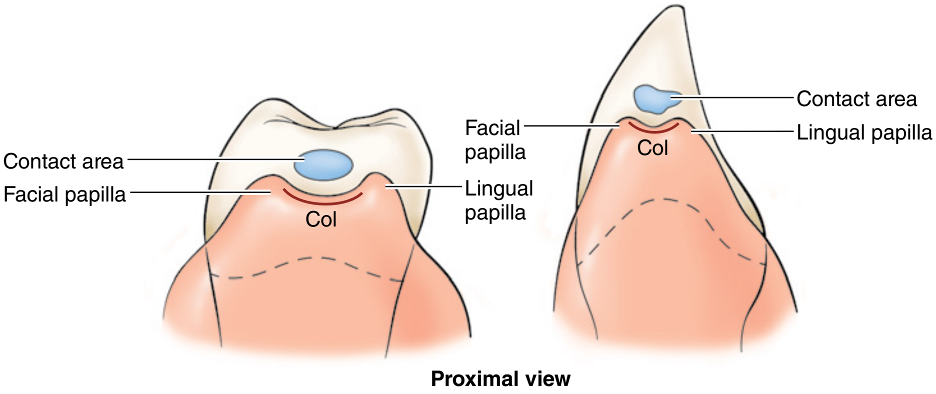

Figure 1-11 Interdental Col. Apical to the contact area between two teeth, the interdental gingiva has a concave (depressed) form. The concavity, the "col," is located between the facial and lingual papillae and passes beneath the contact area of two adjacent teeth.

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b. **Function of Interdental Gingiva.** The interdental gingiva prevents food from becoming packed between the teeth during mastication.

5. **Gingival Sulcus.** The **gingival sulcus** is the *space* between the free gingiva and the tooth surface (**Figure 1-12**).

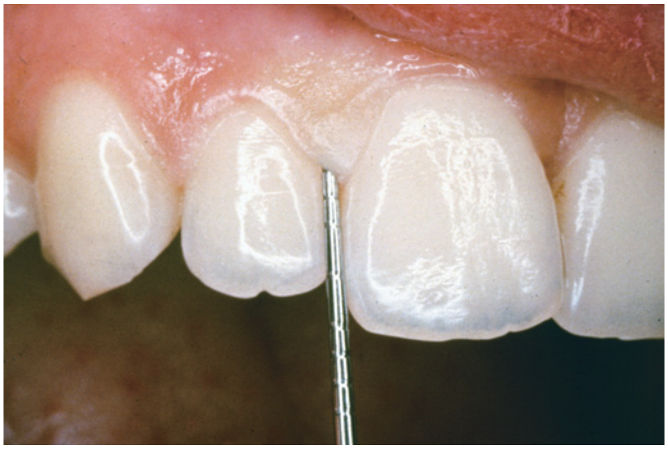

Figure 1-12 **Gingival Sulcus.** This photograph shows a periodontal probe inserted into the gingival sulcus, the space between the free gingiva and the tooth.

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a. **Description.** The sulcus is a V-shaped, shallow crevice space encircling the neck of a tooth.[3]
   1. The depth of a clinically healthy gingival sulcus is from 1 to 3 mm, as measured using a periodontal probe.
   2. Base of Sulcus. The base of the sulcus interfaces with the most coronal portion of the junctional epithelium (a specialized type of epithelium that attaches to the tooth surface).

b. **Gingival Crevicular Fluid.** The **gingival crevicular fluid**, also called the gingival sulcular fluid, is a fluid that seeps from the underlying connective tissue into the sulcular space.[4]
   1. Little or no fluid is found in the healthy gingival sulcus, but the fluid flow increases in the presence of dental plaque biofilm and the resulting gingival inflammation.[5]
   2. Fluid flow increases in response to toothbrushing, mastication, or other stimulation of the gingiva. The flow is greatly increased when the gingiva is inflamed.
   3. If a filter strip is inserted into the sulcus, it absorbs the fluid in the sulcus. Using the filter strip, the amount of gingival crevicular fluid can be measured and used as an index of gingival inflammation.

Periodontal Ligament

1. **Description**
   a. The periodontal ligament (PDL) is a network of soft connective tissue fibers that attach the root of the tooth to the bony walls of the tooth socket (**Figure 1-13**).

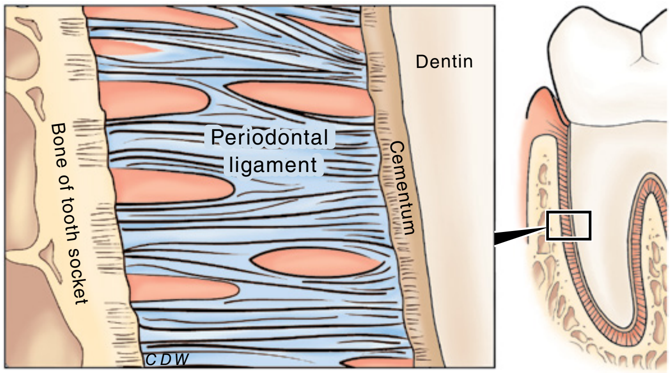

Figure 1-13 **Periodontal Ligament.**

- On the tooth side, the ends of the PDL fibers are anchored to the cementum of the root.
- On the bone side, the ends of the PDL fibers are anchored to the alveolar bone of the tooth socket.

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        1. The PDL is composed mainly of dense fibrous connective tissue.[3]
        2. The fibers of the PDL attach on one side to the root cementum and on the other side to the alveolar bone of the tooth socket.[6]
    b. The PDL not only connects the tooth to the alveolar process, but also supports the tooth in the socket and absorbs mechanical loads placed on the tooth, thus protecting the tooth in its socket.[7]

2. **Functions.** The PDL has five functions in the periodontium:
    a. Supportive function—the ligament fibers suspend and maintain the tooth in its socket.
    b. Sensory function—specialized nerve endings course through the PDL space to relay sensory information (such as pain and pressure sensations) from the tooth and surrounding attachment apparatus to the central nervous system.
    c. Nutritive function—blood vessels course through the PDL space are critical to providing oxygen and vital nutrients to the tissues of the periodontium
    d. Formative function— the tissues of the PDL contain many specialized formative cells such as fibroblasts, cementoblasts, and osteoblasts which build and maintain the attachment apparatus all throughout the lifetime of the tooth. Moreover, dormant epithelial cells (remnants of Hertwig's epithelial root sheath) and undifferentiated periodontal ligament cells populate the periodontal ligament space and may play a critical role in periodontal regeneration. Hence, ongoing periodontal research is focused on finding ways to tap into the regenerative potential of these promising cells.
    e. Remodeling function—can remodel the alveolar bone in response to pressure, such as that applied during orthodontic treatment (braces).

Cementum

1. **Description.** Cementum is a thin layer of hard, mineralized connective tissue that covers the surface of the tooth root (**Figure 1-14**).

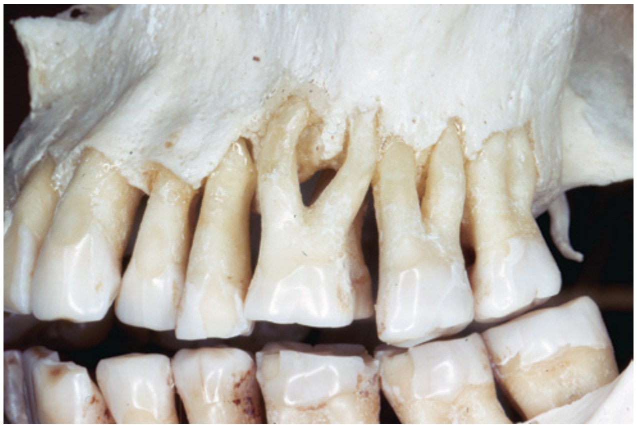

Figure 1-14 **Cementum.** Cementum is mineralized connective tissue that covers the root of the tooth; it is light yellow in color.

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2. **Characteristics of Cementum**
    a. Cementum overlies the dentin of the root. It is light yellow in color and softer than dentin or enamel. In terms of weight, it is composed of 50% to 55% organic substance—primarily, Type I collagen and noncollagenous matrix proteins—and 45% inorganic content (calcium and phosphate forms of hydroxyapatite and trace elements).
    b. Cementum is a bone-like tissue that is more resistant to resorption than bone.[8]
        1. Resistance to resorption (loss of substance) is an important characteristic of cementum that makes it possible for the teeth to be moved during orthodontic treatment.[9]

        2. The high resistance of cementum to resorption allows the pressure applied during orthodontics to cause resorption of the alveolar bone, for tooth movement, without resulting in root resorption.
    c. Like bone remodeling, cementum undergoes a continuous and slow physiologic process of resorption and repair throughout the lifetime of a tooth. This process allows for repair of existing cementum and deposition of new cementum. Thus, as an individual ages, the average thickness of cementum increases.
    d. There are two main types of cementum: cellular and acellular (**Table 1-2**).

Table 1-2 Characteristics of Cementum

	Acellular Cementum	Cellular Cementum
Time of development	Forms before teeth are in occlusion	Forms after teeth have reached occlusion
Histologic features	Devoid of cells	Composed of cementocytes
Location on root	Covers cervical two-thirds of root	Covers apical one-third of root
Function	Plays important role in tooth support	Compensates for active eruption and normal tooth wear by continuous deposition of cementum

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    e. Cementum does not have its own blood or nutrient supply; it receives its nutrients from the PDL.
    f. Cementum is relatively permeable to extrinsic dyes, organic substances, inorganic ions, and bacteria. In periodontal disease, bacteria have been shown to invade the cementum.
3. **Functions of Cementum in the Periodontium.** Cementum performs several important roles in the periodontium; therefore, conservation of healthy cementum should be a goal of periodontal instrumentation.
    a. The primary function of cementum is to give attachment to the collagen fibers of the PDL. Cementum anchors the ends of the PDL fibers (via terminal endings known as **Sharpey's fibers**) to the tooth. Without cementum, the tooth would fall out of its socket.
    b. The outer layer of cementum protects the underlying dentin and seals the ends of the open dentinal tubules.
    c. Cementum formation compensates for tooth wear at the occlusal or incisal surface due to attrition.
        1. Cementum is formed at the apical area of the root to compensate for occlusal attrition. This allows for the tooth to maintain its length. Consequently, the apical third of the root tends to be thicker (150 to 250 μm) than the coronal half of the root (16 to 60 μm).
        2. However, excessive deposition of cementum in the apical third of the root may result in an abnormality known as hypercementosis, which can potentially obstruct the apical foramen. Radiographically, the characteristic hallmark of hypercementosis is a radiopaque thickening of the cementum that is enveloped by the radiolucent shadow of the PDL space and an intact lamina dura. In the absence of any pathology, hypercementosis does not pose a problem and does not require treatment.

Alveolar Bone

1. **Description**
    a. The alveolar process, or alveolar bone, is the bone of the upper or lower jaw that surrounds and supports the roots of the teeth (**Figure 1-15**).

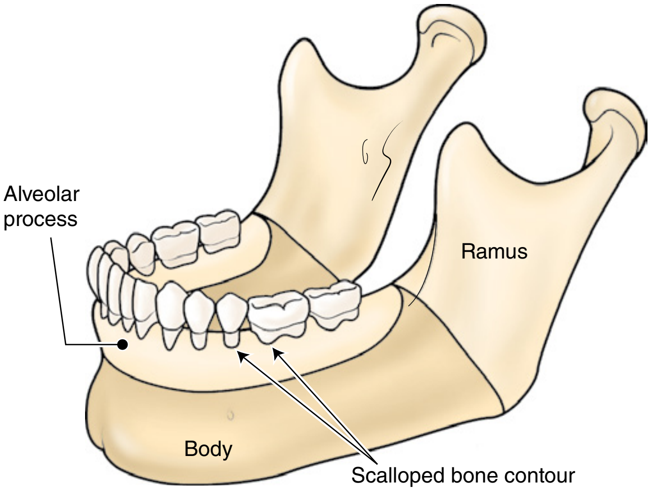
Alveolar process
Ramus
Body
Scalloped bone contour

Figure 1-15 Alveolar Process. The alveolar process is the bone that surrounds and supports the roots of the teeth.

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   b. Bone is mineralized connective tissue and consists by weight of about 60% inorganic material, 25% organic material, and 15% water.
   c. The existence of the alveolar bone is dependent on the presence of teeth; when teeth are extracted, in time, the alveolar bone resorbs. If teeth do not erupt, the alveolar bone does not develop.
2. **Function of the Alveolar Bone in the Periodontium.** The alveolar bone forms the bony sockets that provide support and protection for the roots of the teeth.
3. **Layers That Compose the Alveolar Process.** When viewed in cross section, the alveolar process is composed of three layers of hard tissue and covered by a thin layer of connective tissue (**Figures 1-16** and **1-17**).

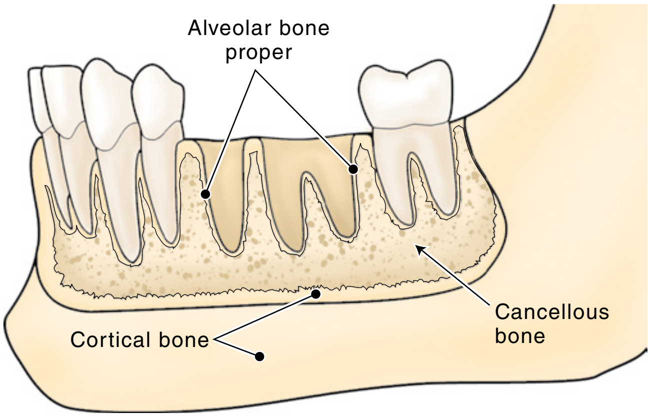
Alveolar bone proper
Cortical bone
Cancellous bone

Figure 1-16 Layers of the Alveolar Process. A lateral section of the mandible reveals three bony layers: the alveolar bone proper, cancellous bone, and cortical bone.

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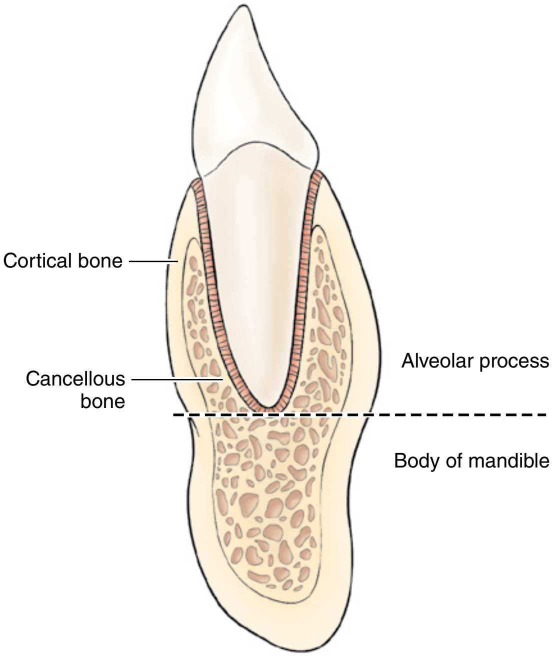
Cortical bone
Cancellous bone
Alveolar process
Body of mandible

Figure 1-17 Cross Section of the Mandible. The dotted line indicates the boundary of the alveolar process with the body of the mandible.

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a. The **alveolar bone proper** (or **cribriform plate**) is the thin layer of bone that lines the socket that surrounds the root of the tooth.
    1. The **alveolus** is the bony socket, a cavity in the alveolar bone that houses the root of a tooth (*alveolus* = singular; *alveoli* = plural) (**Figure 1-18**).

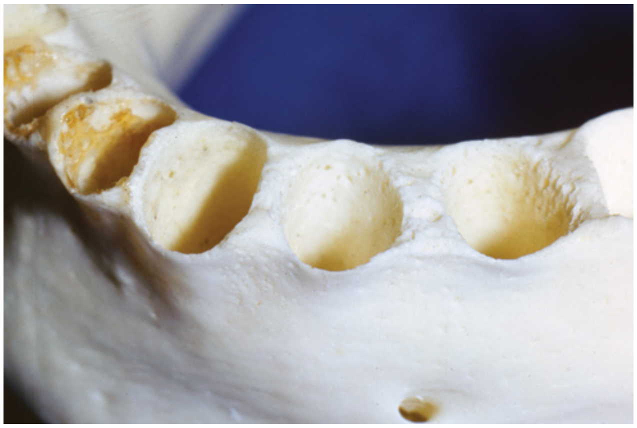

Figure 1-18 Alveoli of the Mandible. The alveoli are the sockets in the alveolar bone that house the roots of the teeth.

Courtesy of Dr. Don Rolfs, Periodontal Foundations, Wenatchee, WA.

    2. The alveolar bone proper has numerous holes (foramina) that allow blood vessels from the cancellous bone to connect with the vessels of the PDL space.
    3. The ends of the PDL fibers (Sharpey's fibers) are embedded in the alveolar bone proper.

b. The **cortical bone** (or cortical plate) is a layer of compact bone that forms the hard, outer wall of the mandible and maxilla on the facial and lingual aspects. This cortical bone surrounds the alveolar bone proper and gives support to the socket.
    1. The buccal cortical bone is thinner in the incisor, canine, and premolar regions; while thicker in molar regions.
    2. Because the cortical plate is only on the facial and lingual sides of the jaw, it will not appear on a radiograph; only the cancellous bone and the alveolar bone proper can be seen on a radiograph.
    3. The **alveolar crest** is the coronal-most portion of the alveolar process.
        a. In health, the alveolar crest is located 1 to 2 mm apical to (below) the CEJs of the teeth (**Figure 1-19**).

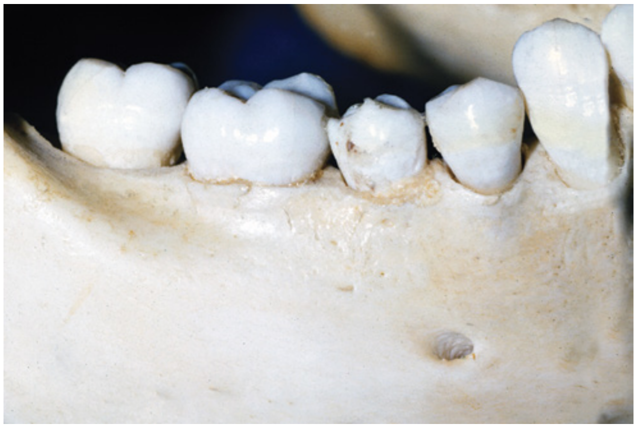

Figure 1-19 Bony Contours. The alveolar crest conforms to a scalloped (curved) line that follows the contours of the cementoenamel junctions.

Courtesy of Dr. Don Rolfs, Periodontal Foundations, Wenatchee, WA.

  b. When viewed from the facial or lingual aspect, the alveolar crest meets the teeth in a scalloped (curved) form that follows the contours of the CEJs.

c. The **cancellous bone** (or **spongy bone**) is the lattice-like bone that fills the interior portion of the alveolar process (between the cortical bone and the alveolar bone proper). Cancellous bone is found mostly in the interproximal areas. Furthermore, a higher proportion of cancellous bone tends to exist in the maxilla than in the mandible, whereas a higher proportion of cortical bone is found in the mandible. The cancellous bone is oriented around the tooth to form support for the alveolar bone proper.

d. The **periosteum** is a layer of connective soft tissue covering the outer surface of bone; it consists of an outer layer of collagenous tissue and an inner layer of fine elastic fibers.

Section 2

Nerve Supply, Blood Supply, and Lymphatic System

Nerve Supply to the Periodontium

1. **Description.** The **innervation** of the periodontium—that is, the nerve supply to the periodontium—occurs via the branches of the **trigeminal nerve**—Cranial Nerve V (**Figure 1-20**). Innervation to the maxilla (**Figure 1-21**) is by the second branch of the trigeminal nerve (the maxillary nerve—Cranial Nerve V2) and the mandible by the third branch (the mandibular nerve—Cranial Nerve V3). The first branch (the ophthalmic nerve—Cranial Nerve V1) does not innervate regions of the oral cavity, so it will not be covered in this section.

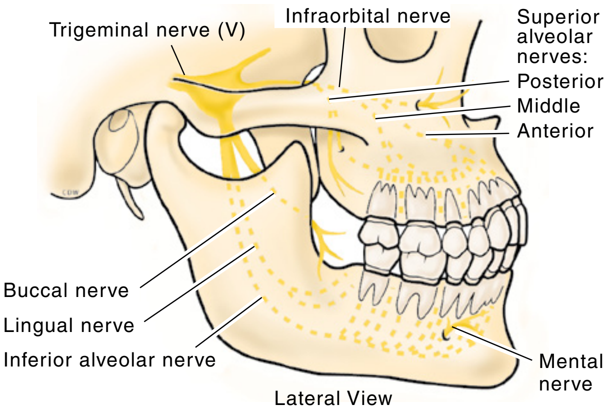

Figure 1-20 Nerve Supply to the Periodontium (Lateral View). The nerve supply to the periodontium is derived from the branches of the trigeminal nerve.

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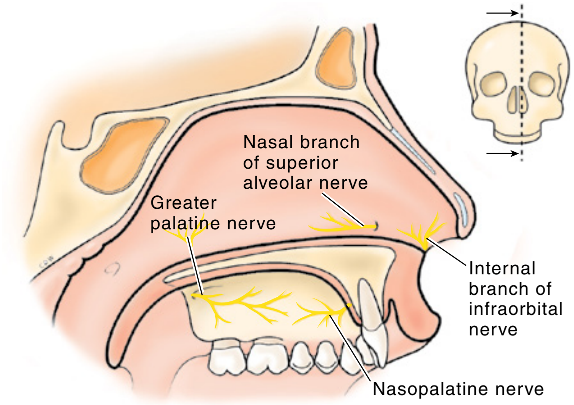
Nasal branch of superior alveolar nerve
Greater palatine nerve
Internal branch of infraorbital nerve
Nasopalatine nerve

Figure 1-21 Nerve Innervation to the Palate (Midsagittal Section). The sensory nerves of the palate are branches of the maxillary nerve.

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  a. The second branch of the trigeminal nerve exits from the skull through the foramen rotundum and courses to the skin of the middle of the face to provide only sensory innervation to the maxilla. The third branch of the trigeminal nerve exits from the skull through the foramen ovale and courses to the lower face to provide both sensory and motor innervation to the mandible.

  b. The trigeminal nerve is responsible for the sensory innervation of most of the skin of the front part of the face and head, the teeth, oral cavity, maxillary sinus, and nasal cavity.

  c. The motor function of the trigeminal nerve regulates movements of the mandible, such as jaw opening, jaw closing and excursive movements of the mandible—all key movements performed in the act of chewing and talking.

2. **Functions of the Nerve Supply to the Periodontium**

  a. Nerve receptors in the gingiva, alveolar bone, and PDL register pain, touch, and pressure.

   b. Nerves in the PDL provide information about movement and tooth position. These nerves provide the sensations of light touch or pressure against the teeth and play an important role in the regulation of chewing forces and movements. When biting down on something hard, it is the nerves of the PDL that are stimulated, allowing the individual to experience a sense of pressure with the teeth against the hard object.

3. **Innervation of the Periodontium**
   a. Innervation of the Gingiva
      1. Innervation of the gingiva of the maxillary arch is from the superior alveolar nerves (anterior, middle, and posterior branches), infraorbital nerve, and the greater palatine and nasopalatine nerves.
      2. Innervation of the gingiva of the mandibular arch is from the mental nerve, buccal nerve, and the sublingual branch of the lingual nerve (**Figure 1-22**).

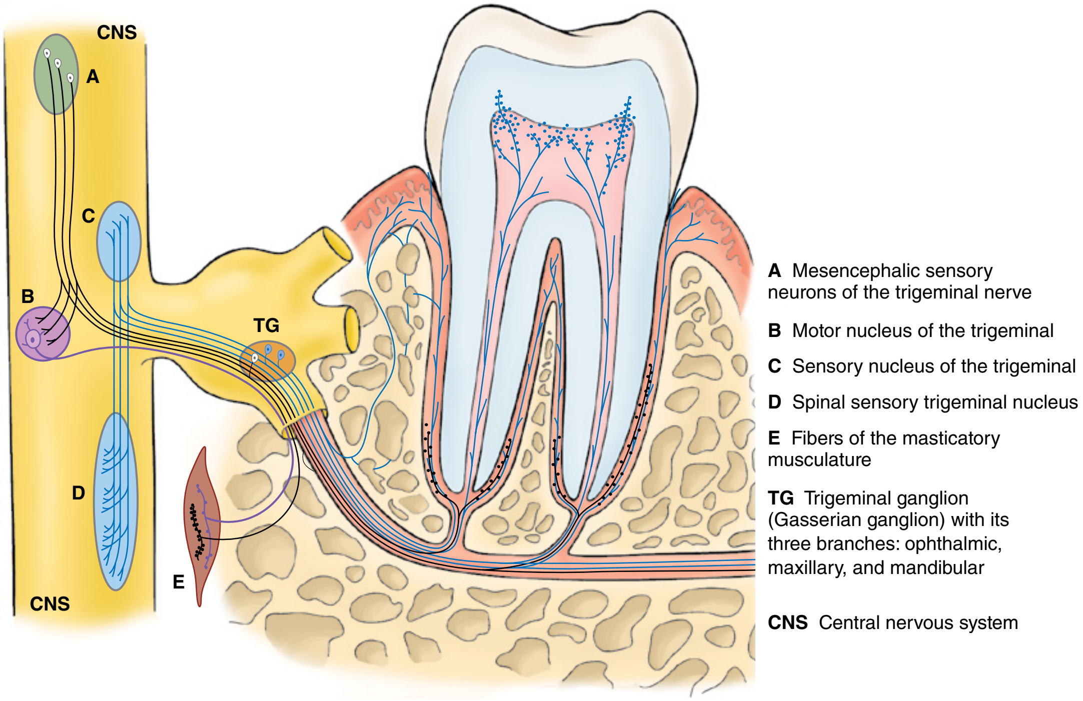
CNS
A
C
B
TG
D
E
CNS
A Mesencephalic sensory neurons of the trigeminal nerve
B Motor nucleus of the trigeminal
C Sensory nucleus of the trigeminal
D Spinal sensory trigeminal nucleus
E Fibers of the masticatory musculature
TG Trigeminal ganglion (Gasserian ganglion) with its three branches: ophthalmic, maxillary, and mandibular
CNS Central nervous system

Figure 1-22 **Innervation of Mandibular Teeth.** Innervation of the gingiva and periodontium is via the mandibular nerve.

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   b. **Innervation of the Teeth and Periodontal Ligament**
      1. Innervation of the teeth and PDL of the maxillary arch is from the superior alveolar nerves (anterior, middle, and posterior branches).
      2. Innervation of the teeth and PDL of the mandibular arch is from the inferior alveolar nerve.

Blood Supply to the Periodontium

1. **Description.** The vessels of the periodontium **anastomose** (join together) to create a complex system of blood vessels that supply blood to the periodontal tissues.
   a. This network of blood vessels acts as a unit, supplying blood to the soft and hard tissues of the maxilla and mandible.
   b. It is the proliferation of this rich blood supply to the gingiva that accounts for the dramatic color changes that are seen in gingivitis.
2. **Function.** The major function of the complex network of blood vessels of the periodontium is to transport oxygen and nutrients to the tissue cells of the periodontium and to remove carbon dioxide and other waste products from the cells for elimination.

3. **Vascular Supply to the Periodontium** (**Figure 1-23**)

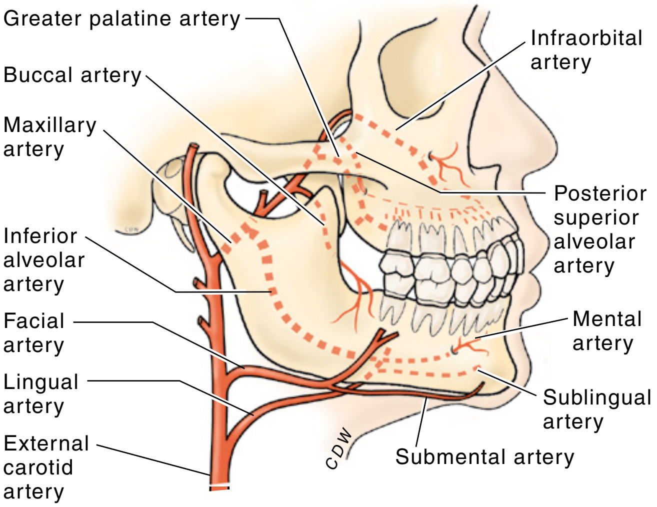

Figure 1-23 Vascular Supply to the Periodontium. A complex network of blood vessels supplies blood to the periodontium.

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- a. Maxillary gingiva, PDL, and alveolar bone
  1. External carotid → maxillary artery
  2. Branches of the inferior alveolar artery that vascularize the maxillary periodontium: anterior and posterior superior alveolar arteries, infraorbital artery, and greater palatine artery
- b. Mandibular gingiva, PDL, and alveolar bone
  1. External carotid → maxillary artery → inferior alveolar artery
  2. Branches of the inferior alveolar artery that vascularize the mandibular periodontium: the incisor branch, mental branch, and mylohyoid branch.

4. **Vascular Supply to the Teeth and Periodontal Tissues**

- a. **The Major Arteries**
  1. Superior alveolar artery—maxillary periodontal tissues
  2. Inferior alveolar artery—mandibular periodontal tissues
- b. **Branch Arteries** (**Figures 1-24** and **1-25**)
  1. The dental artery: a branch of the superior or inferior alveolar artery.
  2. Intraseptal artery: courses through the interdental bone and enters the tooth socket through rami perforantes.
  3. Rami perforantes: terminal branches of the intraseptal artery. These vessels penetrate the tooth socket and enter the PDL space where they anastomose (join) with the blood vessels from the alveolar bone and PDL.
  4. Supraperiosteal blood vessels: located along the outer surfaces of the facial and lingual cortical plates. These vessels provide the main blood supply to the free and attached gingiva; these vessels anastomose with capillaries from the alveolar bone (intraseptal artery) and the PDL (PDL vessels).
  5. Subepithelial plexus: branches of the supraperiosteal blood vessels located in the connective tissue beneath the free and attached gingiva.
  6. PDL vessels: supply the PDL and form a complex network of vessels that surrounds the root. These vessels also anastomose with the blood vessels from the intraseptal artery and the supraperiosteal arterioles.
  7. Dentogingival plexus: branches of the subepithelial plexus of blood vessels, intraseptal artery, supraperiosteal artery, and PDL vessels course coronally, forming a fine-meshed network of blood vessels located in the connective tissue beneath the junctional epithelium.

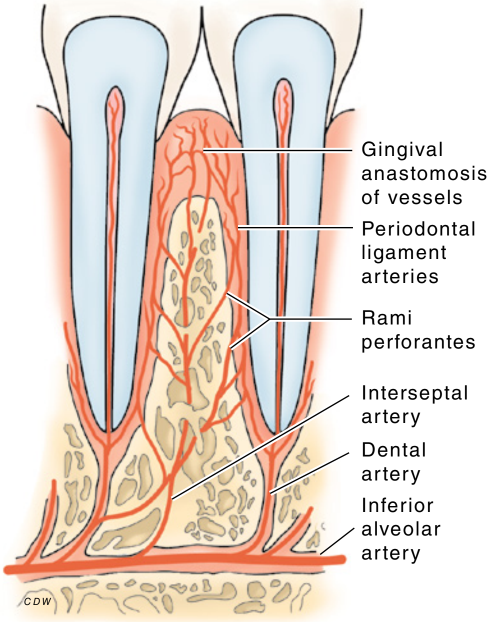
Gingival anastomosis of vessels
Periodontal ligament arteries
Rami perforantes
Interseptal artery
Dental artery
Inferior alveolar artery
CDW

Figure 1-24 Branch Arteries. The branch arteries supply blood to the teeth and periodontium.

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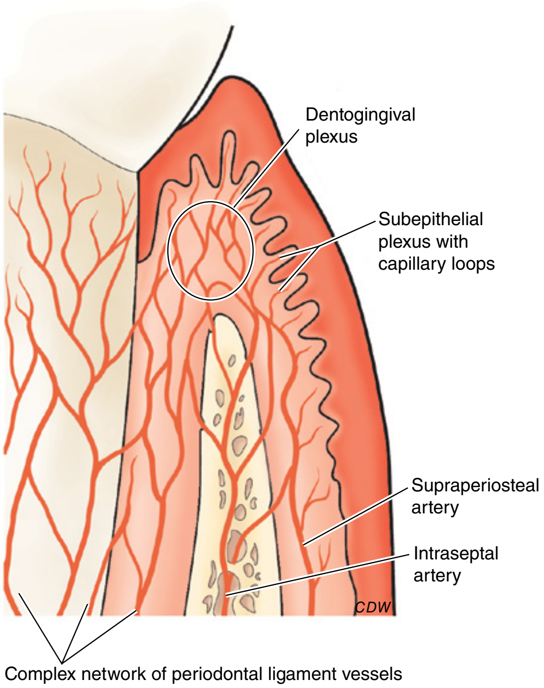
Dentogingival plexus
Subepithelial plexus with capillary loops
Supraperiosteal artery
Intraseptal artery
CDW
Complex network of periodontal ligament vessels

Figure 1-25 Network of Vessels. A fine network of vessels supplies blood to gingiva, gingival connective tissue, and periodontal ligament.

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Lymphatic System and the Periodontium

1. **Description.** The **lymphatic system** is a network of lymph nodes connected by lymphatic vessels that plays an important role in the body's defense against infection.
2. **Function. Lymph nodes** are small bean-shaped structures located on either side of the head, neck, armpits, and groin. These nodes filter out and trap bacteria, fungi, viruses, and other unwanted substances to safely eliminate them from the body.
3. **Lymph Drainage of the Periodontium.** The lymph from the periodontal tissues is drained to the lymph nodes of the head and neck (**Figure 1-26**).

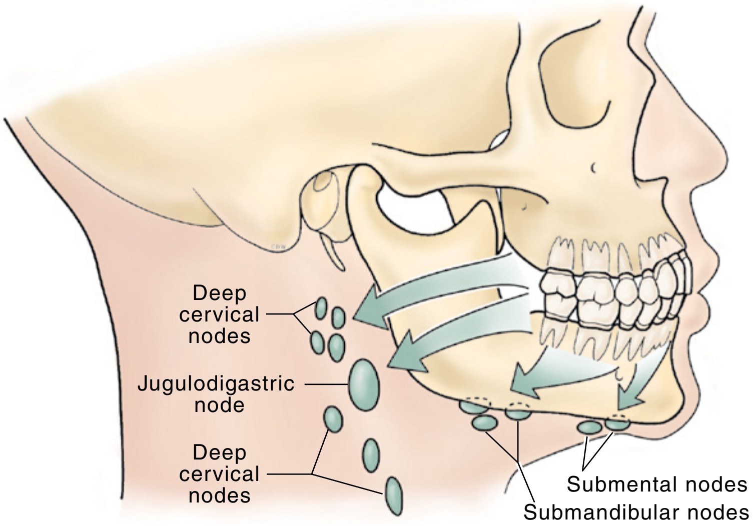
Deep cervical nodes
Jugulodigastric node
Deep cervical nodes
Submental nodes
Submandibular nodes

Figure 1-26 Lymphatic System of the Periodontium. The lymph from the periodontium is drained to the lymph nodes of the head and neck.

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a. Submandibular lymph nodes—drain most of the periodontal tissues
b. Deep cervical lymph nodes—drain the palatal gingiva of the maxilla
c. Submental lymph nodes—drain the gingiva in the region of the mandibular incisors
d. Jugulodigastric lymph nodes—drain the gingiva in the third molar region

Chapter Summary Statement

The gingiva, periodontal ligament, cementum, and alveolar bone make up a system of tissues that surround the teeth. Each tissue of the periodontium plays a vital role in the functioning and retention of the teeth.

- The gingiva provides a tissue seal around the cervical portion of the tooth and covers the alveolar process.
- The periodontal ligament supports the tooth in its socket, provides nutrients and sensory innervation (feeling) to the tooth, and maintains the cementum and alveolar bone of the tooth socket.
- The cementum anchors the periodontal ligament to the tooth and seals the ends of the open dentinal tubules. Cementum formation compensates for tooth wear due to occlusal attrition.
- The alveolar bone forms the bony sockets that provide support and protection for the roots of the teeth.

Section 3

Focus on Patients

Clinical Patient Care

CASE 1

A patient involved in an automobile accident receives a penetrating wound involving the oral cavity. The wound enters the alveolar mucosa near the apex of a lower premolar tooth and extends from the surface mucosa all the way through the tissues to the premolar tooth root. List the periodontal tissues most likely injured by this penetrating wound.

CASE 2

A patient who has lost a maxillary lateral incisor tooth is scheduled to have a dental implant placed. The dental implant placement will require the clinician to prepare a hole with a drill in the bone formerly occupied by the lateral incisor tooth. Name the types of bone that will most probably be penetrated by the drill.

CASE 3

A dentist injects a local anesthetic before working on a maxillary molar tooth. The injection results in complete loss of sensation in the molar tooth and in most of the gingiva surrounding the molar tooth. Name the nerves that most likely have been affected by the injection of the local anesthetic.

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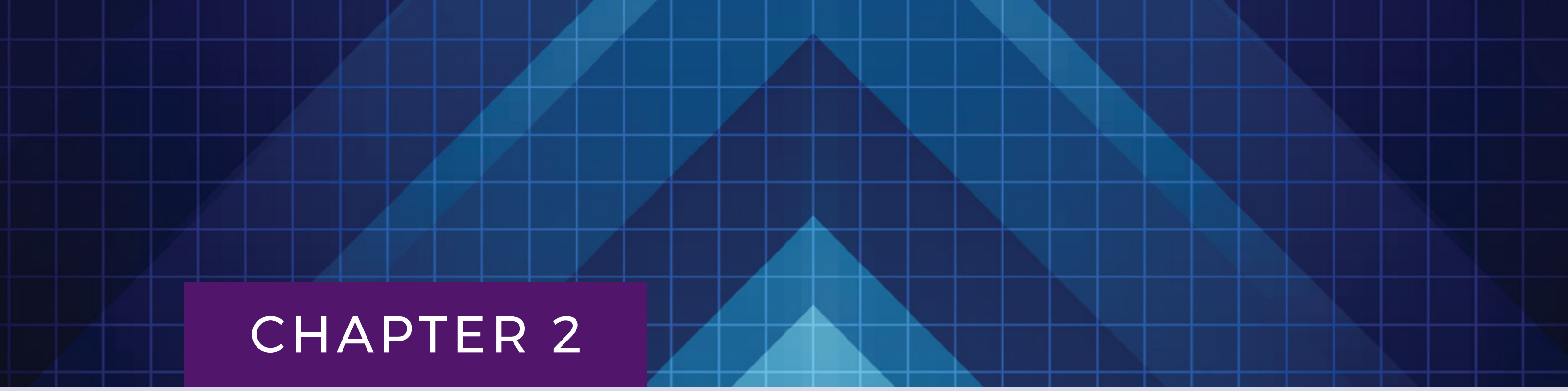

CHAPTER 2

Microscopic Anatomy of the Periodontium

- Section 1 Histology of the Body’s Tissues
  - Microscopic Anatomy of a Tissue
  - Microscopic Anatomy of Epithelial Tissue
  - Microscopic Anatomy of Connective Tissue
  - Epithelial–Connective Tissue Interface
  - Epithelial Cell Junctions
- Section 2 Histology of the Gingiva
  - Microscopic Anatomy of Gingival Epithelium
  - Why the Teeth Need a Junctional Epithelium
  - Attachment of the Cells of the Junctional Epithelium
  - Microscopic Anatomy of Gingival Connective Tissue
- Section 3 Histology of Root Cementum and Alveolar Bone
  - Microscopic Anatomy of Cementum
  - Microscopic Anatomy of Alveolar Bone
- Section 4 Focus on Patients
  - Clinical Patient Care
  - Evidence in Action: Clinical Relevance

CLINICAL APPLICATION

Dental health care providers continuously interact with patients when making clinical decisions, performing clinical procedures, evaluating new techniques, and adapting to emerging treatment approaches. Nearly every action taken by a clinician requires a detailed knowledge of the anatomy of the tooth-supporting structures—the periodontium. Chapter 1 dealt with what is known about the fundamental structure of the complex system of tissues that support the teeth. Chapter 2 deals with the microscopic anatomy of these same structures. The information presented in these two chapters can serve as a basis for organizing thoughts about additional anatomical information as it becomes available through additional research.

LEARNING OBJECTIVES

- Describe the histology of the tissues and the function that each serves in the human body.
- List and define the layers that comprise the stratified squamous epithelium of the skin.
- Define the term *keratin* and describe its function in the epithelium.
- Describe the composition and function of connective tissue.

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- Describe the epithelial–connective tissue interface found in most tissues of the body, such as the interface between the epithelium and connective tissues of the skin.
- Define the term *cell junction* and describe its function in the epithelial tissues.
- Compare and contrast the terms *desmosome* and *hemidesmosome*.
- Identify the three anatomical areas of the gingival epithelium on an unlabeled drawing depicting the microscopic anatomy of the gingival epithelium.
- Describe the location and function of the following regions of the gingival epithelium: oral epithelium, sulcular epithelium, and junctional epithelium.
- State the level of keratinization present in each of the three anatomical areas of the gingival epithelium (keratinized, nonkeratinized, or parakeratinized).
- State which of the anatomical areas of the gingival epithelium have an uneven, wavy epithelial–connective tissue interface in health and which have a smooth junction in health.
- Identify the enamel, gingival connective tissue, junctional epithelium, internal basal lamina, external basal lamina, epithelial cells, desmosomes, and hemidesmosomes on an unlabeled drawing depicting the microscopic anatomy of the junctional epithelium and surrounding tissues.
- Compare and contrast the function of the supragingival fiber bundles and the periodontal ligament in the periodontium.
- Identify the fiber groups of the periodontal ligament on an unlabeled drawing.
- Define the terms *cementum* and *Sharpey's fibers* and describe their function in the periodontium.
- List the three relationships that the cementum may have in relation to the enamel at the cementoenamel junction.
- Define the term *alveolar bone* and describe its function in the periodontium.

KEY TERMS

Histology
Tissue
Cells
Extracellular matrix
Epithelial tissue
Stratified squamous epithelium
Basal lamina
Keratinization
Keratinized epithelial cells
Nonkeratinized epithelial cells
Connective tissue
Epithelial–connective tissue interface
Basement membrane
Epithelial ridges
Connective tissue papillae
Cell junctions
Desmosome
Hemidesmosome
Gingival epithelium
Oral epithelium
Sulcular epithelium
Keratin
Gingival crevicular fluid
Junctional epithelium
Internal basal lamina
External basal lamina
Collagen fibers
Supragingival fiber bundles
Gingival fibers
Dentogingival unit
Periosteum
Periodontal ligament
Fiber bundles of the periodontal ligament
Sharpey's fibers
Cementum
Cementum proteins
OMG (overlap, meet, gap)
Alveolar process
Bone remodeling

Section 1

Histology of the Body's Tissues

Histology is a branch of anatomy concerned with the study of the microscopic features of tissues. Knowledge of the microscopic characteristics of tissues is a prerequisite for understanding the microscopic anatomy of the periodontium. Section 1 reviews the microscopic anatomy of the epithelial and connective tissues of the body.

Microscopic Anatomy of a Tissue

A **tissue** is a group of interconnected cells that perform a similar function within an organism. For example, muscle cells group together to form muscle tissue that functions to move parts of the body. The tissues and organs of the body are composed of several different types of cells and extracellular elements outside of the cells.

1. **Cells**
   a. **Cells** are the smallest structural unit of living matter capable of functioning independently.
   b. Cells group together to form a tissue.
   c. The four basic types of tissue are epithelial, connective, nerve, and muscle tissues.
2. **Extracellular Matrix.** Tissues are not made up solely of cells. Rather, the tissue is made up of a gel-like substance containing interwoven protein fibers which surround the cells.
   a. The **extracellular matrix** is a mesh-like network that surrounds the cells (**Figure 2-1**). It functions as an insoluble three-dimensional structural scaffold that physically supports the cells within the tissue. This network holds cells together and provides a biomechanical framework within which cells can migrate and interact with one another.

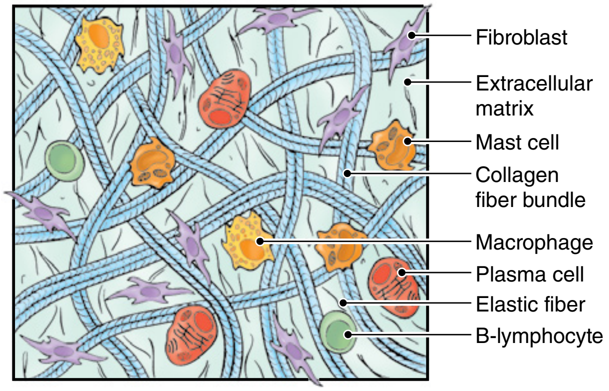

Figure 2-1 **Extracellular Matrix.** The extracellular matrix surrounds the cells of a tissue and is composed of fibers and a gel-like substance.

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   b. The extracellular matrix is composed mainly of ground substance and fibrous proteins.
      1. The ground substance is an amorphous, colorless, viscous, gel-like fluid that fills the space between the cells. The ground substance facilitates the attachment of cells to the matrix and aids in the exchange of nutrients between cells and capillaries.
      2. The fibrous proteins consist of collagen, elastin, and reticular fibers. Of the three types of fibrous proteins, collagens are the main structural component of the extracellular matrix.
   c. Amount of Extracellular Matrix
      1. In epithelial tissue, the extracellular matrix is sparse, consisting mainly of a thin mat called the basal lamina which underlies the epithelium.
      2. In connective tissue, the extracellular matrix is more plentiful than the cells that it surrounds.

Microscopic Anatomy of Epithelial Tissue

1. **Description**. The **epithelial tissue** makes up the outer surface of the body (skin or epidermis) and lines the body cavities such as the mouth, stomach, and intestines (mucosa). The skin and mucosa of the oral cavity are made up of **stratified squamous epithelium**—a type of epithelium composed of flat cells arranged in several layers.
2. **Composition of Epithelial Tissue.** Epithelial tissue mainly consists of plentiful cells supported by a thin mat of extracellular matrix (**Figure 2-2**).
   a. **Plentiful Cells.** Most of the volume of epithelial tissue consists of many closely packed epithelial cells. Epithelial cells are bound together into sheets.
   b. **Sparse Extracellular Matrix**
      1. The extracellular matrix is a minor component of the epithelial tissue existing mainly in the basal lamina.
      2. The **basal lamina** is a thin mat of extracellular matrix secreted by the epithelial cells. This mat supports the overlying epithelium (somewhat like the foundation of a building). The basal lamina also functions like a filter that allows only water and small molecules to pass through.

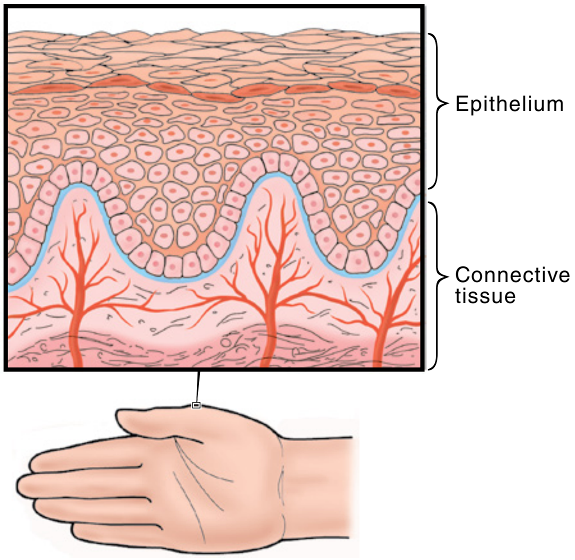

Figure 2-2 Stratified Squamous Epithelium and Connective Tissue of the Skin. The epithelium of the skin consists of many closely packed epithelial cells and a thin basal lamina. The epithelium of the skin rests on a supporting bed of connective tissue. The epithelium does not contain blood vessels; nourishment is received from blood vessels in the underlying connective tissue.

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3. **Keratinization.** **Keratinization** is the process by which epithelial cells on the surface of the skin become stronger and waterproof.
   a. **Keratinized Epithelial Cells**
      1. **Keratinized epithelial cells** have no nuclei and form a tough, resistant layer on the surface of the skin.
      2. The most heavily keratinized epithelium of the body is found on the palms of the hands and soles of the feet. Unlike epithelium found in other parts of the body, the palms of the hands and the soles of the feet have an extra layer of epithelium known as the stratum lucidum. This added stratum lucidum layer makes the epithelial tissues thicker and more resilient to the everyday effects of frictional, physical stress applied to the aforementioned body parts. It should be noted that the stratum lucidum is not an epithelial layer found in the tissues of the oral cavity.
   b. **Nonkeratinized Epithelial Cells**
      1. **Nonkeratinized epithelial cells** have nuclei and act as a cushion against mechanical stress and wear. Nonkeratinized epithelial cells are softer and more flexible.
      2. Nonkeratinized epithelium is found in areas such as the mucosal lining of the cheeks—permitting the mobility needed to speak, chew, and make facial expressions.
4. **Blood Supply.** Epithelial tissues are avascular, meaning that there is a complete lack of blood vessels in the epithelium. The epithelial layer receives oxygen and nourishment from blood vessels located in the underlying connective tissue via a process known as diffusion.

Microscopic Anatomy of Connective Tissue

1. **Description.** **Connective tissue** fills the spaces between the tissues and organs in the body. It supports and binds other tissues. Connective tissue consists of sparse cells separated by abundant extracellular substance.
2. **Composition of Connective Tissue**
   a. **Sparse Cells.** Unlike the cells in the overlying epithelial layer, the connective tissue cells are sparsely distributed in the extracellular matrix. Several types of cells are found in the connective tissue:
      1. Fibroblasts (“fiber-builders”)—cells that form the extracellular matrix (fibers and ground substance) and secrete it into the intercellular spaces

2. Macrophages and neutrophils—phagocytes ("cell-eaters") that devour dying cells and invading microorganisms.
3. Lymphocytes—immune cells that play an essential role in host defense

b. **Plentiful Extracellular Matrix.** The extracellular matrix—a rich gel-like substance containing a network of strong fibers—is the major component of connective tissue. The network of the fiber matrix, rather than the cells, gives connective tissue the strength to withstand mechanical forces.

3. **Dental Connective Tissue.** All dental tissues—cementum, dentin, alveolar bone, and the pulp—are specialized forms of connective tissue *except enamel*. Enamel is classified as an epithelial tissue because the cells that produce enamel (ameloblasts) are derived from the ectoderm.

Epithelial–Connective Tissue Interface

1. **Description.** The **epithelial–connective tissue interface** is the boundary where the epithelial and connective tissues meet.
2. **The Basement Membrane and Basal Lamina**
   a. As discussed previously, the basal lamina is a thin layer secreted by the epithelial cells on which the epithelium sits. The term *basal lamina* often is confused with the term *basement membrane* and is sometimes used inconsistently in the literature.
   b. The basal lamina is not visible under the light microscope but can be distinguished under the higher magnification of an electron microscope. The basal lamina facilitates the attachment of the epithelial cells to adjacent structures, such as the tooth surface.
   c. The term **basement membrane** specifies a thin layer of tissue visible with a light microscope beneath the epithelium. The basement membrane is formed by the combination of a basal lamina and a reticular lamina.
3. **Morphologic Patterns of the Epithelial–Connective Tissue Interface.** Depending on the form, function, and location of the epithelial tissue, two different morphologic patterns can be seen at this interface.
   a. **Tight Interdigitation at the Epithelial-Connective Tissue Interface** In most places in the body—including the oral epithelium—the epithelial layer interdigitates with the underlying connective tissue like the fingers of two clasped hands interlocked together (**Figure 2-3**).

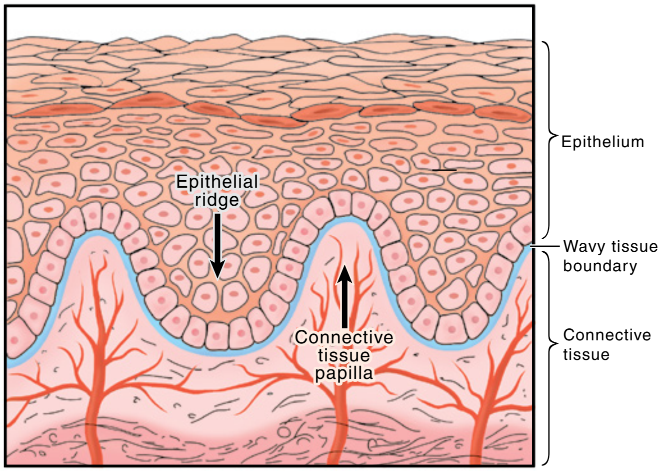

Figure 2-3 The Tight Interdigitation at the Epithelial–Connective Tissue Interface. Histologically, the epithelial ridges (that extend down into the connective tissue) and connective tissue papillae (that extend upward into the epithelium) are similar to finger-like projections that interlock with each other. Clinically, the appearance of gingival stippling represents the extensive tight interdigitation between the two layers.

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        1. **Epithelial ridges**—deep extensions of epithelium that reach down into the connective tissue (see Figures 2-7 and 2-8). The epithelial ridges are also known as rete pegs.
        2. **Connective tissue papillae**—finger-like extensions of connective tissue that project up and interlock with the epithelium.
        3. In periodontally healthy individuals, gingival stippling (as described in Chapter 1) is correlated to the presence of rete pegs which correspond to the microscopic elevations and depressions of the epithelial-connective tissue interface.
        4. By increasing the surface area of the epithelial tissue in contact with the underlying connective tissue, the tight interdigitation at this interface offers added protection from physical shearing forces that may be applied to the overlying epithelial tissue
    b. **Smooth Non-interdigitating Interface**
        1. Contrastingly, in some specialized epithelial tissues in the body, the interface between the epithelial layer and the connective layer has an even, smooth, non-interdigitating interface with no epithelial ridges or connective tissue papillae.
        2. In the oral cavity, this type of interface can be histologically seen in healthy sulcular epithelium and healthy junctional epithelium (refer to Figure 2-5).

4. **Function of the Epithelial-Connective Tissue Interface**
    a. **Enhances Adhesion.** The contours of the tissue interface enhance the adhesion of the epithelium to the connective tissue by increasing the surface area of the junction between the two tissues. This strong adhesion of the epithelium allows the skin to resist mechanical forces.
    b. **Provides Nourishment.** The contours of the junction between the epithelium and connective tissue also increase the area from which the epithelium can receive nourishment from the underlying connective tissue. The epithelium does not have its own blood supply; consequently, it relies on the underlying blood vessels in the connective tissue for nourishment.

Epithelial Cell Junctions

Neighboring epithelial cells attach to one another by specialized cell junctions that give the tissue strength to withstand mechanical forces and to form a protective barrier.

1. **Definition.** **Cell junctions** are cellular structures that mechanically "lock" a cell and its cytoskeleton to its neighboring cells or to the basal lamina.
2. **Purpose.** Cell junctions bind cells together so that they can function as a strong structural unit. Tissues, such as the epithelium of the skin, have the most abundant number of cell junctions to withstand severe mechanical stresses.
3. **Types of Epithelial Cell Junctions**
    a. **Desmosome**—a specialized cell junction that connects two neighboring epithelial cells and their cytoskeletons together. You might think of desmosomes as being like the snaps used to close a denim jacket. Instead of fastening the front of a jacket together, desmosomes fasten epithelial cells together (**Figure 2-4A**).
        1. A cell-to-cell connection
        2. An important form of cell junction found in the gingival epithelium
    b. **Hemidesmosome**—a specialized cell junction that connects the epithelial cells to the basal lamina (**Figure 2-4B**). You might think of hemidesmosomes as specialized structures that represent half of a desmosome.
        1. A cell-to-basal lamina connection
        2. An important form of cell junction found in the gingival epithelium

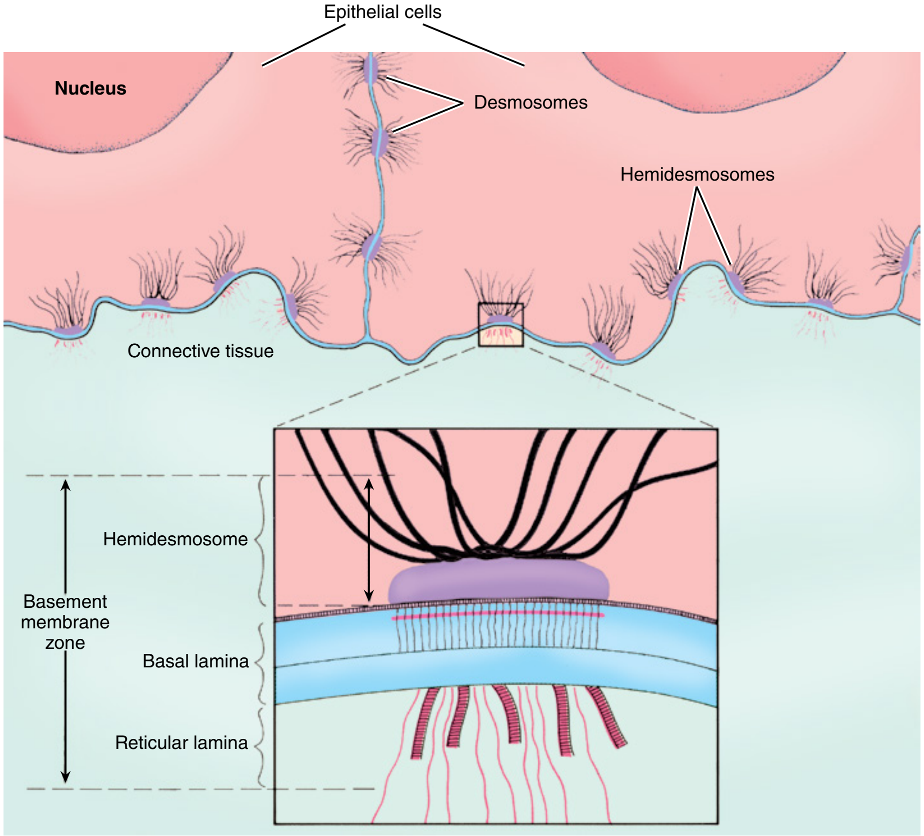
Epithelial cells
Nucleus
Desmosomes
Hemidesmosomes
Connective tissue
Hemidesmosome
Basement membrane zone
Basal lamina
Reticular lamina

Figure 2-4A The Epithelial–Connective Tissue Interface. The epithelial–connective tissue interface is the site of the basement membrane zone, a complex structure mostly synthesized by the epithelial cells. **Inset**: A representation of an electron micrograph showing the hemidesmosomal attachment to the basal lamina.

Modified with permission from Rubin R, Strayer DS. *Rubin's Pathology: Clinicopathologic Foundations of Medicine*. 5th ed. Lippincott Williams & Wilkins; 2008. © Wolters Kluwer.

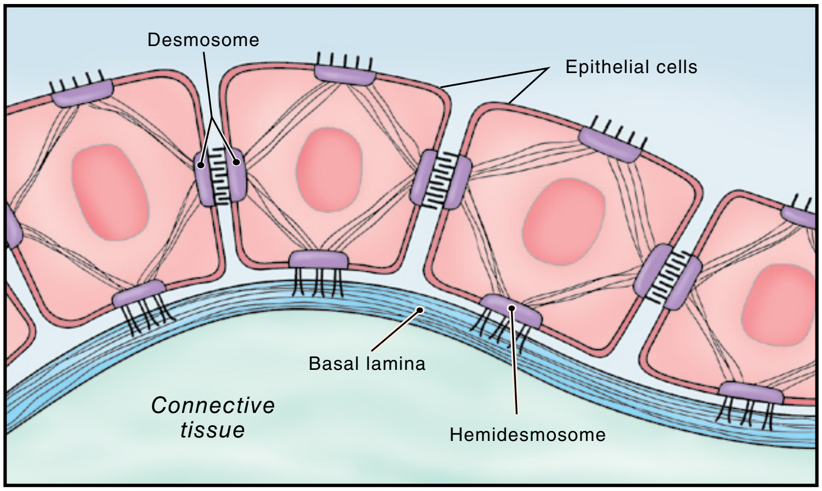
Desmosome
Epithelial cells
Basal lamina
Connective tissue
Hemidesmosome

Figure 2-4B Epithelial Cell Junctions. Epithelial cells attach to each other with specialized cell junctions called desmosomes. Hemidesmosomes attach the epithelial cells to the basal lamina.

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Section 2

Histology of the Gingiva

Knowledge of the microscopic anatomy of the gingiva is a prerequisite for understanding the periodontium in health and in disease. At first glance, the microscopic anatomy of the periodontium may appear complicated.[1] The anatomy of the periodontium, however, is much like that of tissues elsewhere in the body. The gingiva consists of an epithelial layer and an underlying connective tissue layer. This section reviews the microscopic anatomy of the gingival epithelium, junctional epithelium, and gingival connective tissues.

Microscopic Anatomy of Gingival Epithelium

The **gingival epithelium** is a specialized stratified squamous epithelium that functions well in the wet environment of the oral cavity.[2] The microscopic anatomy of the gingival epithelium is like that of the epithelium of the skin. The gingival epithelium may be differentiated into three anatomical areas (**Figure 2-5**):

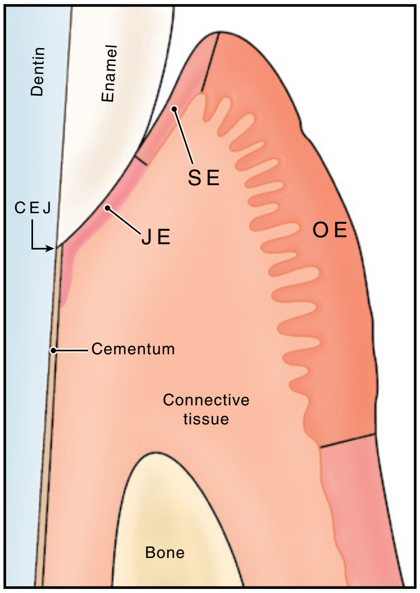

Figure 2-5 Three Areas of the Gingival Epithelium. The gingival epithelium has three distinct areas:

- JE—junctional epithelium at the base of the sulcus that directly attaches to the tooth surface via hemidesmosomes
- SE—sulcular epithelium that lines the sulcus
- OE—oral epithelium covering the free and attached gingiva
- Between the oral epithelial-connective tissue layer, a tight interdigitation interface can be seen. In contrast, between the JE/SE-connective tissue layers, the interface is flush, smooth, and non-interdigitating.

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1. **Oral Epithelium.** The **oral epithelium** covers the outer surface of the free gingiva and attached gingiva; it extends from the crest of the gingival margin to the mucogingival junction. The oral epithelium is the only part of the periodontium that is visible to the unaided eye.
   a. **Cellular Structure of the Oral Epithelium**
      1. The oral epithelium is covered by **keratin**, a tough, fibrous structural protein that protects the outer layer of the oral epithelium.
      2. The oral epithelium is stratified squamous epithelium that can be divided into cell layers (**Figure 2-6**). The layers are listed below in order from the deepest layer to the most superficial layer.
         a. Basal cell layer (stratum basale): cube-shaped cells
         b. Prickle cell layer (stratum spinosum): spine-like cells with large intercellular spaces. The cells of both the basal and prickle cell layers attach to each other via desmosomes.

c. Granular cell layer (stratum granulosum): flattened cells and increased intracellular keratin
d. Keratinized cell layer (stratum corneum): flattened cells with extensive intracellular keratin.

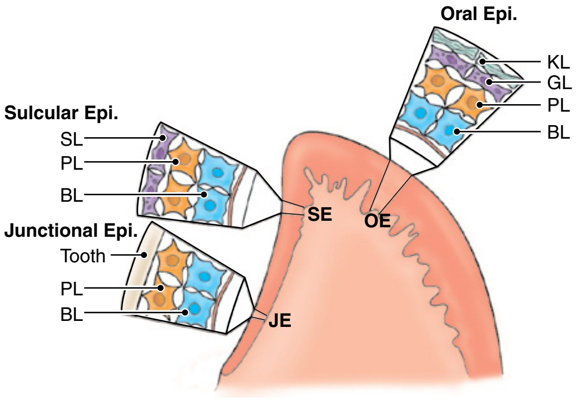
Oral Epi.
KL
GL
PL
BL
Sulcular Epi.
SL
PL
BL
Junctional Epi.
Tooth
PL
BL
SE
OE
JE

Figure 2-6 Cell Layers of the Gingival Epithelium. The cell layers of the oral, sulcular, and junctional epithelium. Illustration key: KL, keratinized cell layer; GL, granular cell layer; SL, superficial cell layer; PL, prickle cell layer; BL, basal cell layer.

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b. **Interface with Gingival Connective Tissue.** In health, oral epithelium tightly interdigitates with the underlying connective tissue. (**Figures 2-7** and **2-8**).

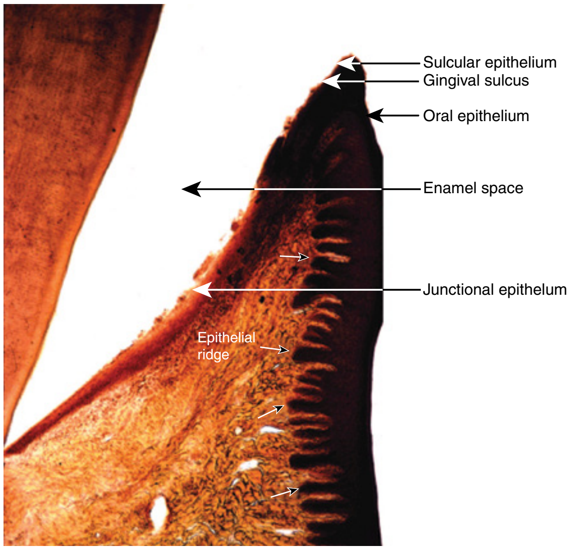
Sulcular epithelium
Gingival sulcus
Oral epithelium
Enamel space
Junctional epithelium
Epithelial ridge

Figure 2-7 Human Gingiva. This photograph shows a decalcified longitudinal section of an incisor tooth as seen through an ordinary light microscope. All the calcium hydroxyapatite crystals have been extracted from the tooth and from its bony alveolus. Because enamel is composed almost completely of calcium hydroxyapatite crystals, only the space where enamel used to be—the enamel space—is represented in this photograph. The sulcular epithelium of the free gingiva borders a space known as the gingival sulcus. Observe the well-defined epithelial ridges (identified by label and arrows) of the oral epithelium; in comparison, note the smooth, flush, non-interdigitating interface between the sulcular epithelium and connective tissue layer and in between the junctional epithelium and connective tissue layer.

Modified with permission from Gartner LP, Hiatt JL. *Color Atlas and Text of Histology*. Lippincott Williams & Wilkins; 2013.
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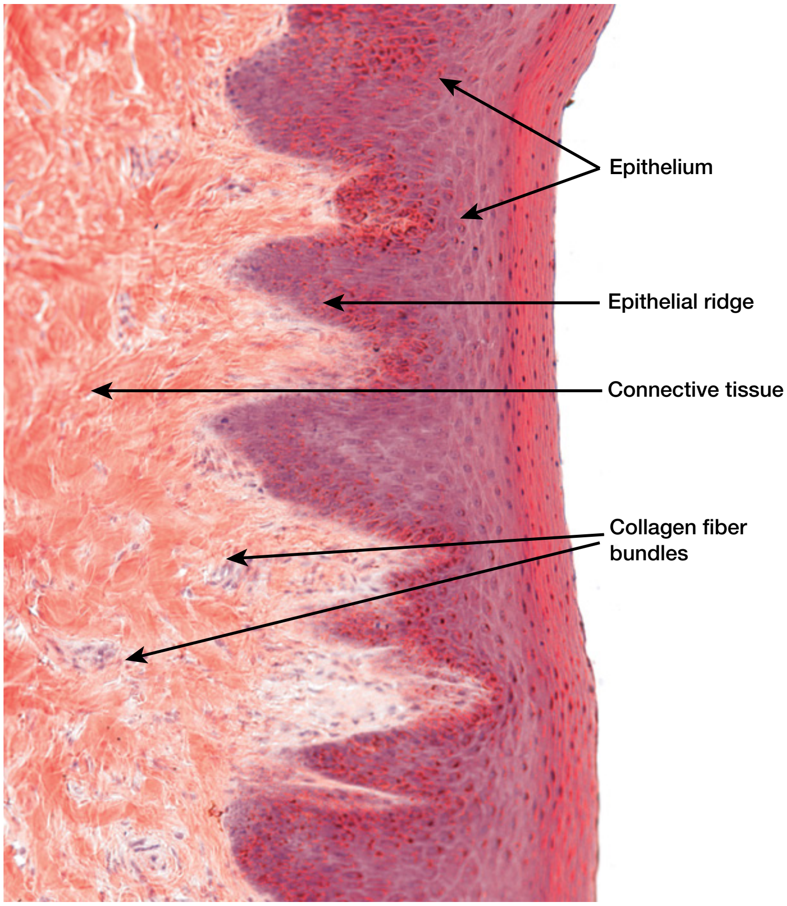

Figure 2-8 Epithelial Ridges. This photograph shows the epithelial–connective junction as seen through an ordinary light microscope. The tall epithelial ridges of the epithelium (in dark red) project into the underlying connective tissue. Collagen fiber bundles are visible in the connective tissue.

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2. **Sulcular Epithelium. Sulcular epithelium** is the epithelial lining of the gingival sulcus. It is continuous with the oral epithelium and extends from the crest of the gingival margin to the coronal border of the junctional epithelium.
   a. **Cellular Structure of the Sulcular Epithelium**
      1. The sulcular epithelium is thin, nonkeratinized epithelium.[3]
      2. The sulcular epithelium has three cellular layers (Figure 2-6):
         a. Basal cell layer
         b. Prickle cell layer
         c. Superficial cell layer: flattened cells without keratin
      3. The sulcular epithelium is permeable, allowing fluid to flow from the gingival connective tissue into the sulcus. This fluid is known as the **gingival crevicular fluid**. The flow of gingival crevicular fluid is slight in health but increases in disease.

  b. **Interface with Gingival Connective Tissue.** In health, the sulcular epithelium joins the connective tissue at a *smooth, flush interface with no epithelial ridges* (no interdigitation junction).

3. **Junctional Epithelium.** **Junctional epithelium** is the specialized epithelium that forms the base of the sulcus and joins the gingiva to the tooth surface. *The gingiva surrounds the cervix of the tooth and attaches to the tooth by means of the junctional epithelium. The base of the sulcus is made up of the coronal-most cells of the junctional epithelium.* In health, the junctional epithelium attaches to the tooth at a level that is slightly coronal to the cementoenamel junction (CEJ).
   a. **Cellular Structure of the Junctional Epithelium**
      1. **Nonkeratinization of the Junctional Epithelium**
         a. Like the sulcular epithelium (but unlike the oral epithelium), the junctional epithelium is thin, nonkeratinized epithelium.
         b. Nonkeratinized epithelial cells of both the sulcular and junctional areas of the gingival epithelium make them a less effective protective covering because of their semipermeable nature. Thus, the sulcular and junctional areas provide the easiest point of entry for bacteria or bacterial products to invade the connective tissue.
      2. The junctional epithelium has only two cell layers:
         a. Basal cell layer
         b. Prickle cell layer
      3. Length and Width of the Junctional Epithelium
         a. The junctional epithelium ranges from 0.71 to 1.35 mm in length.[4]
         b. It is about 15 to 30 cells thick at the coronal zone—the zone that attaches highest on the crown of the tooth.
         c. The junctional epithelium tapers from 4 to 5 cells thick at the apical zone.
   b. **The Junctional Epithelium Interface with the Gingival Connective Tissue.** In health, the junctional epithelium has a smooth flush interface with the connective tissue.

Why the Teeth Need a Junctional Epithelium

1. **The Teeth Create a Break in the Epithelial Protective Covering**
   a. **Protective Epithelial Sheet Covers the Body**
      1. A continuous sheet of epithelium protects the body by covering its outer surfaces and lining the body's cavities, including the oral cavity.
      2. The teeth penetrate this protective covering by erupting through the epithelium, thus creating an opening through which microorganisms can enter the body.
   b. **The Teeth Puncture the Protective Epithelial Sheet**
      1. The body attempts to seal the opening created when a tooth penetrates the epithelium by attaching the epithelium to the tooth.
      2. The word *junction* means "connection"; thus, the epithelium that is connected to the tooth is termed the *junctional epithelium*.
2. **Functions of the Junctional Epithelium**
   a. **Attachment.** The junctional epithelium provides an attachment between the gingiva and the tooth surface, thus providing a seal at the base of the gingival sulcus or periodontal pocket (**Figure 2-9**).
   b. **Barrier.** The junctional epithelium provides a protective barrier between the plaque biofilm and the connective tissue of the periodontium.
   c. **Host Defense.** The epithelial cells play a role in defending the periodontium from bacterial infection by signaling the immune response.[5] The microscopic anatomy of the gingival epithelium–connective tissue interface is illustrated in Figure 2-9.

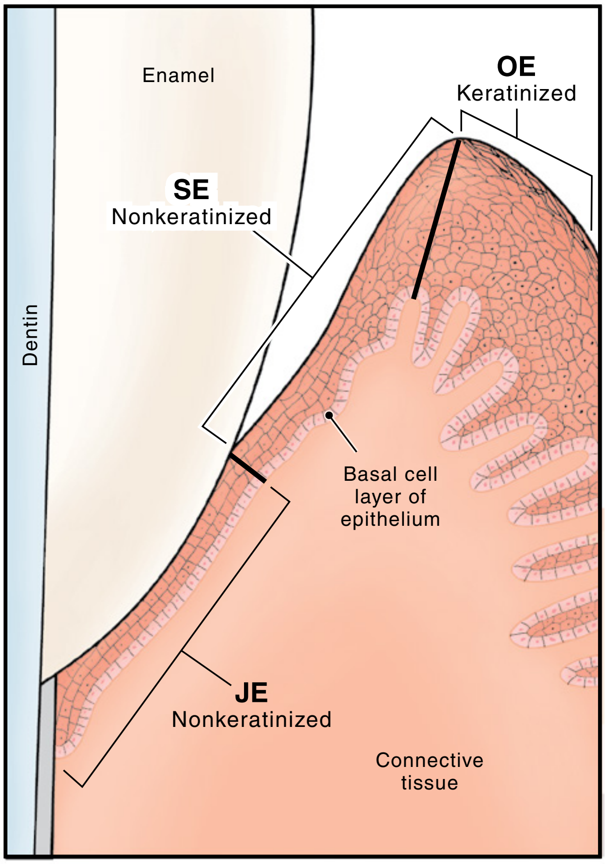

Figure 2-9 Microscopic Anatomy of the Three Areas of the Gingival Epithelium–Connective Tissue Interface.

- OE (oral epithelium)—these epithelial cells form the outer layer of the free and attached gingiva.
- SE (sulcular epithelium)—these epithelial cells extend from the most coronal border of the junctional epithelium to the crest of the gingival margin.
- JE (junctional epithelium)—these epithelial cells join the gingiva to the tooth surface at the base of the sulcus.

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Attachment of the Cells of the Junctional Epithelium

1. **Microscopic Anatomy of Junctional Epithelium**
   a. **Components of the Junctional Epithelium**
      1. **Cells.** The junctional epithelium has an abundance of cells.
         a. Layers of closely packed epithelial cells.
         b. Desmosomes and hemidesmosomes—specialized cell junctions.
      2. **Extracellular Matrix.** The junctional epithelium has a sparse extracellular matrix.
         a. **Internal basal lamina**—a thin basal lamina between the junctional epithelium and the tooth surface.
         b. **External basal lamina**—a thin basal lamina between the junctional epithelium and the gingival connective tissue.

2. **Attachment of Junctional Epithelium to the Tooth Surface**
   a. **Attachment to the Tooth Surface**
      1. The cells of the junctional epithelium attach to the tooth surface via hemidesmosomes and the internal basal lamina.[6–9]
      2. The internal basal lamina is a thin sheet of extracellular matrix adjacent to the tooth surface.
      3. The epithelial cells physically attach to the tooth surface by four to eight hemidesmosomes per micron at the coronal zone and two hemidesmosomes per micron in the apical zone of the junctional epithelium.[10,11] The apical zone is the area of the junctional epithelium with the least adhesiveness.
      4. The attachment of the hemidesmosomes and internal basal lamina to the tooth surface is not static; rather, the cells of the junctional epithelium can move along the tooth surface.

   b. **Attachment to the Underlying Gingival Connective Tissue**
      1. The cells of the junctional epithelium attach to the underlying gingival connective tissue via hemidesmosomes and the external basal lamina.[8,12,13]
      2. In health, the junctional epithelium has a smooth, flush, non-interdigitating interface with the connective tissue. **Figure 2-10** depicts the microscopic anatomy of the junctional epithelium.

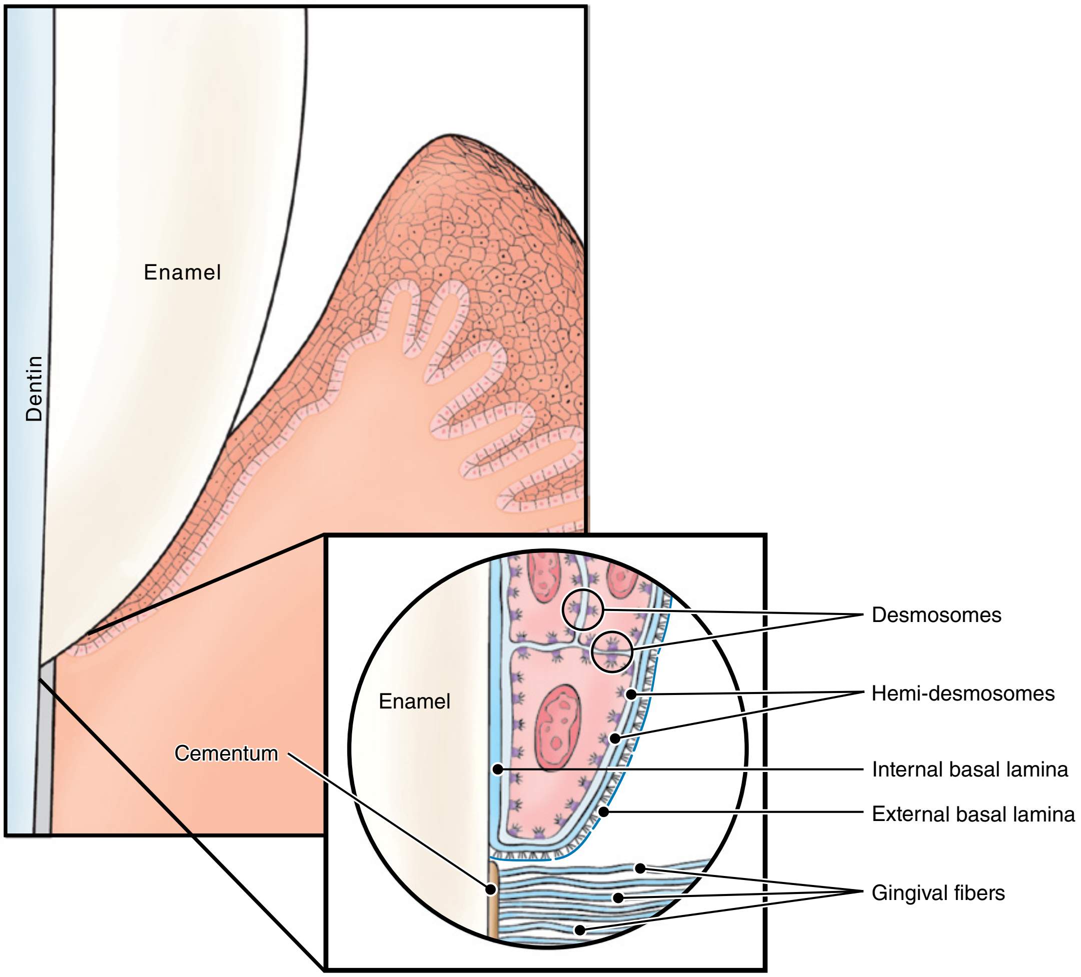

Figure 2-10 Microscopic Anatomy of the Junctional Epithelium. Microscopic structures of the junctional epithelium include the epithelial cells, desmosomes, external and internal basal laminae, and hemidesmosomes.

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Microscopic Anatomy of Gingival Connective Tissue

1. **Function of Gingival Connective Tissue.** The gingival connective tissue of the free and attached gingiva provides solidity to the gingiva and attaches the gingiva to the cementum of the root and the alveolar bone.[14–16] The gingival connective tissue is also known as the lamina propria.
2. **Components of the Gingival Connective Tissue**
   a. **Cells of the Gingival Connective Tissue**
      1. In contrast to the gingival epithelium (which has an abundance of cells and sparse extracellular matrix), the gingival connective tissue has an abundance of extracellular matrix and few cells.
      2. Cells comprise about 5% of the gingival connective tissue.
      3. The different types of cells present in the gingival connective tissue are:
         a. Fibroblasts
         b. Mast cells
         c. Immune cells, such as macrophages, neutrophils, and lymphocytes
      4. The fibers of the connective tissue are produced by the fibroblasts.
   b. **Extracellular Matrix of the Gingival Connective Tissue**
      1. The major components of the connective tissue are collagen fibers, fibroblasts, vessels, and nerves that are embedded in the extracellular matrix. The matrix of the connective tissue is produced mainly by the fibroblasts.
      2. The extracellular matrix is the medium in which the connective tissue cells are embedded and it is essential for the maintenance of the normal function of the connective tissue. The transportation of water, nutrients, metabolites, oxygen, etc. to and from the individual connective tissue cells occurs within the matrix.
      3. Protein fibers account for about 55% to 65% of the gingival connective tissue. Most of these are **collagen fibers** that form a dense network of strong, rope-like cables that secure and hold the gingival connective tissues together.
      4. The collagen fibers enable the gingiva to form a rigid cuff around the tooth.
      5. Gel-like material between the cells makes up about 30% to 35% of the gingival connective tissue. This gel-like material helps to hold the tissue together. The microscopic anatomy of the gingival connective tissue is depicted in **Figure 2-11**.

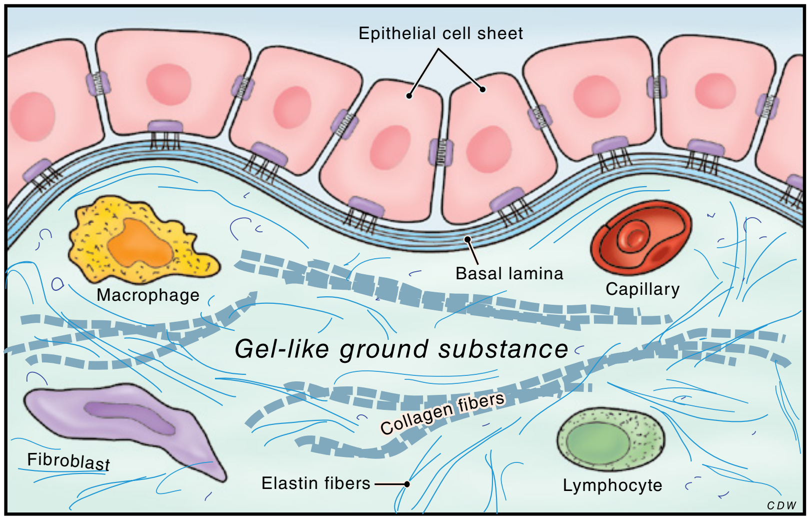

Figure 2-11 Microscopic Anatomy of Gingival Connective Tissue. The gingival connective tissue is composed of a gel-like substance, protein fibers, and cells.

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3. **The Supragingival Fiber Bundles of the Gingival Connective Tissue.** The **supragingival fiber bundles** (**gingival fibers**) are a network of rope-like collagen fiber bundles in the gingival connective tissue. These fibers are located coronal to (above) the crest of the alveolar bone (**Figure 2-12**).

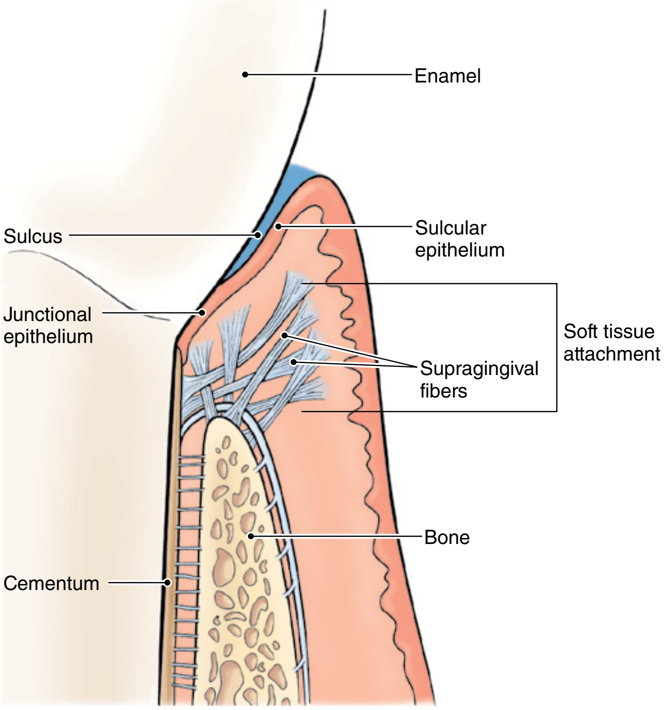

Figure 2-12 Supragingival Fibers of Gingival Connective Tissue. The supragingival fibers are rope-like collagen fiber bundles in the gingival connective tissue. These fibers form a soft tissue attachment coronal to the alveolar bone.

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a. **Features of the Supragingival Fiber Bundles**
   1. The supragingival fiber bundles are embedded in the gel-like extracellular matrix of the gingival connective tissue.
   2. The supragingival fiber bundles strengthen the attachment of the junctional epithelium to the tooth by bracing the gingiva against the tooth surface.
   3. Together the junctional epithelium and the supragingival fibers are referred to as the **dentogingival unit**. The dentogingival unit provides structural support to the gingival tissue.

b. **Functions of the Supragingival Fiber Bundles**
   1. Brace the gingiva firmly against the tooth and reinforce the attachment of the junctional epithelium to the tooth.
   2. Provide the gingiva with the rigidity needed to withstand the masticatory (chewing) forces.
   3. Connect the gingiva to the underlying cementum and alveolar bone.
   4. Stabilize adjacent teeth to one another so as to maintain tooth positioning within the dental arch.

c. **Classification of Supragingival Fiber Groups.** The supragingival fiber bundles are classified based on their orientation, sites of insertion, and the structures that they connect (**Figures 2-13** and **2-14**).
   1. Alveologingival fibers—extend from the periosteum of the alveolar crest into the gingival connective tissue. These supragingival fibers attach the gingiva to the bone. (The **periosteum** is a dense membrane composed of fibrous connective tissue that closely wraps the outer surface of the alveolar bone.)
   2. Circular fibers—encircle the tooth in a ring-like manner coronal to the alveolar crest and are not attached to the cementum of the tooth.
   3. Dentogingival fibers—embed in the cementum near the CEJ and fan out into the gingival connective tissue. These fibers act to attach the gingiva to the teeth.
   4. Periosteogingival fibers—extend laterally from the periosteum of the alveolar bone. These fibers attach the gingiva to the bone.
   5. Intergingival fibers—extend in a mesiodistal direction along the entire dental arch and around the last molars in the arch. These fiber bundles link adjacent teeth into a dental arch unit.
   6. Intercircular fibers—encircle several teeth. These fiber groups link adjacent teeth into a dental arch unit.

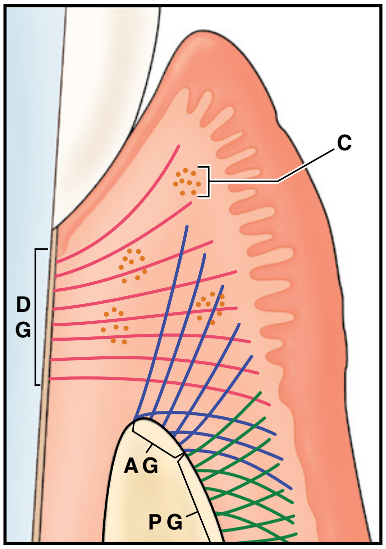

Figure 2-13 Supragingival Fiber Groups. (Cross-Sectional View, a view which cuts parallel to the long axis of the tooth to show the internal anatomy of the supragingival fiber groups.)

C—circular
AG—alveologingival
DG—dentogingival
PG—periosteogingival

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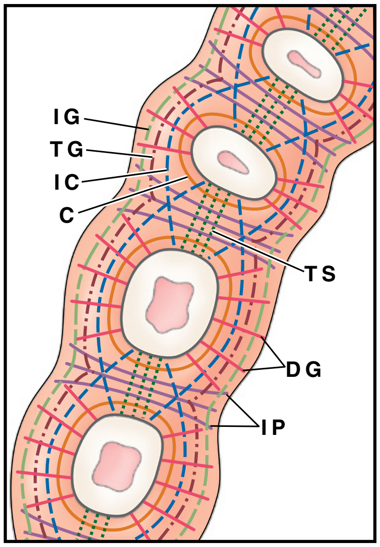

Figure 2-14 Supragingival Fiber Groups of the Mandibular Arch. (Occlusal View, Looking Down on the Mandibular Arch.)

C—circular
IG—intergingival
IC—intercircular
IP—interpapillary
DG—dentogingival
TG—transgingival
TS—transseptal

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7. Interpapillary fibers—located in the papillae coronal to (above) the transseptal fiber bundles. These fiber groups connect the oral and vestibular interdental papillae of posterior teeth.
8. Transgingival fibers—extend from the cementum near the CEJ and run horizontally between adjacent teeth. These fiber bundles link adjacent teeth into a dental arch unit.
9. Transseptal fibers—pass from the cementum of one tooth, over the crest of alveolar bone, to the cementum of the adjacent tooth. These fiber bundles connect adjacent teeth to one another and secure alignment of teeth in the arch.

4. **The Periodontal Ligament Fibers of the Gingival Connective Tissue**
   a. **Definition.** The **periodontal ligament** is a thin sheet of fibrous connective tissue that surrounds the roots of the teeth and joins the root cementum to the socket wall. The thickness of the periodontal ligament space ranges from 0.05 to 0.25 mm depending on the age of the patient and the function of the tooth.[17,18]
   b. **Components of the Periodontal Ligament.** The periodontal ligament consists of connective tissue fibers, cells, and extracellular matrix.
      1. **Cells.** The cells of the periodontal ligament are mainly fibroblasts with some cementoblasts and osteoblasts.
      2. **Extracellular Matrix**
         a. The extracellular matrix of the periodontal ligament is similar to the extracellular matrix of other connective tissue. This rich gel-like substance contains specialized connective fibers.
         b. **Fiber Bundles.** The **fiber bundles of the periodontal ligament** are a specialized connective tissue that surrounds the root of the tooth and connects it to the alveolar bone. These fibers are the largest component of the periodontal ligament space.
            1. The rope-like collagen fiber bundles of the periodontal ligament stretch across the space between the cementum and the alveolar bone of the tooth socket.
            2. The collagen fiber bundles are anchored on one side in the cementum covering the tooth root; on the other side, they are embedded in the bone of the tooth socket.
         c. **Blood Vessels and Nerve Supply.** The periodontal ligament has a rich supply of nerves and blood vessels.

c. **Functions of the Periodontal Ligament**
   1. Supportive function—the major function of the periodontal ligament is to anchor the tooth to its bony socket and to separate the tooth from the socket wall so that the root does not traumatize the bone during mastication.
   2. Sensory function—the periodontal ligament is supplied with specialized nerve endings that transmit tactile pressure (such as a tap with dental instrument against tooth) and pain sensations from the tooth and surrounding attachment apparatus to the central nervous system.
   3. Nutritive function—the periodontal ligament is supplied with blood vessels that provide nutrients to the cementum and bone.
   4. Formative function—within the periodontal ligament space resides many different types of cells, such as fibroblasts which produce the collagen that is the major component of the periodontal ligament; cementoblasts ("cementum builders") which produce cementum throughout the life of the tooth; osteoblasts ("bone builders") which maintain the bone of the tooth socket; epithelial cells which may be latent or active remnants of Hertwig's epithelial root sheath (known as epithelial rests of Malassez) which may play a role in periodontal repair or regeneration; and undifferentiated cells which may be a source of new cells after depletion of old cells that have undergone programmed cell death and hold the potential of being an important source of cells for periodontal regeneration.
   5. Resorptive function—in response to severe pressure, cells of the periodontal ligament (osteoclasts) can induce rapid bone resorption and, sometimes, resorption of cementum.

d. **Principal Fiber Groups of the Periodontal Ligament.** The tooth is joined to the bone by bundles of collagen fibers that can be divided into the five groups based on their location and orientation (**Figure 2-15**).

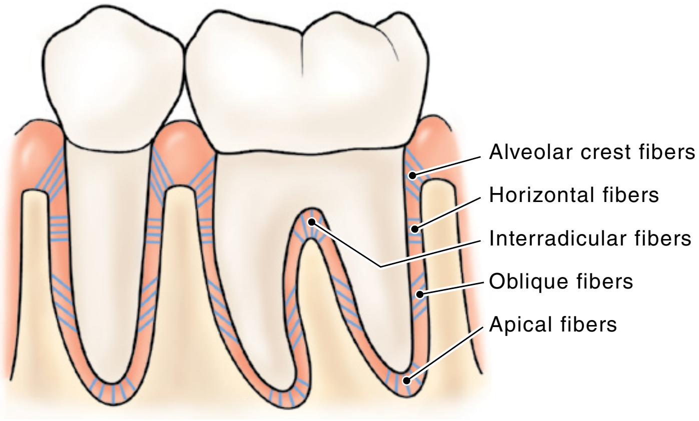

Figure 2-15 Principal Fiber Groups of the Periodontal Ligament. The fibers of the periodontal ligament are classified as the alveolar crest, horizontal, interradicular, oblique, and apical.

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1. Alveolar crest fiber group—extend from the cervical cementum, running downward in a diagonal direction, to the alveolar crest. This fiber group resists horizontal movements of the tooth and prevents tooth extrusion.
2. Horizontal fiber group—located apical to the alveolar crest fibers. These fibers extend from the cementum to the bone at right angles to the long axis of the root. This fiber group resists horizontal pressure against the crown of the tooth.
3. Oblique fiber group—located apical to the horizontal group. These fibers extend from the cementum to the bone, running in a diagonal direction. This fiber group resists vertical pressures that threaten to drive the root into its socket.
4. Apical fiber group—extend from the apex of the tooth to the bone. This fiber group secures the tooth in its socket and resists forces that might lift the tooth out of the socket.
5. Interradicular fiber group (present only in the furcation region of multirooted teeth)—extend from the cementum in the furcation area of the tooth to the interradicular septum of the alveolar bone. These fibers help to stabilize the tooth in its socket.

e. Sharpey's Fibers of the Periodontal Ligament

1. The calcified terminal ends of the periodontal ligament fibers that are embedded in the cementum and alveolar bone are known as **Sharpey's fibers** (**Figure 2-16**).

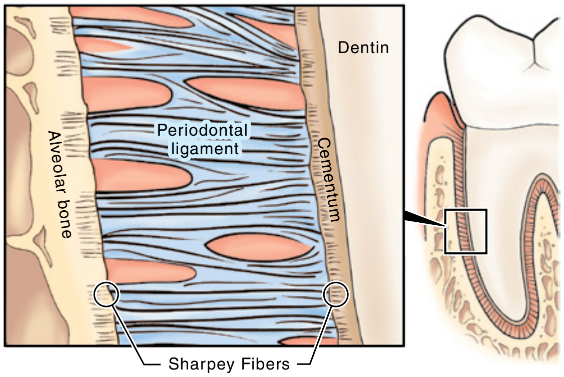
Dentin
Periodontal ligament
Alveolar bone
Cementum
Sharpey Fibers

Figure 2-16 **Sharpey's Fibers.** The terminal ends of the periodontal ligament fibers that are embedded in the alveolar bone and the cementum are known as Sharpey's fibers.

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2. The attachment of the fiber bundles occurs when the cementum and bone are forming. As cementum forms, the tissue calcifies around the ends of the periodontal ligament fibers (Sharpey's fibers), surrounding them with cementum. The same process occurs during bone formation. As the bony wall of the tooth socket calcifies, it surrounds the ends of the periodontal ligament fibers with bone. The ends of the fiber bundles become trapped in the bone that forms around them (**Figure 2-17**).

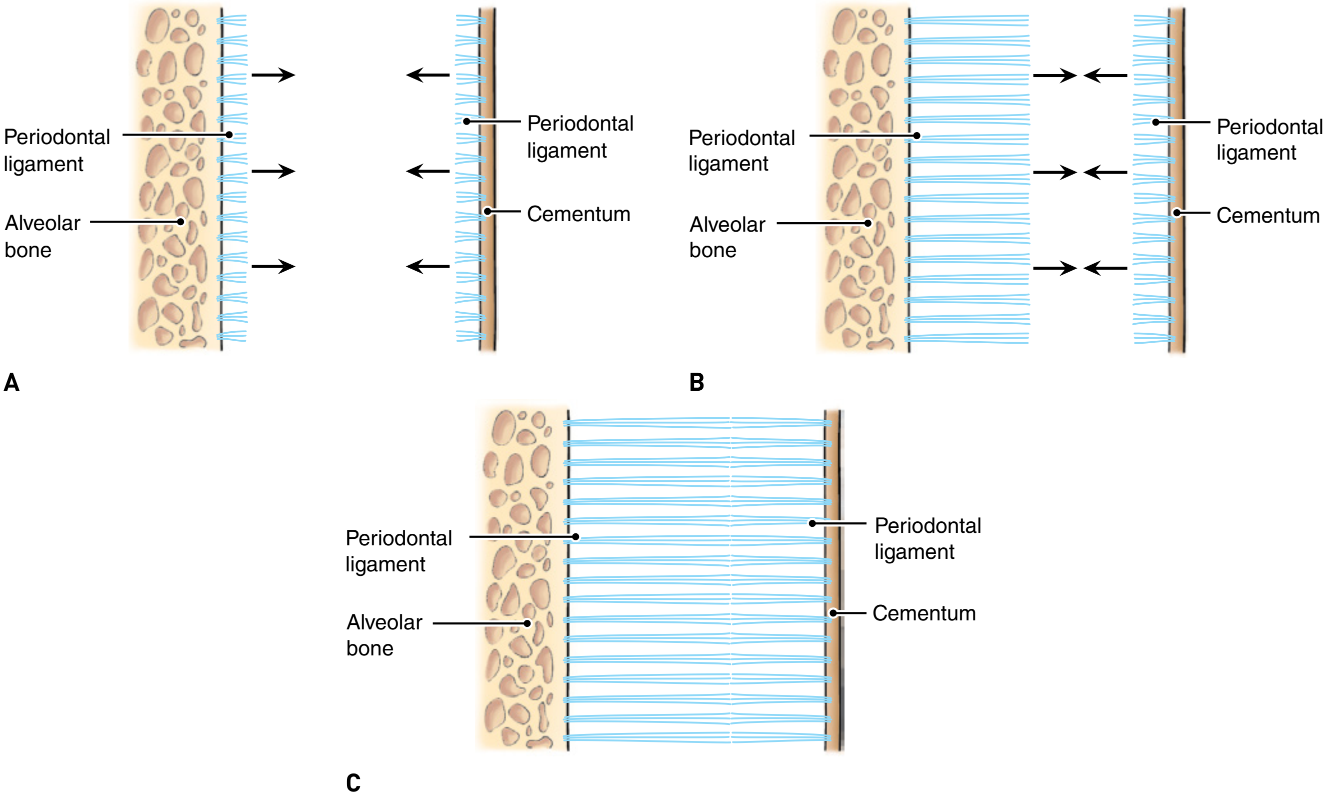
Periodontal ligament
Alveolar bone
Periodontal ligament
Cementum
A
Periodontal ligament
Alveolar bone
Periodontal ligament
Cementum
B
Periodontal ligament
Alveolar bone
Periodontal ligament
Cementum
C

Figure 2-17 **Development of the Periodontal Ligament Fibers.** Figures **A**, **B**, and **C** depict the stages in the development of the periodontal ligament between the alveolar bone and the cementum of the tooth root.

A. Fine collagen fibers arise from the root cementum. Similarly, collagen fibers arise from the alveolar bone proper.
B. The fibers grow into the midportion of periodontal ligament space.
C. The fibers from the root cementum fuse with fibers from the alveolar bone proper.

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Section 3

Histology of Root Cementum and Alveolar Bone

Section 3 reviews the microscopic anatomy of the cementum and alveolar bone. Knowledge of the microscopic anatomy of these structures is a prerequisite to understanding the function of these structures in health and the alterations in disease.

Microscopic Anatomy of Cementum

1. **Definition.** **Cementum** is a mineralized tissue that covers the roots of the teeth and serves to attach the tooth to alveolar bone via collagen fibers of the periodontal ligament (**Figure 2-18**). Anatomically, cementum is part of the tooth; however, functionally, it is part of the periodontium.

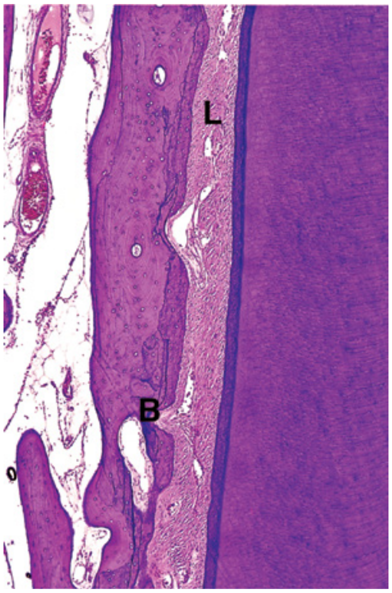

Figure 2-18 **Cementum and Tooth Supporting Structures.** A thin layer of cementum (appearing as a thin blue band) covers the dentin of the root. The periodontal ligament **(L)** holds the tooth in the bony socket of the alveolar bone **(B)**.

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   a. **Functions of Cementum**
      1. The prime function of cementum is to attach the periodontal ligament fibers to the root of the tooth.
      2. In addition, cementum maintains the integrity of the root, helps to maintain the tooth in its functional position in the mouth, and is involved in tooth repair and regeneration.
         a. Cementum seals and covers the open dentinal tubules and acts to protect the underlying dentin.
         b. Cementum is a metabolically active tissue that undergoes continual resorption and reformation (remodeling) throughout the lifetime of the tooth.
            1. Constant cementum remodeling facilitates the continuous reattachment of the periodontal ligament fibers to the root.
            2. Cementum continues to grow in thickness throughout life to compensate for attrition of teeth at their occlusal or incisal surfaces.[19] Cementum continuously forms at the apical areas of the roots to compensate for loss of tooth tissues due to attrition and maintains the length of the root.

        3. Cementum is a key component of periodontal tissues, and its preservation is of paramount importance for the quality of healing at completion of periodontal treatment. Periodontal reattachment or new attachment as an end-result of therapy strongly relies on the presence of healthy and intact cementum surface following root instrumentation.[20]
        4. Cementum may influence the activities of various types of periodontal cells. It is believed that molecules stored in the cementum matrix may induce periodontal ligament regeneration when needed.[20]
    b. **Components of Mature Cementum.** As in bone and dentin, the major organic component of cementum is collagen. Cementum contains collagen fibers embedded in an organic matrix.[21]
        1. **Organic Portion**
            a. The organic matrix of cementum is composed of a framework of densely packed collagen fibers held together by the gel-like extracellular ground substance. These fibers are oriented more or less parallel to the long axis of the tooth.
            b. In addition to collagens, groups of proteins are present in cementum.
                1. Recent research suggests that these **cementum proteins** may have an important role in regulating the mineralization process associated with cementum formation.[22]
                2. There is still a great deal to learn about cementum proteins. From the current status of knowledge, it appears that cementum proteins may play an important role in future therapies to achieve regeneration of the periodontal structures.[22]
        2. **Mineralized Portion.** The mineralized portion of cementum is made up of hydroxyapatite crystals (calcium and phosphate).
        3. **Vessels and Innervation.** Cementum does not contain blood vessels or nerves. (Hypersensitivity of the root surface occurs when the cementum is removed exposing the dentin. It is the dentin, rather than the cementum, that is sensitive to brushing, the touch of a dental instrument, or sudden temperature changes in the mouth (such as drinking hot coffee while eating ice cream).
        4. **Biologic Components.** The periodontium has been shown to contain biologically active mediators,[21,23–26] and these molecules are elevated in alveolar bone and cementum.[26–29] It is believed that growth factor molecules are produced during cementum formation and then stored in the cementum matrix to induce periodontal ligament regeneration when needed.
    c. **Conservation of Cementum During Periodontal Instrumentation.** Subgingival instrumentation during periodontal therapy results in the removal of root cementum, which can eventually lead to exposure of underlying dentin, pulp injury, and dentin hypersensitivity.[30]
        1. **Historical Perspective.** Previously, it was theorized that bacterial products penetrate the cementum of periodontally diseased root surfaces. This concept resulted in the intentional, aggressive removal of all or most cementum during periodontal instrumentation of root surfaces.[31,32] Overzealous instrumentation can result in removal of all cementum, which can result in exposure of underlying dentin, tooth sensitivity, or even external root resorption. More specifically, in the past the goal of periodontal therapy was to obtain a treated root surface with smooth and hard surface characteristics that was free of endotoxins.[31,33]
        2. **Current Research**
            a. More recent studies show that bacterial products are not located within cementum[34,35] and that removal of cementum is not necessary for a successful periodontal outcome.[36]
            b. The preservation of cementum on the root surface is further supported by Saygin et al., who report that cementum is necessary for new attachment and as a source of growth factors.[21,37]
            c. Grzesik and Narayanan suggested that cementum plays an important regulatory role in periodontal regeneration.[38] From these studies, it can be concluded that nonaggressive removal of cementum is necessary for optimal periodontal health as well as for periodontal regeneration.
2. **Types of Cementum.** Functional, morphological, and histological differences appear to exist along the length of the root.[1,21,39] Cementum is classified as follows: intermediate, acellular, and cellular cementum (**Figure 2-19**).

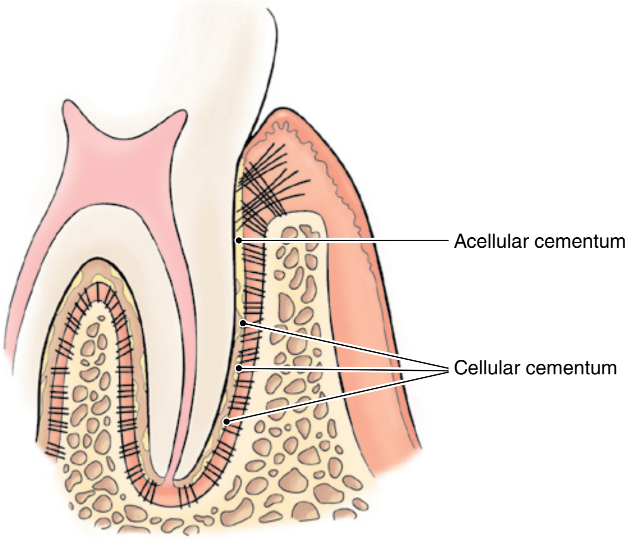

Figure 2-19 **Types of Cementum.** Acellular cementum covers approximately the cervical third or half of the root. New acellular cementum normally is not produced after the tooth has reached its occlusal plane. Cellular cementum covers the apical half of the root. It is continuously deposited throughout the life of the tooth after it has reached its occlusal plane and increases in thickness with age.

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a. **Intermediate Cementum.** Cementum located at the cementoenamel junction.
b. **Acellular (Primary) Cementum.** Acellular cementum attaches the tooth to the surrounding alveolar bone via periodontal ligament fibers.
    1. Contains no living cells within its mineralized tissue (no cementocytes).
    2. First cementum to be formed and covers approximately the cervical third or half of the root.
    3. No new acellular cementum is produced after the tooth reaches the occlusal plane
    4. Thickness ranges from 30 to 230 microns.
    5. Sharpey's fibers make up most of the structure of acellular cementum.
c. **Cellular (Secondary) Cementum.** Cellular cementum is distinguished by the presence of living cells in its structure.
    1. Contains cementocytes and fibroblasts within its mineralized tissue.
    2. Present in the apical and interradicular portions of the root.
    3. Formed after the tooth reaches the occlusal plane and is less calcified than acellular cementum.
    4. Deposited in intervals throughout the life of the tooth (thickness increases with age, where its increased thickness compensates for tooth attrition).
    5. Thickness ranges from 150 to 200 microns.
    6. Sharpey's fibers make up a smaller portion of cellular cementum.

3. **Relationship of Cementum to Enamel at the CEJ.** The cementum covering the root may have any one of three relationships with the enamel of the tooth crown (**Figure 2-20**).
    a. **Three Possible Arrangements of Enamel and Cementum**
        1. Overlap—the cementum overlaps the enamel for a short distance.
        2. Meet—the cementum meets the enamel.

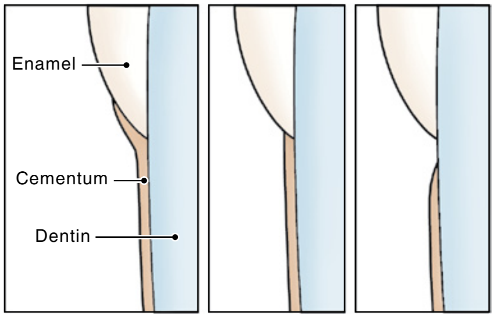

Figure 2-20 Three Patterns for the Arrangement of Cementum to Enamel at the Cementoenamel Junction. The cementum may **(1)** overlap the enamel, **(2)** meet the enamel, or **(3)** not meet, leaving a gap between the cementum and enamel.

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3. Gap—there is a small gap between the cementum and enamel (exposing the dentin in this area). The patient may experience discomfort (dentinal sensitivity) during instrumentation. The use of local anesthesia may be helpful during instrumentation, and desensitization of sensitive areas should be performed following instrumentation.

b. These three relationships commonly are abbreviated as **OMG (Overlap, Meet, Gap)**.

c. In any one tooth, all three arrangements of the junction between the cementum and enamel may be present.[19,21]

Microscopic Anatomy of Alveolar Bone

1. **Definition.** The **alveolar process**, or alveolar bone, is the part of the maxilla and mandible that forms and supports the sockets of the teeth (**Figures 2-21** and **2-22**).

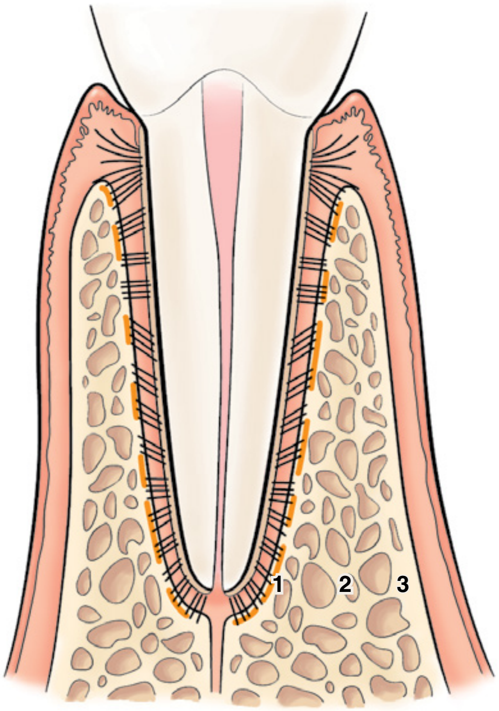

Figure 2-21 Anatomy of Alveolar Bone. **(1)** Alveolar bone proper, **(2)** trabecular bone, and **(3)** cortical (compact) bone.

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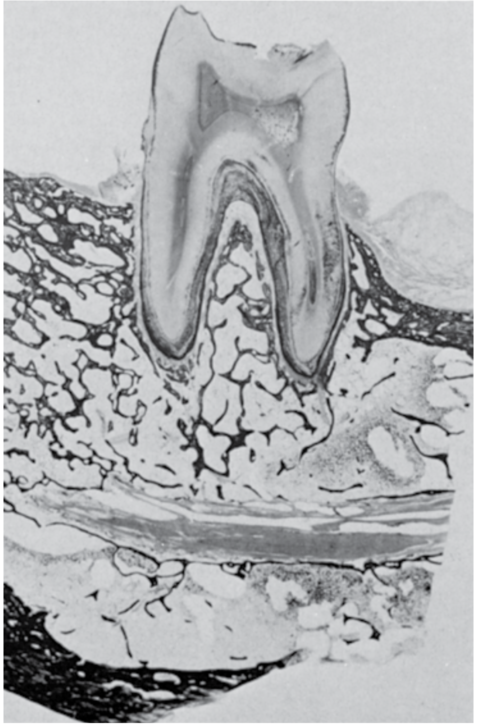

Figure 2-22 Histology of Alveolar Bone. A histologic section through a mandibular first molar and its alveolar process.

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2. **Function of Alveolar Bone in the Periodontium**
    a. **Protects Roots of Teeth.** The alveolar bone forms the bony sockets that provide support and protection for the roots of the teeth.
    b. **Changes in Response to Mechanical Forces and Inflammation.** Alveolar bone constantly undergoes periods of bone formation and resorption (loss) in response to mechanical forces on the tooth and inflammation of the periodontium. This process of bone formation and resorption is known as **bone remodeling**.
3. **Characteristics of Alveolar Bone**
    a. **Components.** Alveolar bone is mineralized connective tissue made by cells called osteoblasts ("bone builders").[21]
        1. **Major Cell Types**
            a. Osteoblasts—bone-formers—cells that produce the bone matrix consisting of collagen fibers and other protein fibers.
            b. Osteoclasts—bone consumers—cells that remove the mineral materials and organic matrix of alveolar bone.
        2. **Extracellular Matrix**
            a. Collagen fibers and gel-like substance form the major component of the alveolar bone.
            b. The bone matrix is rigid because it undergoes mineralization by the deposition of minerals such as calcium and phosphate, which are subsequently transformed into hydroxyapatite.
    b. **Vessels and Innervation.** Unlike cementum, the alveolar bone has blood vessels and nerve innervation.

Chapter Summary Statement

Knowledge of the microscopic anatomy of the periodontium is fundamental in understanding (1) the function of the periodontium in health and (2) the changes that occur during the periodontal disease process. The junctional epithelium plays an important role in the health of the periodontium by attaching the gingival epithelium to the tooth via hemidesmosomes and an internal basal lamina. In health, the periodontal ligament, cementum, and alveolar bone act as a functional unit to support and maintain the teeth in the oral cavity.

Section 4

Focus on Patients

Clinical Patient Care

CASE 1

A clinician penetrates the oral mucosa with a needle before injecting a local anesthetic. The needle tip stops in the loose connective tissue underlying the surface structures. Name the layers of epithelium that have been penetrated by the needle.

CASE 2

A clinician finds it necessary to use a unique type of injection to achieve total anesthesia of a tooth being treated. The injection involves sliding a small-diameter needle into the periodontal ligament space to a point halfway down the tooth root. Name the periodontal ligament fibers most likely encountered by the needle tip during insertion.

CASE 3

Recession of the gingival margin exposes a portion of tooth root on a maxillary canine tooth. Microscopic examination of the cementum in the area of the crown margin on the canine will reveal what possible relationships between the level of cementum and the level of the tooth crown?

Evidence in Action: Clinical Relevance

Conservation of Cementum

Cementum is a key component of periodontal tissues, and its preservation is of paramount importance for the quality of healing at the completion of periodontal treatment. Conservation of cementum is ideal because loss of cementum is accompanied by exposure of the dentinal tubules and by a loss of attachment of periodontal ligament fibers to the root surface. Periodontal reattachment or new attachment as an end-result of therapy strongly relies on the presence of cementum after root instrumentation.

Research in the past 20 years has greatly advanced understanding of the cellular and molecular events involved in the developing periodontium. As understanding of the structure, function, and composition of cementum increases, so does the potential for new therapies for periodontal regeneration using molecules formed by these tissues.

Improper or aggressive periodontal instrumentation may reduce the thickness or eventually remove all the cementum over the root surface leading to a loss of growth factor reservoirs in the cementum. For this reason, it is strongly suggested that the root surface instrumentation should aim not only at thorough removal of calculus deposits but also at preserving root substance to improve healing following periodontal therapy.

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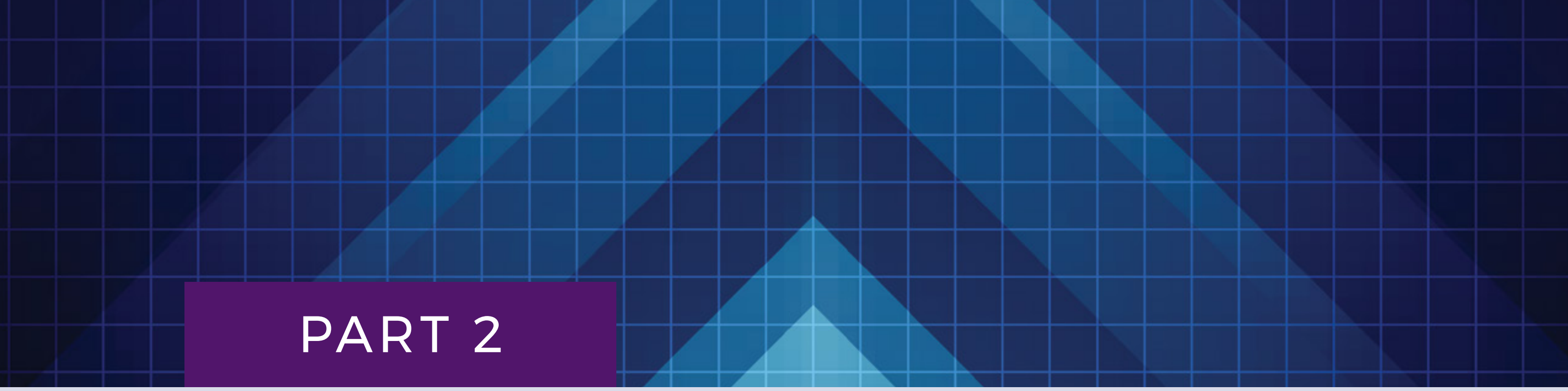

PART 2

Diseases Affecting the Periodontium and Peri-Implant Tissues

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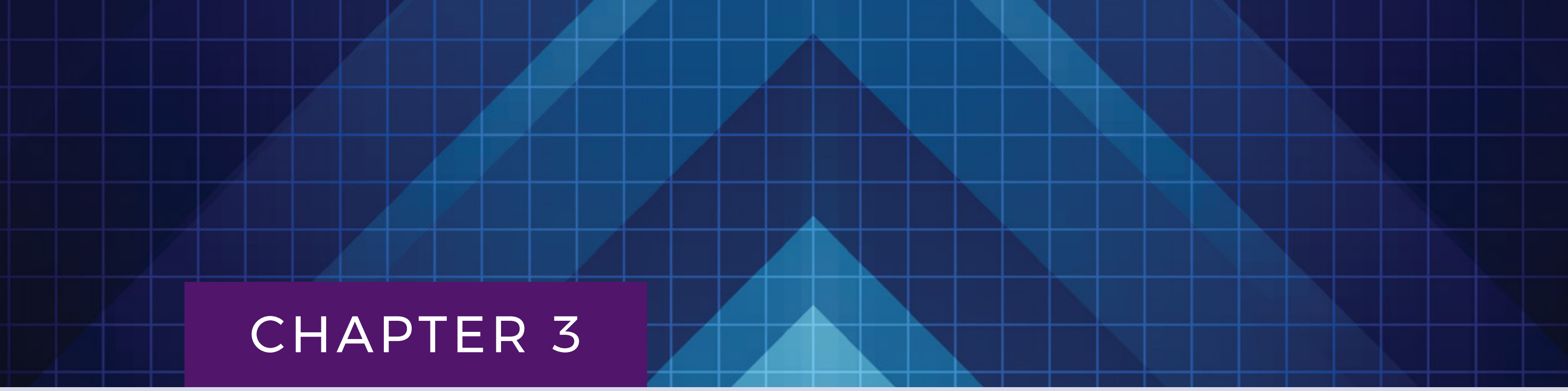

CHAPTER 3

Overview of Diseases of the Periodontium

- Section 1 The Periodontium in Health and Disease
  - Three Basic States of the Periodontium
  - Periodontium in Health
  - Gingivitis—Reversible Tissue Damage
  - Periodontitis—Permanent Tissue Destruction
- Section 2 Pathogenesis of Bone Destruction
  - Changes in Alveolar Bone Height in Disease
  - Patterns of Bone Loss in Periodontitis
- Section 3 Periodontal Pockets
  - Characteristics of Periodontal Pockets
  - Pocket Formation
- Section 4 Theories of Disease Progression
- Section 5 Epidemiology of the Diseases of the Periodontium
- Section 6 Focus on Patients
  - Clinical Patient Care

CLINICAL APPLICATION

The term *periodontal disease* encompasses three broad categories of conditions, namely: (1) gingival diseases and conditions, (2) periodontitis, and (3) other conditions affecting the periodontium. The complexities of each of these diseases and conditions will be highlighted in detail in subsequent chapters of this book. This chapter introduces the reader to the fundamental features and characteristics that distinguish the healthy periodontium from the diseased periodontium. Additionally, concepts related to disease epidemiology, pathogenesis, and progression of periodontal diseases will be discussed in this chapter.

LEARNING OBJECTIVES

- Define the term *disease progression*.
- Define the term *periodontal disease* and contrast it with the term *periodontitis*.
- Compare and contrast (1) the position of the junctional epithelium; (2) the characteristics of the epithelial–connective tissue junction; and (3) the position of the crest of the alveolar bone in health, gingivitis, and periodontitis.
- Explain why there is a band of intact transseptal fibers even in the presence of severe bone loss.
- Describe the progressive destruction of alveolar bone loss that occurs in periodontitis.
- Describe the pathway of inflammation that occurs in horizontal bone loss and contrast it with the pathway of inflammation that occurs in vertical bone loss.
- Compare and contrast the characteristics of gingival and periodontal pockets.
- Be able to clinically identify the visible signs of periodontal health and periodontal disease.

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- In a clinical setting, for a patient with periodontal disease, measure the probing depth of the sulci (pockets) on the facial aspects of all teeth in one sextant of the mouth. Using the information gathered visually and with the periodontal probe, explain whether this patient's disease is gingivitis or periodontitis.
- Given a drawing of a periodontal pocket, determine whether the pocket illustrated is a suprabony or infrabony pocket.
- Describe variables associated with periodontal disease that an epidemiologist might include in a research study.
- Define *prevalence* and *incidence* as measurements of disease within a population.
- Describe how clinical dental hygiene practice can be affected by epidemiological research.

KEY TERMS

Disease progression
Gingivitis
Reversible tissue damage
Periodontitis
Irreversible tissue damage
Apical migration of the junctional epithelium
Pathologic tooth migration
Inflammation
Alveolar bone loss
Horizontal bone loss
Vertical bone loss
Osseous defect
Infrabony defect
Suprabony defect
Osseous crater
Furcation involvement
Attachment loss
Disease site
Inactive disease site
Active disease site
Gingival pocket
Periodontal pocket
Suprabony pocket
Infrabony pocket
Continuous disease progression theory
Intermittent disease progression theory
Epidemiology
Incidence
Prevalence
National Health and Nutrition Examination Survey (NHANES)
Centers for Disease Control and Prevention (CDC)

Section 1

The Periodontium in Health and Disease

Three Basic States of the Periodontium

Disease progression (pathogenesis) is the sequence of events that occur during the development of a disease or abnormal condition. The periodontium can exist in one of three basic states: health, gingivitis, or periodontitis (**Figure 3-1**). It is therefore important that the clinician be able to recognize the distinguishing features of gingival health,

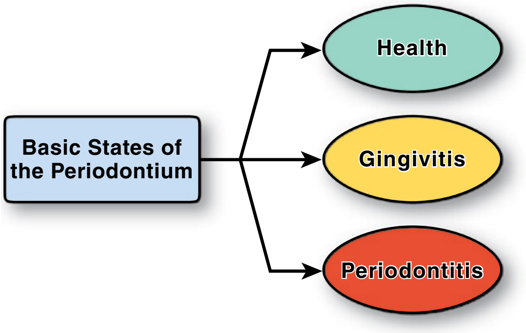

Figure 3-1 Three Basic States of the Periodontium. In the absence of disease, the periodontium is healthy. In the presence of disease, however, the periodontium may fall into either the gingivitis or periodontitis categories.

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gingivitis, and periodontitis (**Table 3-1**). This section provides an overview of these three basic states at the clinical and microscopic levels (**Figures 3-2** to **3-4**). Gingivitis and periodontitis are the two basic categories of periodontal disease that effect the tooth-supporting periodontal structures.[1–4]

- Gingivitis is a bacterial infection that is confined to the gingiva. The tissue damage that occurs in gingivitis is reversible if the plaque biofilm is removed and the ensuing inflammation is abated (Figure 3-3).
- Periodontitis is a bacterial infection affecting all parts of the periodontium, including the gingiva, periodontal ligament, bone, and cementum. The tissue damage that occurs in periodontitis results in irreversible destruction to the tissues of the periodontium (Figure 3-4).
- The term *periodontal disease* should not be confused with the term *periodontitis* (as periodontitis is one of the many types of periodontal disease).

Table 3-1 **Histologic Changes in Gingivitis and Periodontitis**

State	Junctional Epithelium	Connective Tissue Attachment	Periodontal Ligament Fibers	Alveolar Bone
Health	JE coronal to CEJ Tight intercellular junctions	Intact; supragingival fiber bundles provide support to gingiva and JE	Intact; attach root to the bone of the tooth socket	Intact; supports and protects root of tooth
Gingivitis	JE at CEJ Widened intercellular junctions; epithelial extensions into connective tissue	Connective tissue damage	Intact	Intact
Periodontitis	JE apical to CEJ Widened intercellular junctions; epithelial extensions into connective tissue	Destruction of supragingival fiber bundles	Destruction of periodontal ligament fibers; exposure of cementum to pocket environment	Destruction of bone; eventual tooth loss

JE, junctional epithelium; CEJ, cementoenamel junction.

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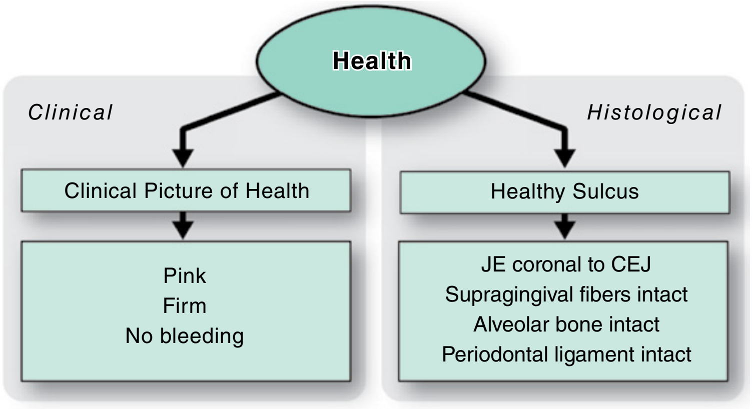

Figure 3-2 **Characteristics of Healthy Periodontium.** The clinical and histologic characteristics of the tissues of the periodontium in health.

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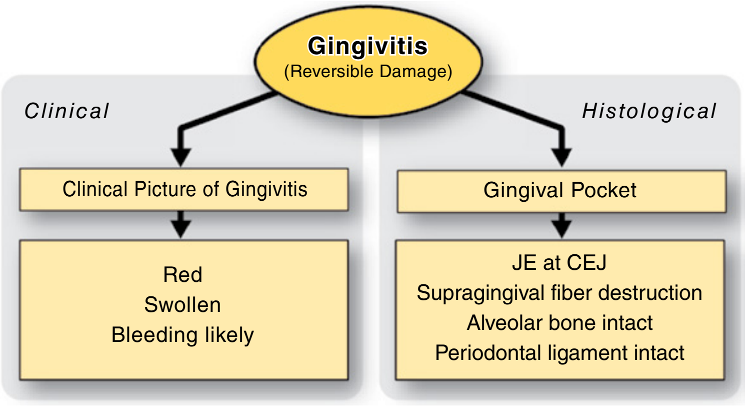
Gingivitis
(Reversible Damage)
Clinical
Histological
Clinical Picture of Gingivitis
Red
Swollen
Bleeding likely
Gingival Pocket
JE at CEJ
Supragingival fiber destruction
Alveolar bone intact
Periodontal ligament intact

Figure 3-3 **Characteristics of Gingivitis.** The clinical and histologic characteristics of gingivitis. Some reversible tissue damage occurs in gingivitis.

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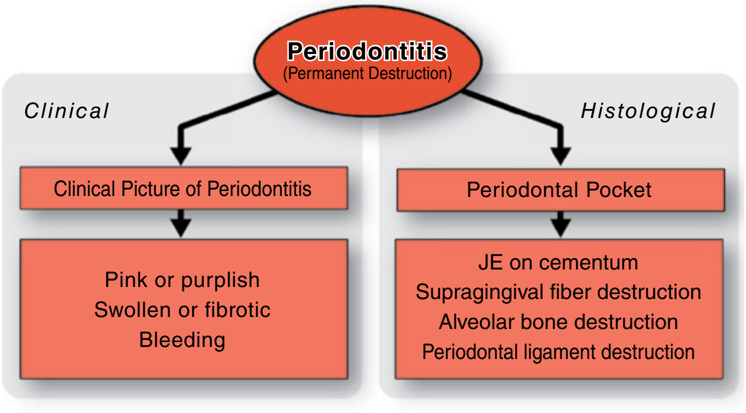
Periodontitis
(Permanent Destruction)
Clinical
Histological
Clinical Picture of Periodontitis
Pink or purplish
Swollen or fibrotic
Bleeding
Periodontal Pocket
JE on cementum
Supragingival fiber destruction
Alveolar bone destruction
Periodontal ligament destruction

Figure 3-4 **Characteristics of Periodontitis.** The clinical and histologic characteristics of periodontitis. Permanent tissue damage occurs in periodontitis.

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Periodontium in Health

1. **Clinical Picture of Healthy Gingiva**
   a. **Color.** Pink, may be pigmented, and is resilient in consistency.
   b. **Gingival Margin**
      1. Scalloped outline
      2. Located slightly coronal to (above) the cementoenamel junction (CEJ).
   c. **Interdental Papillae.** Firm and occupy the embrasure spaces apical to the contact areas.
   d. **Absence of Bleeding.** No bleeding upon probing.
   e. **Sulcus.** Probing depths range from 1 to 3 mm.
2. **The Microscopic Picture of Healthy Gingiva**
   a. **Junctional Epithelium.** The junctional epithelium is firmly attached by hemidesmosomes to the enamel slightly coronal to (above) the CEJ.
   b. **Epithelial–Connective Tissue Junction.** The junctional epithelium has no epithelial ridges.
   c. **Gingival Fibers.** Intact supragingival fiber bundles support the gingiva and the junctional epithelium.
   d. **Alveolar Bone.** The crest of the alveolar bone is intact and located 2 to 3 mm apical to (below) the base of the junctional epithelium.

   e. **Periodontal Ligament Fibers.** Intact periodontal ligament fiber bundles stretch between the bony walls of the tooth socket and the cementum of the root.
   f. **Cementum.** Cementum is normal. **Figure 3-5** illustrates the periodontium in health.

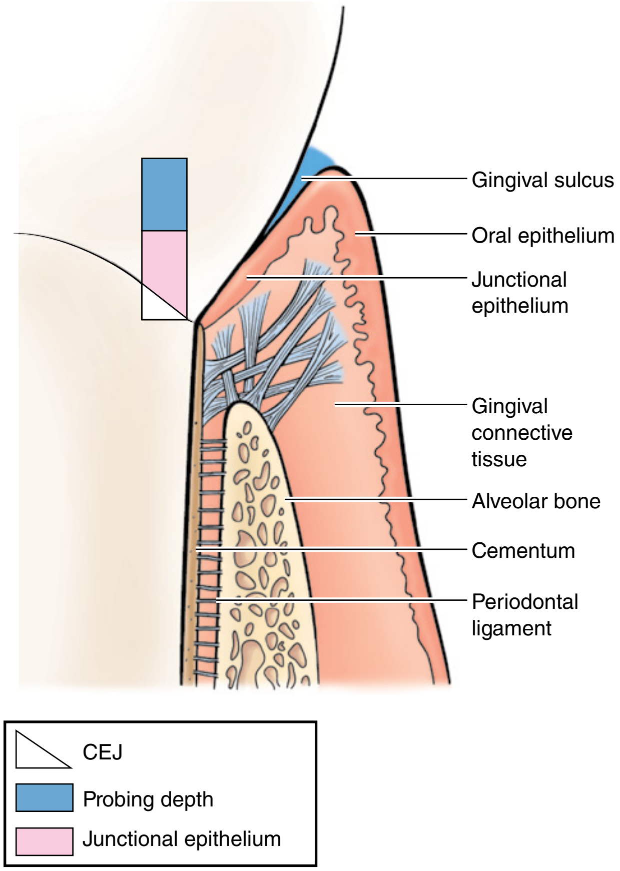
Gingival sulcus
Oral epithelium
Junctional epithelium
Gingival connective tissue
Alveolar bone
Cementum
Periodontal ligament
CEJ
Probing depth
Junctional epithelium

Figure 3-5 **The Healthy Periodontium.**

Plaque biofilm: light accumulation.
Junctional epithelium: slightly coronal to the CEJ, no epithelial ridge formation
Supragingival fibers: intact
Periodontal ligament fibers: intact
Alveolar bone: intact
Illustration Key: The white wedge indicates the location of the CEJ; the depth of the sulcus is shown by the blue vertical rectangle; the length of junctional epithelium is indicated by the pink vertical rectangle.

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Gingivitis—Reversible Tissue Damage

1. **Characteristics of Gingivitis.** **Gingivitis** is a type of periodontal disease characterized by changes in the color, contour, and consistency of the gingival tissues (**Figure 3-6**).
   a. **Onset of Gingivitis.** Gingivitis is observed clinically 4 to 14 days after plaque biofilm accumulates in the gingival sulcus.[5]
      1. Acute gingivitis is a gingivitis that lasts for a short period of time. It is characterized by fluid in the gingival connective tissues that results in swollen gingiva.

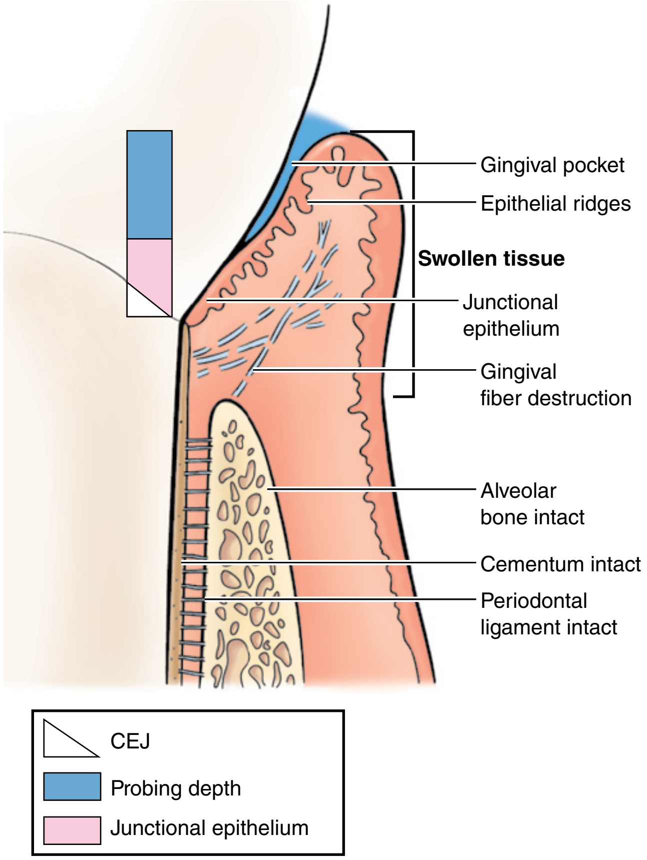
Gingival pocket
Epithelial ridges
Swollen tissue
Junctional epithelium
Gingival fiber destruction
Alveolar bone intact
Cementum intact
Periodontal ligament intact
CEJ
Probing depth
Junctional epithelium

Figure 3-6 Gingivitis.

Plaque biofilm: increased numbers of bacteria
Junctional epithelium: slightly coronal to the CEJ; the coronal portion of the JE detaches from the tooth; probing depth increases; epithelial ridges form and extend down into gingival connective tissue
Supragingival fibers: some fiber destruction
Periodontal ligament fibers: intact
Alveolar bone: intact
Illustration Key: Note in this illustration that the JE, as indicated by the pink rectangle, is still coronal to the CEJ (white wedge). The blue rectangle has increased in depth due to the swelling of the gingival margin, so that the gingival tissue covers more of the anatomic crown.

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2. Chronic gingivitis is a gingivitis that lasts for months or years.
    a. When gingivitis is chronic, the body may attempt to repair the tissue damage by forming new collagen fibers in the gingival connective tissue.
    b. Excess collagen fibers lead to gingival tissues that are enlarged and fibrotic (leathery) in consistency.
    c. The excess collagen fibers conceal the redness caused by the increased blood flow, making the tissue appear less red.

b. **Tissue Enlargement.** Gingival enlargement may be caused by swelling (acute gingivitis) or fibrosis (chronic gingivitis).
    1. Tissue enlargement causes the gingival margin to cover more of the anatomic crown which results in deeper probing depths.
    2. This enlargement of the gingival tissue is said to produce a false gingival pocket, known as a pseudopocket.

3. A gingival pocket has a sulcus depth greater than 3 mm. This increased probing depth is caused solely by enlarged gingival tissue. Microscopically, the junctional epithelium remains in its normal position coronal to CEJ on the tooth in a gingival pocket.

c. **Reversible Tissue Damage.** The tissue damage in gingivitis is **reversible tissue damage**; that is, with good patient self-care, the body can repair the damage.

d. **Duration of Gingivitis.** In many cases, gingivitis may persist for years without ever progressing to the next stage, periodontitis. In some cases, however, a combination of risk factors may result in gingivitis progressing to periodontitis.

2. **Clinical Picture of Gingivitis**

a. **Color.** In gingivitis, the gingival tissue usually is red or reddish-blue in color.

1. Blood flow increases in the gingival connective tissue, and the gingival blood vessels become engorged with blood, causing the gingiva to appear red.
2. Over time, as the gingivitis persists, the gingival blood vessels may become congested, which slows the flow of oxygenated blood to the gingival tissues and the flow of unoxygenated (deoxygenated) blood away from the gingival tissues. Consequently, unoxygenated blood pools in the tissue, thereby giving the gingiva a dark-bluish discoloration (**Figure 3-7**). When inflamed tissue has this distinct dark bluish discoloration, the tissue is said to be cyanotic.

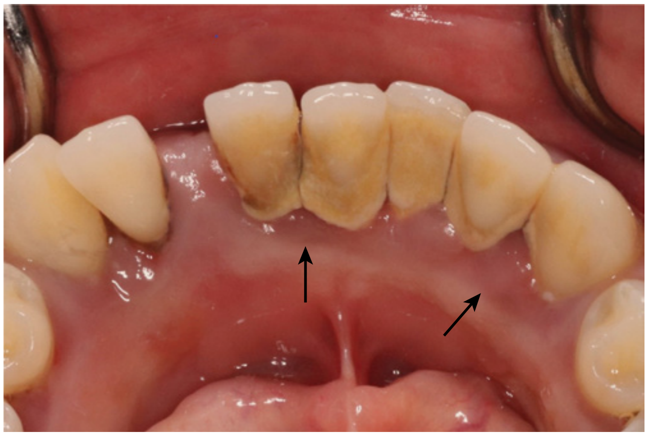

Figure 3-7 **Cyanotic Gingiva.** Note the dark-bluish color of the gingiva (arrows). Additionally, observe the thick band of supragingival calculus deposits residing along the gingival margin. These calculus deposits are areas of plaque biofilm retention, which further triggers the host inflammatory response and contributes to the cyanotic appearance of the gingiva.

b. **Gingival Margin**

1. The gingival margin appears swollen and loses its knife-edge adaptation to the tooth due to excessive fluid accumulation in the tissues, known as edema. Thus, the term *edematous* may be given to inflamed tissues that appear swollen. (**Figure 3-8**).
2. Gingival tissue covers more of the anatomic crown due to tissue swelling or fibrosis.

c. **Interdental Papillae.** The interdental papillae often are bulbous and swollen.

d. **Bleeding.** There is bleeding upon gentle probing.

e. **Sulcus.** Probing depths may be greater than 3 mm due to swelling of the tissues. Note that *there is no apical migration of the junctional epithelium in gingivitis.* Instead, the deeper probing measurements in gingivitis sites is due to the inflammatory-induced tissue overgrowth (pseudopockets), rather than true periodontal pockets.

3. **The Microscopic Picture of Gingivitis**

a. **Junctional Epithelium.** The hemidesmosomes still attach to the enamel coronal to the CEJ.

b. **Epithelial–Connective Tissue Junction**

1. Unlike periodontal health, in gingivitis the epithelial ridges form and extend from the junctional epithelium into the adjacent connective tissue zone.

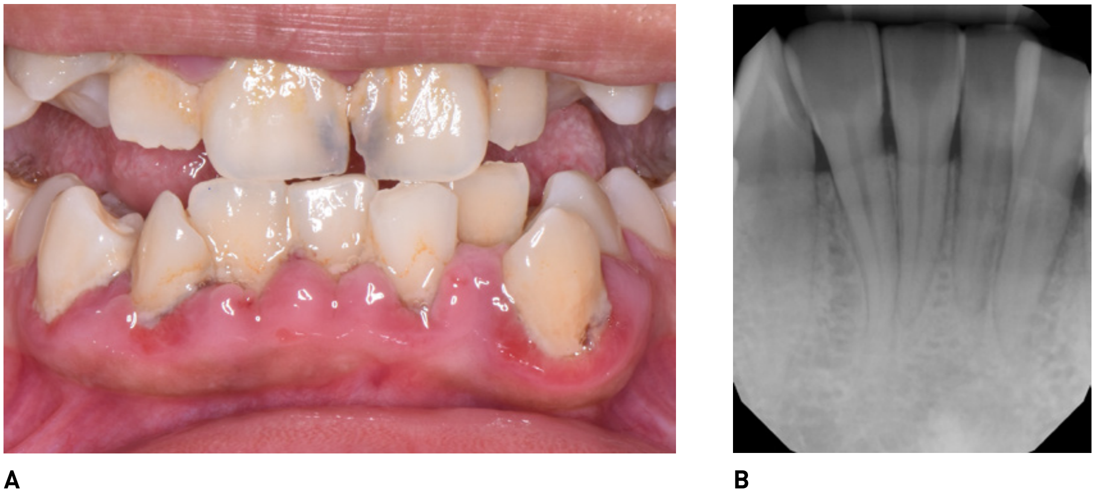

Figure 3-8 Rolled, Swollen (Edematous) Gingival Margins of a Gingivitis Patient. A. Note the edematous gingival tissues of all six mandibular anterior teeth along with the erythematous gingiva of the facial aspects of the mandibular canines. **B.** Radiographic view of the same case. Note the absence of bone loss around the mandibular anterior teeth.

        2. Such extension of the epithelial ridges only can occur because destruction of the gingival fibers creates space for the growing epithelium.
    c. **Gingival Fibers.** Damage has occurred to the supragingival fiber bundles. This damage is reversible if the bacterial infection is brought under control.
    d. **Alveolar Bone.** The bacterial infection has not progressed into the alveolar bone. There is no destruction of alveolar bone.
    e. **Periodontal Ligament Fibers.** The bacterial infection has not progressed into the periodontal ligament fibers.
    f. **Cementum.** The cementum covering the root of the tooth is normal.

Periodontitis—Permanent Tissue Destruction

1. **Characteristics of Periodontitis**
    a. **Extent of Tissue Destruction**
        1. **Periodontitis** is a type of periodontal disease that is characterized by (1) the apical migration of the junctional epithelium, (2) the loss of connective tissue attachment, and (3) the loss of alveolar bone.[6]
        2. The tissue damage of periodontitis is permanent (**irreversible tissue damage**).
    b. **Process of Tissue Destruction**
        1. The tissue destruction of periodontitis is not a continuous process. Rather, the disease process occurs in an intermittent manner with extended periods of disease inactivity followed by short bursts of destruction.[3]
        2. Tissue destruction progresses at different rates throughout the mouth. Destruction does not occur in all parts of the mouth at the same time, but instead, destruction usually occurs in only a few specific sites (tooth surfaces) at a time.
2. **Clinical Picture of Periodontitis**
    a. **Color.** The gingival tissue shows visible alternations color, contour, and consistency.
        1. Edematous tissue (spongy tissue)—bluish- or purplish-red with a smooth, shiny appearance.
        2. Fibrotic tissue (firm, nodular tissue)—light pink with a leathery consistency. Beginning clinicians often mistakenly interpret this light pink color as a sign of tissue health.
    b. **Gingival Margin**
        1. The gingival margin may be swollen or fibrotic and does not have a close knife-edged adaptation to the neck of the tooth (**Figure 3-9**).

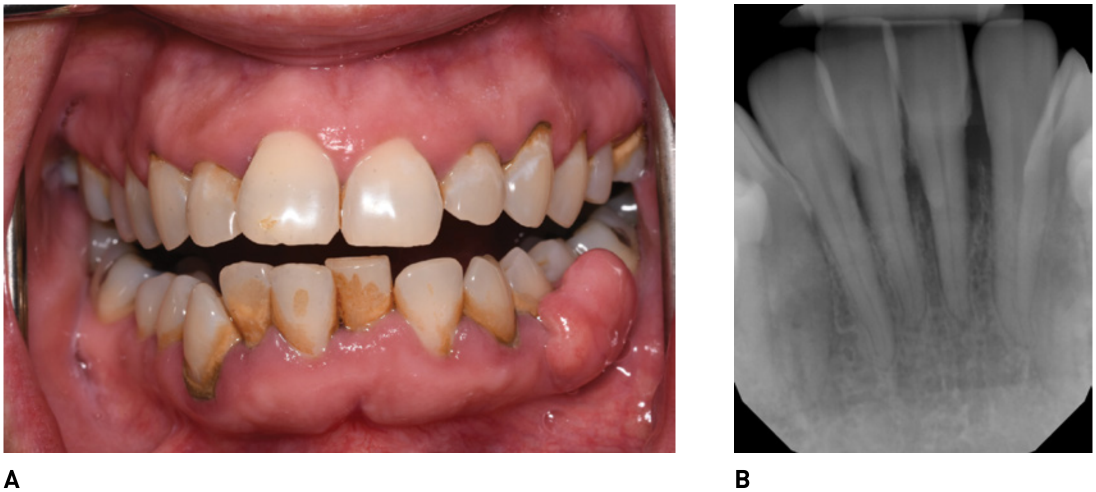

Figure 3-9 Periodontitis Patient. A. Note the irregular gingival contours, enlarged interdental papillae, and heavy supragingival plaque, calculus, and staining. **B.** Radiograph of the same case. Note the loss of alveolar bone and the heavy bands of calculus deposits on the radicular surfaces.

    2. The position of the gingival margin varies greatly in periodontitis. The margin may be apical to the CEJ (known as positive recession), resulting in a portion of the root being visible in the mouth. Or, in some instances, the margin may be notably coronal to the CEJ (known as negative recession), leading to the appearance of tissue overgrowth.
  c. **Interdental Papillae**
    1. The interdental papillae may appear as if it is ballooning out of the gingival embrasure space.
    2. However, in some cases of periodontitis, partial or complete loss of the interdental papilla (blunted papilla) may occur.
  d. **Bleeding.** There often is bleeding upon probing. In some cases, suppuration (a discharge of pus) may be visible.
  e. **Pocket.** Probing depths are 4 mm or greater in depth because the junctional epithelium is no longer firmly attached to the tooth. Consequently, the tissue offers less resistance as the periodontal probe is gently inserted into the sulcus.
    1. Pus may or may not be evident upon probing.
    2. Pain is usually absent; however, probing may cause some pain due to ulceration of the pocket epithelium.

3. **The Microscopic Picture of Periodontitis**
  a. **Junctional Epithelium**
    1. The junctional epithelium is located on the cementum, apical to (below) its normal location. Movement of the junctional epithelium apical to its normal location is termed the **apical migration of the junctional epithelium** (Figure 3-10).
    2. The coronal-most portion of the junctional epithelium detaches from the tooth surface. As the bacterial infection progresses, the apical portion of the junctional epithelium moves further in an apical direction along the root surface creating a periodontal pocket.
    3. The extracellular matrix of the gingiva and the attached collagen fibers at the apical edge of the junctional epithelium are destroyed.
  b. **Epithelial–Connective Tissue Junction**
    1. The *junctional* epithelium proliferates and extends epithelial ridges into the connective tissue.
    2. The *sulcular* epithelium of the pocket wall thickens and extends epithelial ridges deep into the connective tissue. Small ulcerations of the pocket epithelium expose the underlying inflamed connective tissue.

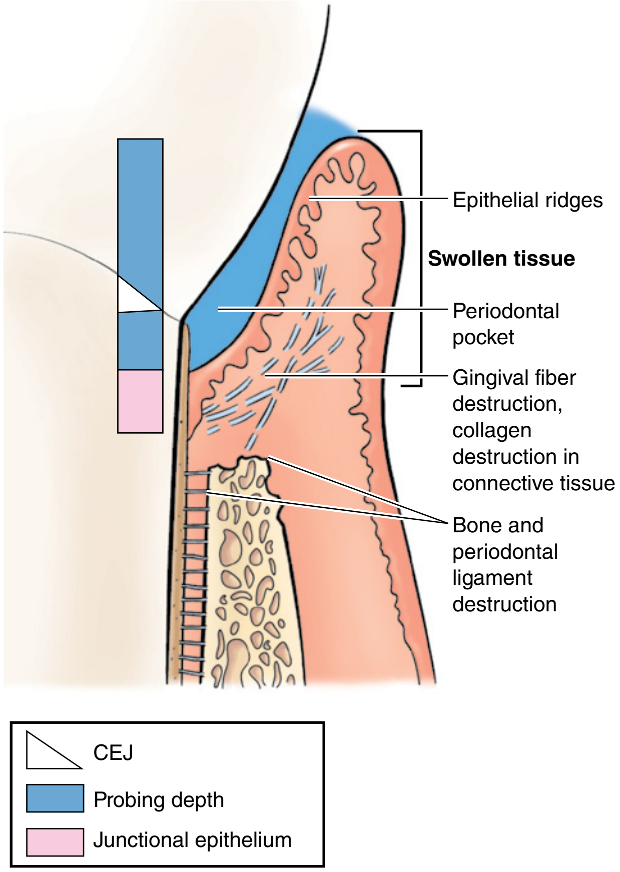
Epithelial ridges
Swollen tissue
Periodontal pocket
Gingival fiber destruction, collagen destruction in connective tissue
Bone and periodontal ligament destruction
CEJ
Probing depth
Junctional epithelium

Figure 3-10 **Periodontitis.**

Plaque biofilm: vast numbers of bacteria
Junctional epithelium: apical to the CEJ with attachment on cementum; a remnant of the JE persists at the base of the periodontal pocket; epithelial ridges extend down into gingival connective tissue
Supragingival fibers: fiber destruction
Periodontal ligament fibers: fiber destruction
Alveolar bone: portions of alveolar bone destroyed
Illustration Key: Note the blue rectangle shows that the pocket in this illustration is increased by (1) the swollen gingival margin and (2) the location of the JE apical to (below) the CEJ. The level of the CEJ is indicated by the white wedge.

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c. **Gingival Connective Tissue**
   1. Changes to the gingival connective tissue are severe. Collagen destruction in the area of inflammation is almost complete.
   2. There is widespread destruction of the supragingival fiber bundles, reducing them to fiber fragments. The destruction of the periodontal ligament fiber bundles makes it easier for the junctional epithelium to migrate apically along the root surface.
   3. The transseptal fiber bundles are tough and resilient bands of gingival ligament fibers that run from one tooth to another across the crest of the bone.
      a. In the presence of mild to moderate periodontitis, transseptal fibers are strong enough to withstand the inflammatory consequences of periodontal disease (**Figure 3-11**).

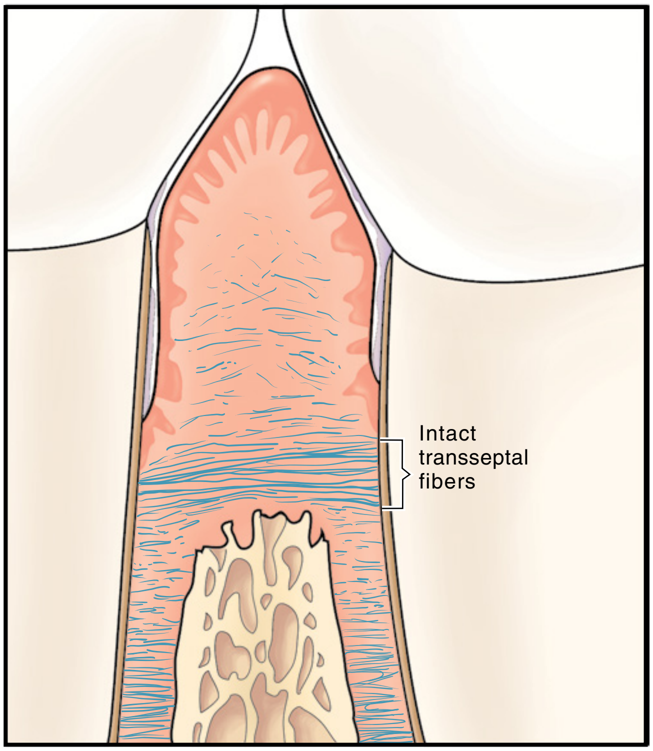

Figure 3-11 Band of Intact Transseptal Fibers. Even in the presence of mild to moderate inflammation, an intact band of transseptal fibers above the remaining alveolar bone is able to withstand disruption and preserve arch integrity. However, if the inflammation is severe enough to overcome the capabilities of the transeptal fibers to hold the arch in place, pathologic migration of the teeth may result.

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b. However, if the inflammation is severe it may overcome the continuity of the transseptal fibers, thereby leading to tooth displacement. This is known as **pathologic tooth migration**. Pathologic tooth migration may be a foreboding sign of severe periodontitis and may be a motivating factor for patients to seek treatment (**Figure 3-12**).

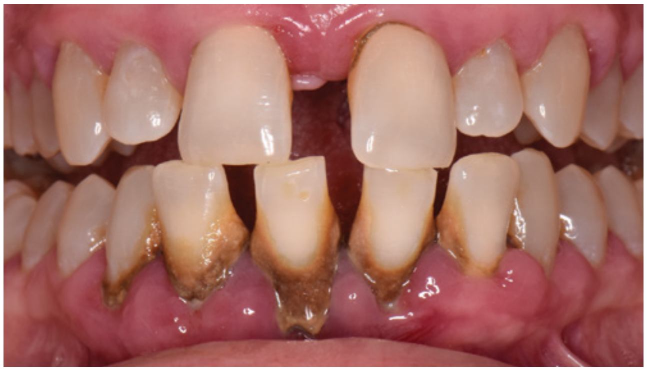

Figure 3-12 Pathologic Tooth Migration. This photo shows a 41-year-old periodontitis patient with a chief complaint of pathologic tooth migration. The patient first noticed that her "teeth were moving apart" approximately 8 months ago. Note the facial flaring and the spacing between the teeth. The patient's periodontal inflammation led to a disruption of the transseptal fibers, ultimately leading to a loss in arch integrity with subsequent migration of teeth.

4. Epithelium grows over the root surface in areas where the fiber bundles have been destroyed. The loss of fiber attachment is permanent because the epithelium growing over the root surface prevents the reinsertion of the periodontal ligament fibers in the cementum.

d. **Alveolar Bone.** The alveolar bone that supports the teeth is permanently destroyed. Tooth mobility may be present.
e. **Periodontal Ligament Fibers**. Some or all of the periodontal ligament fiber bundles are permanently destroyed.
f. **Cementum.** Cementum within the periodontal pocket is exposed to dental plaque biofilm.
g. **Pulp.** Histologic studies of the dental pulp of patients with severe periodontitis show inflamed, edematous pulps, pulpal necrosis, vascular congestion, and dentin demineralization.[7,8]

Table 3-2 summarizes and contrasts the histologic changes seen in gingivitis and periodontitis.

Table 3-2 Histologic Changes in Disease

Disease State	Histology
Gingivitis	Epithelial ridges extend down into connective tissue. Destruction of supragingival fiber bundles.
Periodontitis	Changes in epithelial tissues: ■ Junctional epithelium located apical to the cementoenamel junction. ■ Junctional epithelium grows along the root surface. ■ Sulcular epithelium thickens and extends epithelial ridges down into the connective tissue. Changes in connective tissues and alveolar bone: ■ Collagen destruction. ■ Destruction of supragingival fiber bundles. ■ Destruction of periodontal ligament fibers; transseptal fibers regenerate and remain intact. ■ Junctional epithelium grows over the root surface in areas where the periodontal ligament fibers are destroyed. ■ Root cementum is exposed to the plaque biofilm. ■ Destruction of alveolar bone.

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Section 2

Pathogenesis of Bone Destruction

Inflammation is the body's response to injury or invasion by disease-producing organisms. The inflammatory process that occurs in periodontitis results in permanent destruction to the tissues of the periodontium, including the destruction of gingival connective tissue, periodontal ligament, and alveolar bone. **Alveolar bone loss** is the resorption of alveolar bone due to periodontitis. This section discusses the patterns of bone destruction that occur in periodontitis. The pattern of bone destruction that occurs depends on the pathway of inflammation as it spreads from the gingiva into the alveolar bone. It is important to understand the changes that occur in the alveolar bone because it is the reduction in bone height that eventually results in tooth loss (**Figures 3-13** to **3-15**).

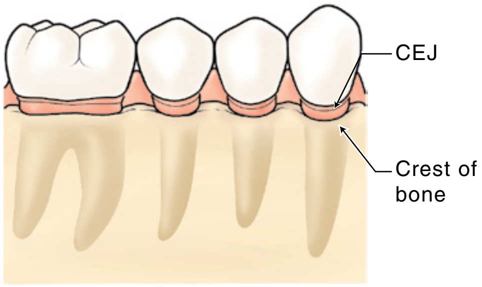

Figure 3-13 Level of Alveolar Crest in Health and Gingivitis. In health and gingivitis, the crest of the alveolar bone is located approximately 2 to 3 mm apical to the cementoenamel junction.

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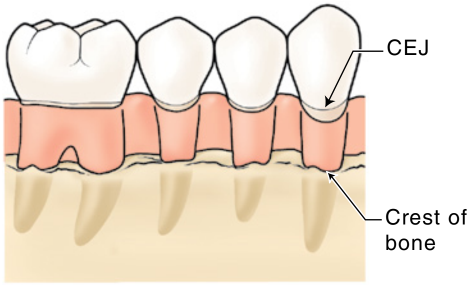

Figure 3-14 Level of Alveolar Crest in Periodontitis. In periodontitis, the crest of the alveolar bone is located more than 2 to 3 mm apical to the cementoenamel junction.

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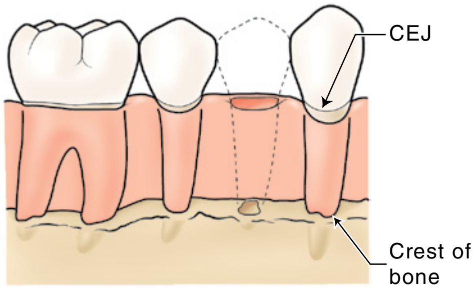

Figure 3-15 Level of Alveolar Crest as Periodontitis Progresses. Progressive alveolar bone loss occurs in periodontitis. Bone destruction may eventually lead to tooth mobility or tooth loss due to insufficient bone support.

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Changes in Alveolar Bone Height in Disease

1. **Reduction in Bone Height**
    a. **Bone Height in Health and Gingivitis.** In health and gingivitis, the crest of the alveolar bone is located approximately 2 to 3 mm apical to (below) the CEJs of the teeth.
    b. **Bone Height in Periodontitis.** In periodontitis, alveolar bone height is reduced as the result of inflammation. As periodontal disease progresses (worsens), tooth loss may occur from lack of alveolar bone support.

Patterns of Bone Loss in Periodontitis

1. **Patterns of Bone Loss.** The two types of bone loss are (1) horizontal and (2) vertical bone loss (**Figures 3-16** and **3-17**).

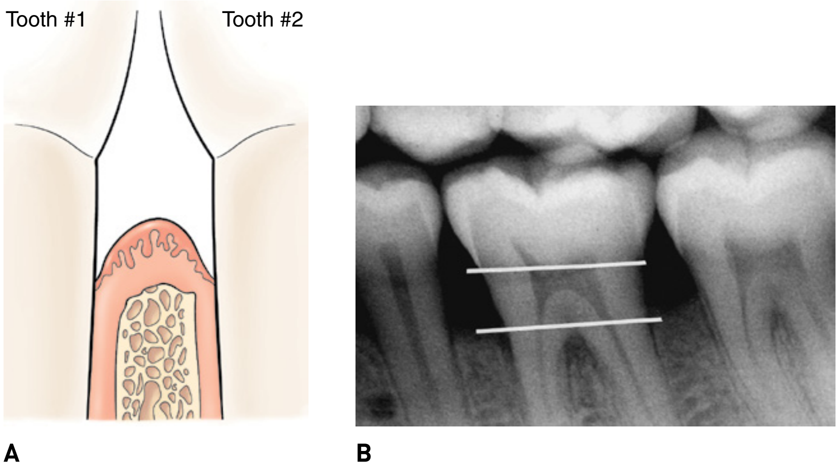

Figure 3-16 **Horizontal Pattern of Bone Loss. A.** Horizontal bone loss results in bone levels that are approximately at the same height on adjacent tooth roots. **B.** As depicted on the radiograph, alveolar bone resorption is uniform on both mesial and distal surfaces. On a radiograph, if an imaginary line drawn between the CEJs of adjacent teeth is approximately parallel, then the bone loss is described as horizontal bone loss.

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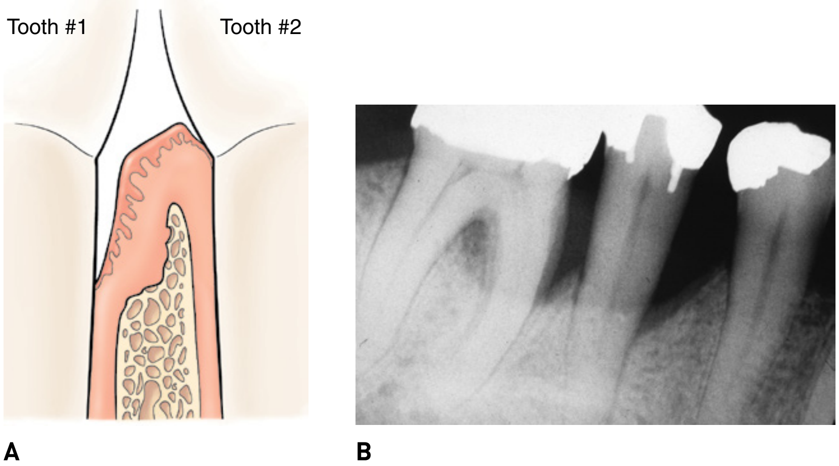

Figure 3-17 **Vertical Pattern of Bone Loss. A.** Vertical bone loss results in an uneven reduction in bone height on adjacent tooth roots, resulting in a trench-like area of missing bone alongside the root of one tooth. **B.** On a radiograph, if an imaginary line drawn between the CEJs of adjacent teeth is not parallel, then the bone loss is described as vertical bone loss.

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a. **Horizontal Bone Loss**
   1. **Horizontal bone loss** is the most common pattern of bone loss.
   2. This type of bone loss results in a fairly even, overall reduction in the height of the alveolar bone.
   3. The alveolar bone is reduced in height, but the margin of the alveolar crest remains more or less perpendicular to the long axis of the tooth.

b. **Vertical Bone Loss**
   1. **Vertical bone loss** is a less common pattern of bone loss. Vertical bone loss is also known as angular bone loss.
   2. This type of bone loss results in an uneven reduction in the height of the alveolar bone.
   3. In vertical bone loss, the resorption progresses more rapidly in the bone next to the root surface. This uneven pattern of bone loss leaves a trench-like area of missing bone alongside the root.

2. **Pathways of Inflammation into the Alveolar Bone.** The pathway of inflammation into the alveolar bone varies according to whether the bone loss is horizontal or vertical (**Figure 3-18**).

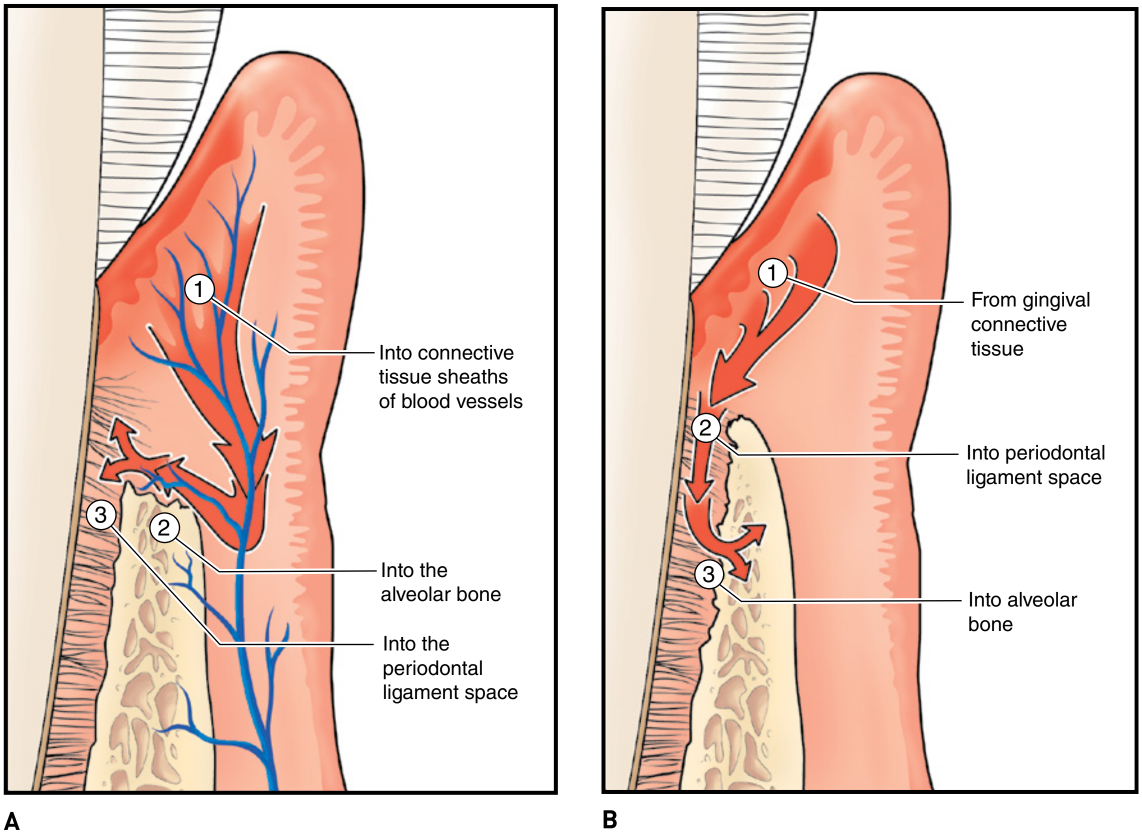
1
Into connective tissue sheaths of blood vessels
2
Into the alveolar bone
3
Into the periodontal ligament space
A
1
From gingival connective tissue
2
Into periodontal ligament space
3
Into alveolar bone
B

Figure 3-18 Pathway of Inflammation into Alveolar Bone. A. In horizontal bone loss, inflammation spreads through the tissues in this order: into the gingival connective tissue, into the alveolar bone, and then into the periodontal ligament. **B.** In vertical bone loss, inflammation spreads through the tissues in this order: into the gingival connective tissue, into the periodontal ligament, and then into the alveolar bone.

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a. **Pathway of Inflammation in Horizontal Bone Loss**
   1. In horizontal bone loss, inflammation spreads into the tissues in this order: (1) within the gingival connective tissue along the connective tissue sheaths surrounding the blood vessels, (2) into the alveolar bone, and (3) finally, into the periodontal ligament space.
   2. Inflammation usually spreads in this manner because it is the path of least resistance. The periodontal ligament fiber bundles act as an effective barrier to the spread of inflammation. Thus, the inflammation spreads into the alveolar bone and then into the periodontal ligament space.

b. **Pathway of Inflammation in Vertical Bone Loss**
   1. In vertical bone loss, inflammation spreads into the tissues in this order: (1) within the gingival connective tissue, (2) directly into the periodontal ligament space, and (3) finally, into the alveolar bone.

2. Inflammation spreads in this manner whenever the crestal periodontal ligament fiber bundles are weakened and no longer present an effective barrier. Prior events such as occlusal trauma can be responsible for the weakened condition of the fiber bundles.

3. **Bone Defects in Periodontitis.** In contrast to a **suprabony defect**, (in which the base of the pocket is located *coronal* to the alveolar crest), an **infrabony defect** is characterized by a pocket in which the base of the defect extends *apical* to the residual alveolar crest. As such, an infrabony defect is an **osseous defect** because it involves the loss of bone. In turn, infrabony defects can be subdivided into two types: intrabony defects and osseous craters.
   a. **Intrabony Defects**
      1. Intrabony defects result when bone resorption occurs in an uneven, oblique direction. In intrabony defects, the bone resorption primarily affects one tooth.
      2. **Classification of Intrabony Defects.** Intrabony defects are box-shaped defects that are classified according to the number of residual (remaining) alveolar bony walls surrounding the defect.
         a. A one-wall defect, also known as a hemiseptal defect, is a defect where half of the interdental septum is lost while the other half remains attached to a tooth (**Figure 3-19**).
         b. A two-wall defect is shown in **Figure 3-20**.
         c. A three-wall defect is shown in **Figure 3-21**.

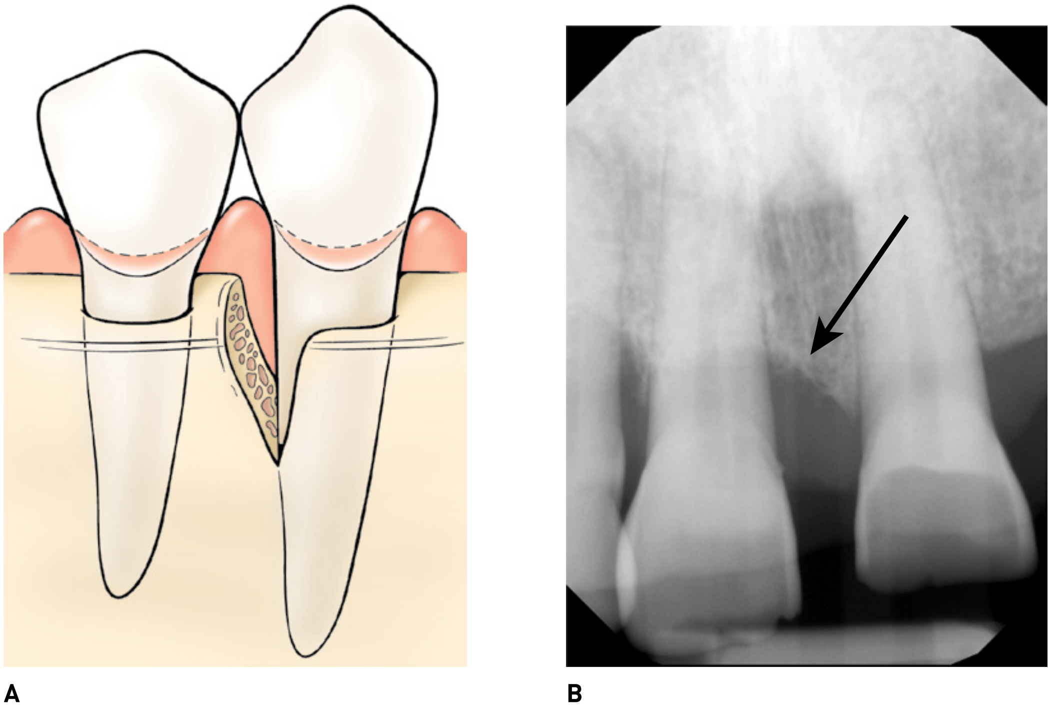

Figure 3-19 One-Wall Intrabony Defect. A. Looking from the canine tooth root distally toward the premolar, only one wall of bone remains, and it is on the mesial surface of the premolar. The facial plate and lingual plate of bone are missing. **B.** Radiograph of a one-wall intrabony defect. Note the "slope-like" pattern of a one-wall intrabony defect, with the peak of the slope (on the mesial side of the maxillary left central incisor) at a different level compared to the base of the slope (on the mesial side of the maxillary right central incisor). Also note that half of the interdental septum is missing (from the mesial of the maxillary right central incisor), while the other half is still preserved and remains attached to the maxillary left incisor (arrow).

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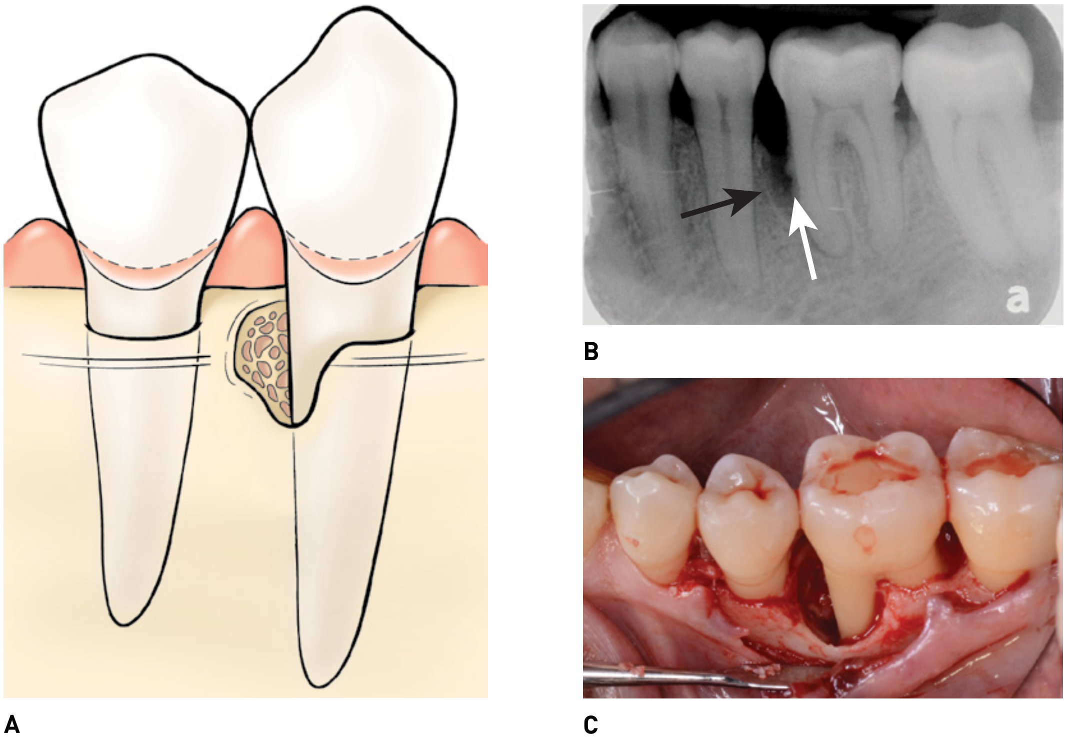

Figure 3-20 Two-Wall Intrabony Defect. A. The "two walls" surrounding this defect are the remaining lingual plate and the bony wall abutting the mesial surface of the premolar tooth root. **B.** Radiograph of a two-wall intrabony defect. Similar to a one-wall defect, the two-wall defect appears slope-like. But unlike a one-wall defect, the two-wall defect is bounded by two remaining osseous walls: a mesial bony wall (black arrow) and a lingual bony wall (white arrow). Note the faint cloudy appearance of the trabecular bone depicting the presence of an intact lingual bony wall bounding the defect. **C.** Clinical photograph of the same case from panel B. Following surgical flap reflection, note the presence of a two-wall intrabony osseous defect that is bounded by a mesial bony wall and a lingual bony wall.

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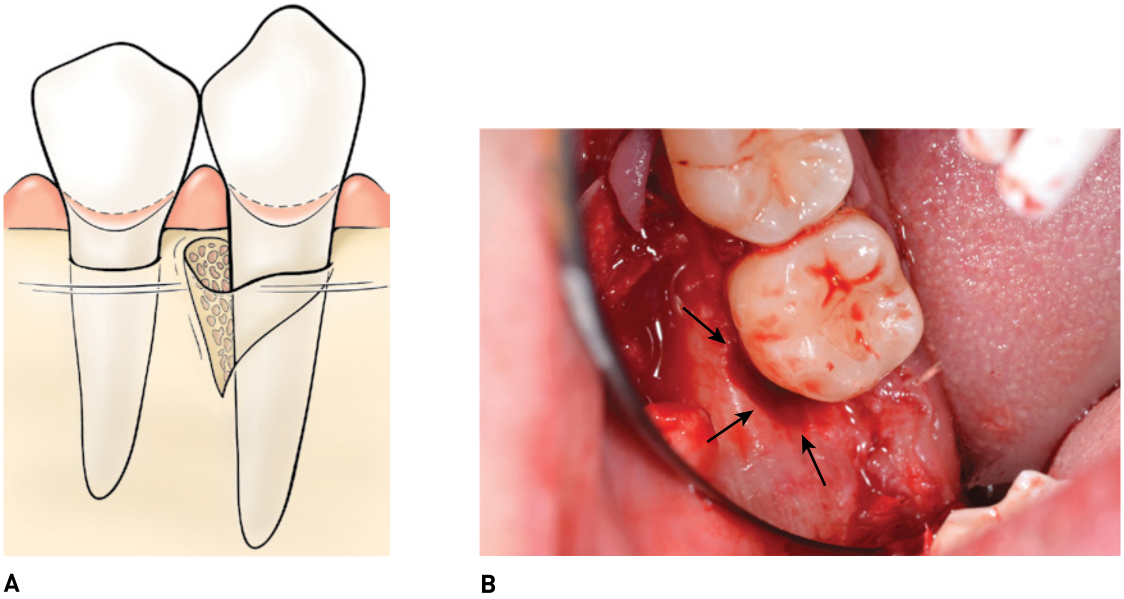

Figure 3-21 Three-Wall Intrabony Defect. A. The "three walls" of remaining bone that surround this defect are the lingual plate, facial plate, and bone on the mesial surface of the adjacent premolar root. **B.** A surgical photograph of a three-wall defect associated with a mandibular molar. Note that three bony walls surround this defect: a buccal bony wall, a mesial bony wall, and a distal bony wall (black arrows).

A: © Wolters Kluwer; **B:** Photo courtesy of Dr. Celine Cornelius, Augusta, GA.

b. **Proximal Bone Contour.** In health, the normal contour of the alveolar bone on the proximal surface of a posterior tooth from the facial to lingual is a relatively flat interproximal contour. When bone loss occurs in the interdental alveolar bone, the contour of the bone from facial to lingual dips apically to form what is described as an **osseous crater**.

1. Whereas intrabony defects primarily affect *one* tooth, osseous craters affect *two* adjacent root surfaces to a similar extent (**Figures 3-22** and **3-23**).
2. The presence of an osseous crater causes dental plaque biofilm to collect and makes it difficult to clean the interdental area.

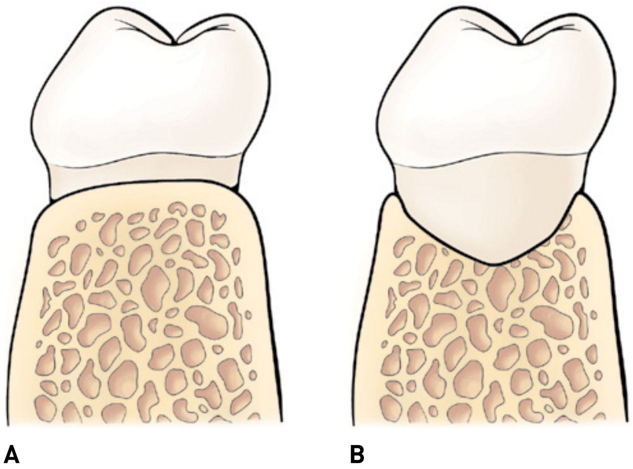

Figure 3-22 Interdental Bone: Normal Contour vs. Osseous Crater. A. Normal contour of the alveolar bone on the proximal (mesial or distal) surface of a posterior tooth in health. Note that the bone contour from the facial to lingual is a relatively flat interproximal contour. **B.** Osseous crater on the proximal surface of a posterior tooth. Note that the contour of the bone from the facial to the lingual dips apically and forms what is described as a "crater" between the facial and lingual bone margins with the bone loss nearly equal on the roots of the two adjacent teeth.

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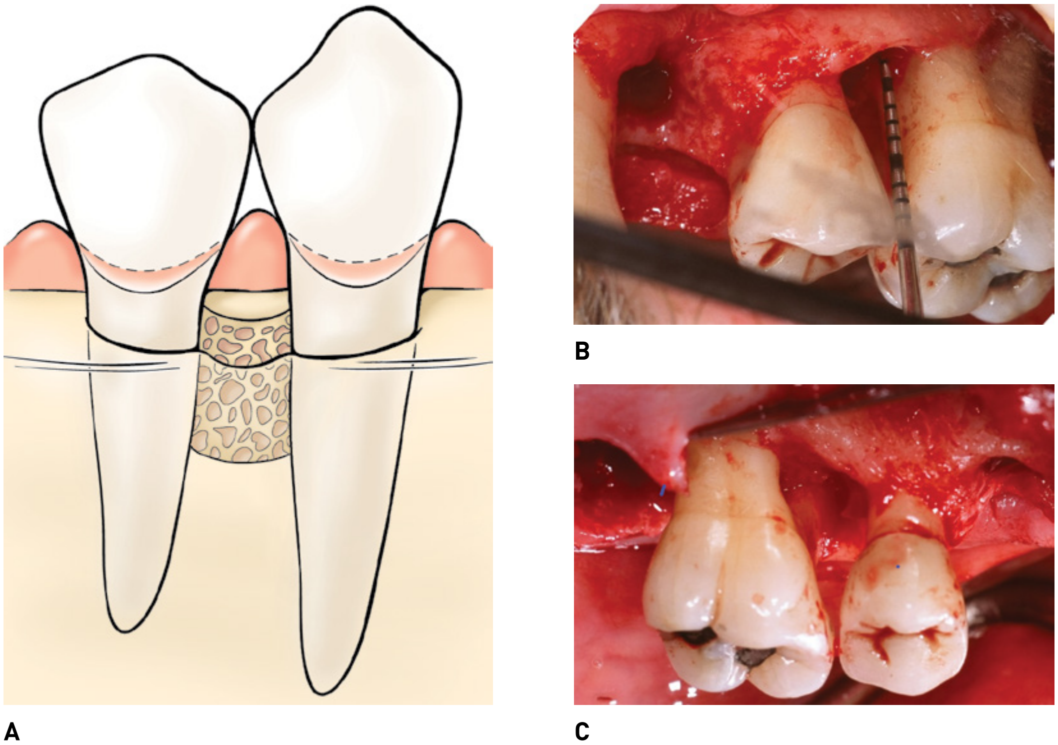

Figure 3-23 **Interproximal Osseous Crater. A.** Interproximal osseous crater with the lingual plate and facial plate of bone remaining. The bone between these plates is missing resulting in bone being lost on the mesial surface of the premolar and the distal surface of the adjacent canine. The term *crater* refers to the dip in the contour of the interproximal bone between the facial and lingual plates. **B.** Buccal view of an osseous crater between the maxillary premolar and maxillary molar. Note the presence of an intact buccal bony wall with an infrabony defect depth of 3 mm. **C. Palatal View of Osseous Crater.** Palatal view of the same defect as B. Note the intact palatal bony wall which is bounded by two avascular root surfaces (the mesial surface of the molar on one side and the distal surface of the premolar on the other).

A: © Wolters Kluwer; **B, C:** Case Courtesy of Dr. Brian Goldstein, Providence, RI.

c. Bone Loss in Furcation Areas

1. **Furcation involvement** occurs on a multirooted tooth when periodontal infection invades the area between and around the roots, resulting in a loss of alveolar bone between the roots of a multirooted tooth.
2. Bone loss in the furcation area may be hidden by the gingival tissue or may be clinically visible in the mouth (**Figure 3-24**).

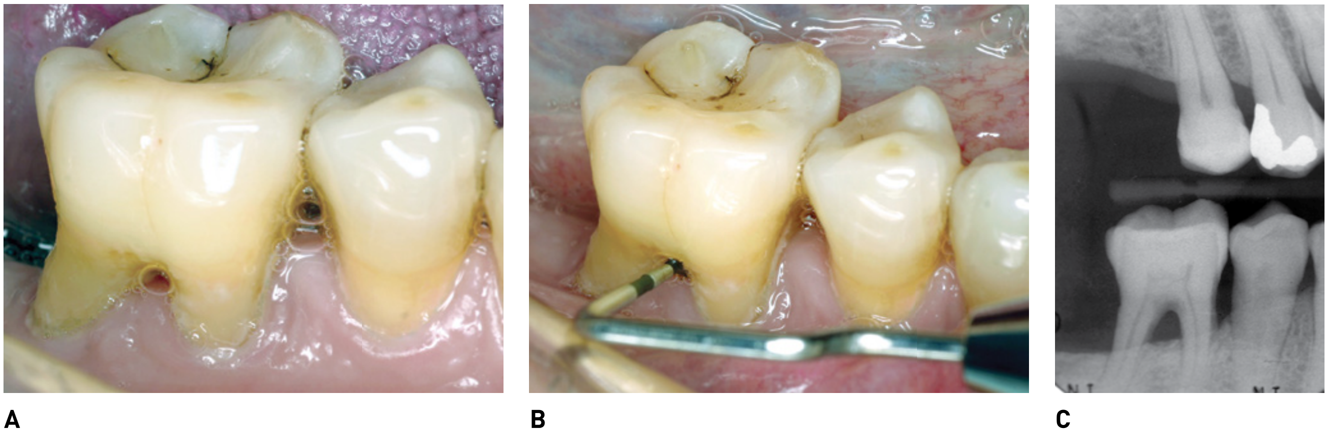

Figure 3-24 **Furcation Involvement. A.** Due to recession, the furcation involvement on this molar is clinically evident. **B.** A periodontal probe can easily be inserted between the two roots of this mandibular molar. **C.** A radiograph shows the extensive bone loss around this molar.

Courtesy of Dr. Richard J. Foster, Educational Solutions and Design, Greensboro, NC.

Section 3

Periodontal Pockets

Characteristics of Periodontal Pockets

1. **Attachment Loss in Periodontal Pockets**
   a. **Attachment loss** is the destruction of the fibers and bone that support the teeth.
   b. Tissue destruction does not spread only in an apical (vertical) direction but also in a lateral (side-to-side) direction.
   c. *A pocket on different root surfaces of the same tooth can have different depths.* The loss of attachment may vary from surface to surface of the tooth, with the base of the pocket exhibiting very irregular patterns of tissue destruction (**Figure 3-25**).

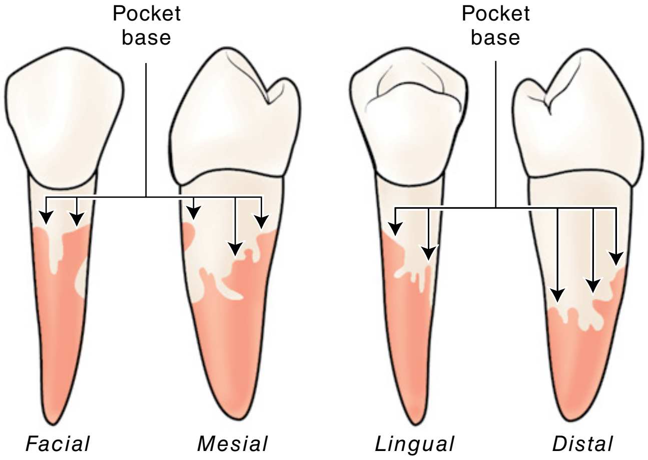

Figure 3-25 **Irregular Pattern of Attachment Loss.** The amount of attachment loss can vary greatly on different surfaces of the same tooth. The base of a pocket may exhibit very irregular patterns of destruction.

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2. **Disease Sites.** A **disease site** is an area of tissue destruction. A disease site may involve only a single surface of a tooth, for example, the distal surface of a tooth. In other cases, the disease site may involve several surfaces of the tooth or all four surfaces (mesial, distal, facial, and lingual).
   a. **Inactive disease site**—a disease site that is stable, with the attachment level of the junctional epithelium remaining the same over time.
   b. **Active disease site**—a disease site that shows continued apical migration of the junctional epithelium over time.
   c. **Assessment of Disease Activity.** The disease activity of each site in the mouth should be assessed using a periodontal probe and recorded in the patient chart at regular intervals (scheduled check-up appointments).
   d. **Periodontal Pockets.** *A periodontal pocket is an area of tissue destruction left by the disease process.* The pocket is much like a demolished home that is left after a hurricane.
      1. The presence of a periodontal pocket does not indicate necessarily that there is active disease at that site. Likewise, a demolished house does not necessarily indicate that a hurricane still is pounding the shoreline. A demolished house may indicate that the hurricane is still active or that a hurricane passed through a day, a week, or a year ago.
      2. Most pockets in adult patients with periodontitis are inactive disease sites.

Pocket Formation

1. **Gingival Sulcus.** In health, the average histologic depth of the sulcus is 1 to 3 mm in depth.[9] *The junctional epithelium is coronal to the CEJ and attaches along its entire length to the tooth* (**Figure 3-26**).

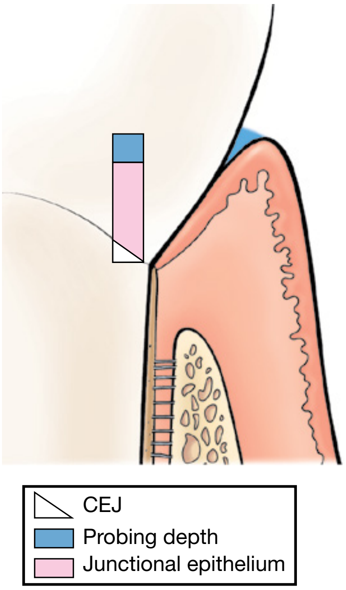

Figure 3-26 Gingival Sulcus. In health, the average clinical probing depth of the gingival sulcus ranges from 1 to 3 mm (indicated by the blue rectangle). The junctional epithelium (JE) attaches along its entire length to the enamel of the tooth (as represented by the pink rectangle).

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2. **Gingival Pockets.** A **gingival pocket** is a deepening of the gingival sulcus as a result of swelling or enlargement of the gingival tissue.
   a. **Why are gingival pockets often called "false" pockets?**
      1. Gingival pockets sometimes are referred to *pseudopockets*, meaning "false pockets," because there is *no apical migration of the junctional epithelium*.
      2. *In gingivitis, however, the coronal portion of the junctional epithelium detaches from the tooth resulting in a slight increase in probing depth*.
   b. **What causes the increased probing depth of a gingival pocket?** The increased probing depth seen in a gingival pocket is due to (1) detachment of the coronal portion of the JE from the tooth and/or (2) increased tissue size due to swelling of the tissue (**Figure 3-27**).

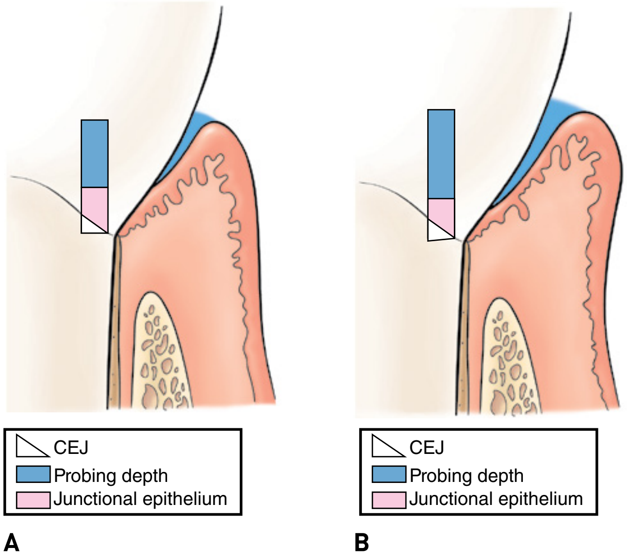

Figure 3-27 **Gingival Pockets. A.** The JE does not experience apical migration; however, the coronal portion of the JE detaches from the tooth, resulting in increased probing depth. **B.** In some cases the gingival tissue swells, resulting in a pseudopocket.

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3. **Periodontal Pockets**
   a. A **periodontal pocket** is a pathologic deepening of the gingival sulcus.
      1. Pocket formation occurs as the result of the (1) apical migration of the junctional epithelium, (2) destruction of the periodontal ligament fibers, and (3) destruction of alveolar bone.
      2. Apical migration is the movement of the cells of the junctional epithelium from their normal position—coronal to the CEJ—to a position apical to the CEJ. In health, the junctional epithelial cells attach to the enamel of the tooth crown. In periodontitis, the junctional epithelial cells attach to the cementum of the tooth root.
   b. **Two Types of Periodontal Pockets.** The type of periodontal pocket is determined based on the relationship of the junctional epithelium to the crest of the alveolar bone.
      1. **Suprabony Pocket**
         a. **Suprabony pockets** occur when there is horizontal bone loss, but the base of the periodontal pocket is coronal to the alveolar crest.
         b. The junctional epithelium, forming the base of the pocket, is located coronal to the crest of the alveolar bone (**Figure 3-28**).
      2. **Infrabony Pocket**
         a. **Infrabony pockets** occur when there is vertical bone loss. As previously described, infrabony pockets are classified as either intrabony defects (one-, two-, three-wall defects) or osseous defects.
         b. The junctional epithelium, forming the base of the pocket, is located apical to (below) the crest of the alveolar bone. The base of the pocket is located within the cratered-out area of the bone alongside of the root surface (**Figure 3-29**).

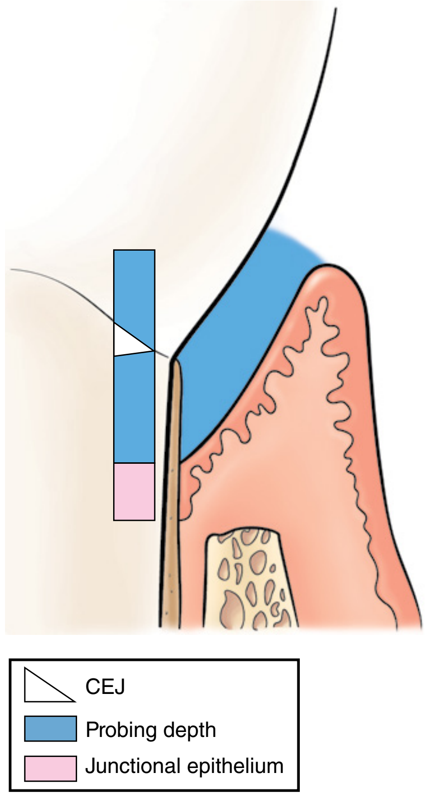

Figure 3-28 Suprabony Pocket. Characteristics of a suprabony pocket are (1) horizontal bone loss and (2) a pocket base located coronal to (above) the crest of the alveolar bone.

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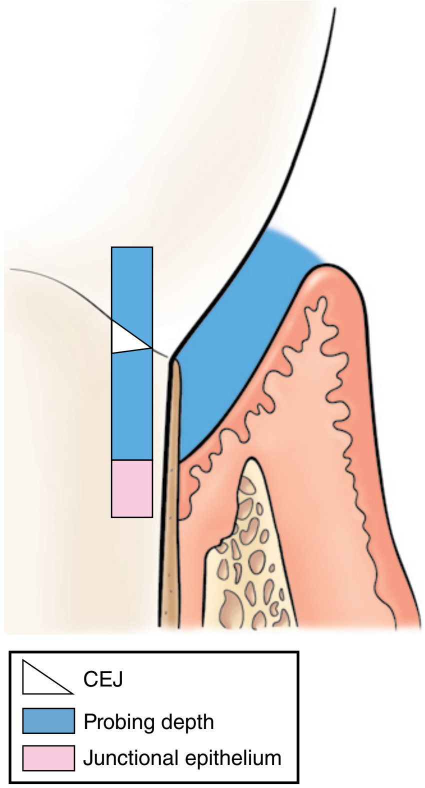

Figure 3-29 Infrabony Pocket. Characteristics of an infrabony pocket are (1) vertical bone loss and (2) a pocket base located below the crest of the alveolar bone within a trench-like area of the bone.

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Section 4

Theories of Disease Progression

For years, clinical researchers have been trying to find an answer to the question, "How does untreated periodontal disease progress?" In this context, disease progression means that the disease gets worse. Data from ongoing studies suggest that the pattern of disease progression may vary from (1) one individual to another, (2) one site to another in a person's mouth, and (3) one type of periodontal disease to another. **Figures 3-30** and **3-31** contrast the historical theory of disease progression with the current theory of periodontal disease progression.

1. **Historical Perspective on Disease Progression**
   a. **Continuous Progression Theory (Historical View of Disease Progression: Prior to 1980).** According to the **continuous disease progression theory**, periodontal disease progresses throughout the entire mouth in a slow and constant rate over the adult life of the patient.
      1. All cases of untreated gingivitis progress to periodontitis.
      2. All cases of periodontitis progress at a slow and steady rate of tissue destruction.
      3. However, research conducted in the early 1980s indicated that periodontal disease does not progress at a constant rate nor affect all areas of the mouth simultaneously. The continuous progression theory did not accurately reflect the complex nature of periodontal disease.

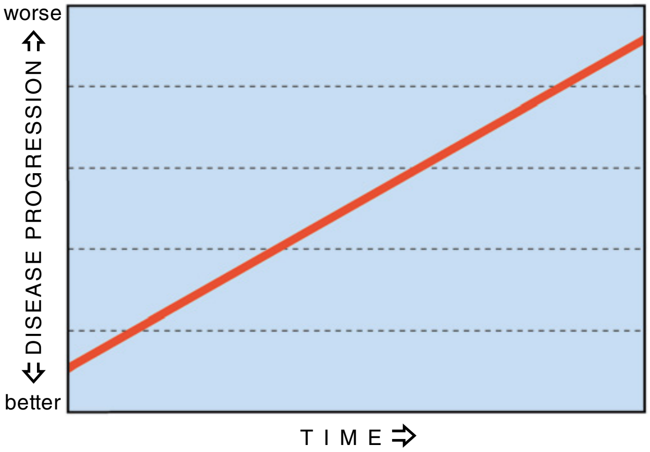

Figure 3-30 The Continuous Disease Model of Disease Progression (Prior to 1980). In the past, clinicians theorized that periodontal disease progressed (worsened) throughout the entire mouth in a slow and constant rate over the life of the patient. It was believed that all cases of untreated gingivitis led to periodontitis.

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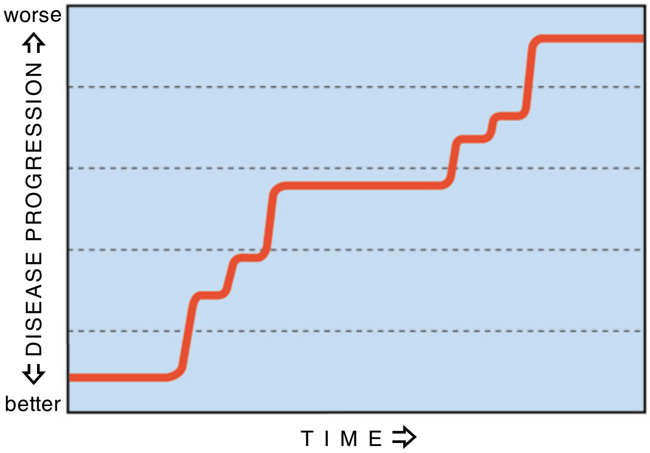

Figure 3-31 Intermittent Disease Progression Theory. Current research suggests that periodontal disease is characterized by periods of disease activity and inactivity. Furthermore, destruction does not occur in all parts of the mouth at the same time.

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2. **Current Theory of Disease Progression**
   a. **Intermittent Progression Theory (Current View). Intermittent disease progression theory** states that periodontal disease is characterized by periods of disease activity and inactivity (remission).
      1. Tissue destruction is sporadic, with short periods of tissue destruction alternating with periods of disease inactivity (no tissue destruction). The period of inactivity with no disease progression may last for months or for a much longer period.
      2. Tissue destruction progresses at different rates throughout the mouth. Destruction does not occur in all parts of the mouth at the same time. Instead, tissue destruction occurs in only a few specific sites (tooth surfaces) at a time.
      3. In most cases, untreated gingivitis does not progress to periodontitis.
      4. Different forms of periodontitis may progress at widely different rates.
      5. Susceptibility to periodontitis varies greatly from individual to individual and appears to be determined by the host response to periodontal pathogens.

Section 5

Epidemiology of the Diseases of the Periodontium

Many generations of researchers have asked the question, "What causes periodontal disease?" This section discusses the study of disease in the population (epidemiology) and reviews historical and current perspectives on the causes and progression of periodontal disease.

1. **What is epidemiology?**
   a. **Epidemiology** is the study of the health and disease within the total population (rather than an individual) and the behavioral, environmental, and genetic risk factors that influence health and disease. An epidemiologist assesses how much disease is prevalent in a given population, investigates possible causes, and applies results to treatment recommendations. This translates into developing measures to prevent or control disease.
      1. Epidemiological research has three objectives: (1) to determine the amount and distribution of a disease in the total population and in subgroups, (2) to investigate the causes of a disease, and (3) to apply this knowledge to the control and prevention of disease.

2. Through research of population groups, epidemiologists strive to identify the risk factors associated with disease, such as race/ethnicity, heredity, gender, physical environment, systemic factors, socioeconomic status, and personal behavior.
3. An understanding of the risk factors associated with a certain disease can lead to theories of the cause of that disease and then to treatment standards for patient care.

b. **Epidemiology of Periodontal Disease**

1. A large percentage of the adult population has periodontal disease. Epidemiologists study periodontal disease to determine its occurrence in the population and to identify risk factors for periodontal disease. Some of the questions epidemiologists ask when researching periodontal disease are illustrated in **Figure 3-32**.

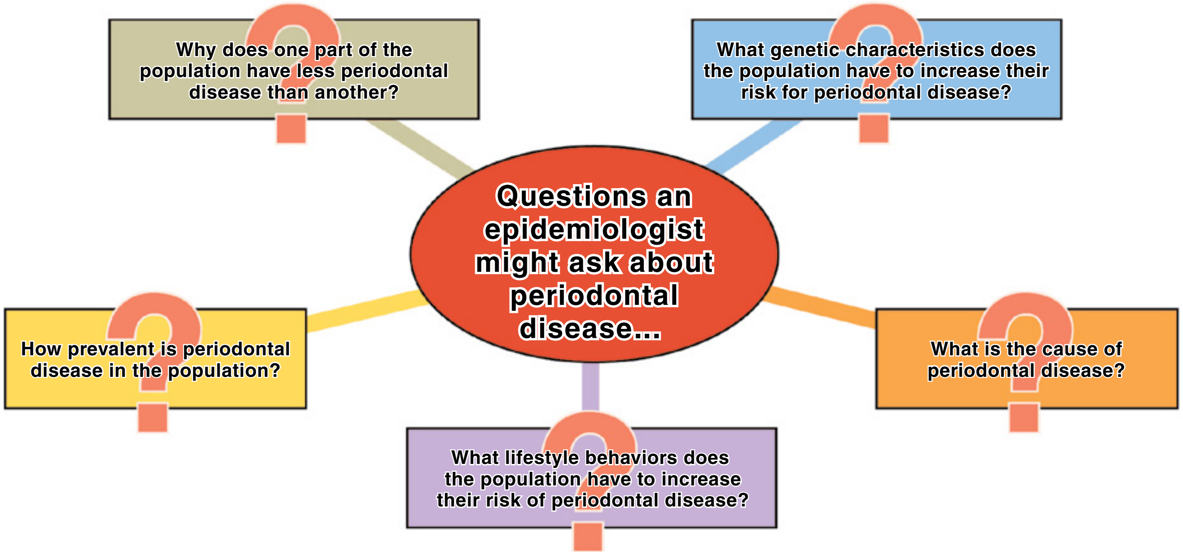
Why does one part of the population have less periodontal disease than another?
What genetic characteristics does the population have to increase their risk for periodontal disease?
Questions an epidemiologist might ask about periodontal disease...
How prevalent is periodontal disease in the population?
What is the cause of periodontal disease?
What lifestyle behaviors does the population have to increase their risk of periodontal disease?

Figure 3-32 **Researching Periodontal Disease.** This diagram illustrates the types of questions asked by epidemiologists when studying periodontal disease.

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2. Epidemiological research also provides current information to the clinical dental hygienist about methods and behaviors that are successful in the treatment and prevention of periodontal disease. Current research also may define the level of risk a patient may have for periodontal disease.
3. Studies can be designed to look at the disparities or inequities of disease patterns. For instance, a study may explore why more periodontal disease is found in a specific segment of the population than in another group of people. Oral diseases occur disproportionately more among individuals with low socioeconomic status and poor general health.[10]

2. **Prevalence and Incidence of Disease**

a. **Disease Incidence and Prevalence**

1. **Incidence** is the number of new disease cases in a population that occur over a given period. For example: A 2011 clinical study evaluated 250 white adult males with the Gingival Index (GI) to assess gingivitis. The results of this study indicated that 125 of the men had gingivitis. In a 2012 follow-up study evaluating the same patient population, 150 of the men exhibited clinical signs of gingivitis. Therefore, the incidence of new cases of gingivitis in this study population was 25 cases.
2. **Prevalence** refers to the number of all cases (both old and new) of a disease that can be identified within a specified population at a given point in time. For example: Given the above example, the prevalence of gingivitis within the total 2011 study population was 50% (125/250). On the date of examination using the GI, the clinician does not know the length of time the men have had gingivitis, and thus cannot classify them as new cases of disease (or incidence).

b. **Variables Associated with the Prevalence of Disease.** Research findings show that variables associated with the prevalence of periodontal disease include a person's gender, race, socioeconomic status, and age. The **National Health and Nutrition Examination Survey (NHANES)** is a large-scale program of studies to assess the health and nutritional status of adults and children in the United States. This survey protocol combines a survey with a physical examination. NHANES examines a nationally representative sample of about 5,000 persons each year.
   1. **Gender**
      a. Males have a greater prevalence and severity of periodontal disease than females.[11] The 2009–2010 NHANES reports periodontal disease in about 56% of men versus 38% of women.[12]
      b. It has been speculated that females tend to practice better and more frequent self-care than males. These differences in self-care behaviors may lead to the greater prevalence of disease in males.
   2. **Race/Ethnicity.** Black and Hispanic males living in the United States have poorer periodontal health and a greater incidence of periodontal disease than White males. Results from the 2009–2010 NHANES indicate a disparity with the highest prevalence of periodontal disease in Mexican Americans when compared to other races.
   3. **Education and Socioeconomic Status**
      a. Incidence of periodontal disease is greater in individuals with less than a high school education and who live below the federal poverty level.
      b. Underdeveloped countries have a higher incidence of periodontitis, possibly due to a lack of adequate information about disease prevention.
   4. **Age**
      a. Studies have shown that the severity of periodontal disease increases with age; however, the exact role that age plays in periodontal disease is difficult to assess.
         1. As people age, the chances increase that they will be exposed to additional risk factors for periodontal disease. Systemic illness, medications, and stress may contribute to disease risk in this population. Because they have less edentulism, the elderly population has a greater risk of developing periodontal disease. Results from the 2009–2010 NHANES indicate that 64% of adults older than 65 years of age were found to have moderate to severe periodontitis.
         2. Therefore, the higher incidence of periodontal disease in the elderly might not be due to age, but rather because of other risk factors that older individuals have been exposed to over their lifespan.
      b. Diminished dexterity is sometimes a problem in elderly individuals and can impact the individual's ability to perform self-care. Limited dexterity may also shorten the length of time that self-care is performed on a daily basis.
   5. **Behavior**
      a. Tobacco use has been identified as a behavioral risk factor for the development of periodontal disease. Both smoking tobacco and the use of smokeless tobacco products negatively impact the periodontium.[13,14] The 2009–2010 NHANES found that over 64% of current smokers examined had periodontal disease.
      b. With tobacco use as a risk factor, dental offices may consider stronger efforts towards including tobacco-cessation programs with patient education. Chapter 17 offers a more thorough review of the impact of tobacco on periodontal health.
   6. **Access to Dental Care.** Individuals who desire care or need care may not have access to dental care. Barriers to obtaining dental care include transportation, geographic distances to a dental office, financial expense of dental care, and time available to seek care.

c. **Measuring Disease Prevalence**
   1. The prevalence of periodontal disease in the US adult population is determined by performing clinical examinations on cross-sections of groups using indices. Indices measure the amount and severity of disease. Indices used to measure both gingivitis and periodontitis vary across epidemiological studies, as does the extent of disease present when a study begins. Refer to **Table 3-3** for a list of indices commonly used to assess periodontal disease.

Table 3-3 Commonly Used Periodontal Indices

Index	Measurement
Community and Periodontal Index of Treatment Needs (CPITN) (Federation Dentaire Internationale: Ainamo et al.)	Assesses probing depths and bleeding; developed to attain more uniform worldwide epidemiologic data; may be used for measuring group periodontal needs.
Eastman Interdental Bleeding Index (EIBI) (Abrams, Caton, and Polson; Caton and Polson)	Assesses presence of inflammation and bleeding in the interdental area upon toothpick insertion.
Gingival Bleeding Index (GBI) (Carter and Barnes)	Assesses presence of gingival inflammation by bleeding from interproximal sulcus within 10 seconds of flossing.
Gingival Index (GI) (Loe and Silness)	Assesses severity of gingivitis based on color, consistency, and bleeding on probing.
Modified Gingival Index (MGI)	Similar to GI but assesses severity of gingivitis without probing; redefined scoring for inflammation.
Periodontal Index (PI) (Russell)	Assesses the severity of gingival inflammation without probing.
Periodontal Disease Index (PDI) (Ramfjord)	Assesses the severity of gingival inflammation, pocket depth, and the level of gingival attachment.
Periodontal Screening and Recording (PSR) (American Academy of Periodontology and the American Dental Association)	Assesses periodontal health in a rapid manner, including probing depths, bleeding, and presence of hard deposits.

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2. Prevalence is affected by new cases of disease (incidence), cures or deaths within a population, and the longer lives of subjects.
3. Historically, gingival indices have used criteria to measure variables of inflammation such as color changes, presence of edema, and bleeding upon probing. Clinical indices for measuring periodontitis include variable of probing depth, clinical attachment level (CAL), and interpretation of radiographic bone levels (BL). Many studies use sample groups numbering in the thousands. Several groups are then compared and statistically analyzed. Epidemiologists will have different approaches to research and will include different variables in studies. The selected population can be studied over time.

d. **Difficulties in Measurement of Periodontal Disease**
   1. It is far easier to evaluate a population for prevalence and incidence of dental caries than for periodontal disease because dental caries lend themselves to more objective measurement. The development and process of dental caries is well known and involves only tooth structure. The **Centers for Disease Control and Prevention (CDC)** has provided guidance and standardized methods for public health surveillance of dental caries for many years.
   2. Periodontal disease, in contrast, involves both hard and soft tissues and has multiple variables that must be considered. Determining the presence of gingivitis with or without the presence of periodontitis further complicates the assessment of disease. Assessment can include:
      a. Soft tissue color changes (redness)
      b. Tissue swelling (edema)
      c. Loss of periodontal ligament fibers that support the teeth
      d. Loss of alveolar bone/furcation involvement
      e. Bleeding upon probing/spontaneous bleeding
      f. Probing depths

3. The multiple variables used to define periodontal disease make the numbers for prevalence and incidence of periodontal disease less specific, more of a range, and more subject to change. NHANES most recently used the definition/classification of periodontal disease, as determined by the collaboration of CDC and the American Academy of Periodontology (AAP) to assess the population.
4. Historically, national periodontal health surveys (NHANES) have used partial mouth examination protocols that may not accurately represent disease because periodontal disease is not evenly distributed in the entire mouth. For example, attachment loss can vary on tooth surfaces being examined. The 2009–2010 NHANES did use a full-mouth examination and is being described as the most comprehensive survey of periodontal health in the United States.[15]

e. **What the Research Shows.** Research on periodontal disease indicates that it is one of the most widespread diseases in adult Americans, with most individuals who have periodontal disease being unaware of its presence. These findings are based on data collected as part of the CDC's 2009–2010 NHANES, designed to assess the health and nutritional status of adults and children in the United States. The 2009–2010 NHANES included for the first time a full-mouth periodontal examination to assess for mild, moderate, or severe periodontitis, making it the most comprehensive survey of periodontal health ever conducted in the United States. Researchers measured periodontitis because it is the most destructive form of periodontal disease. Gingivitis, the earliest stage of periodontal disease, was not assessed.

1. **Prevalence or Periodontitis**
   a. A study titled "Prevalence of Periodontitis in Adults in the United States: 2009 and 2010" estimates that 47.2%, or 64.7 million, American adults have mild, moderate, or severe periodontitis. In adults 65 and older, prevalence rates increase to 70.1%.[15] These findings also indicate disparities among certain segments of the US population. Periodontal disease is higher in men than women (56.4% vs. 38.4%) and is highest in Mexican Americans (66.7%) compared to other races. Other segments with high prevalence rates include current smokers (64.2%), those living below the federal poverty level (65.4%), and those with less than a high school education (66.9%).
   b. According to data from 2009–2010 NHANES, over 47% of the population had periodontitis:
      1. 8.7% had mild periodontitis.
      2. 30.0% had moderate periodontitis.
      3. 8.5% had severe periodontitis.
   c. According to data from 2009–2010 NHANES, in adults over 65 years of age, 64% had moderate/severe periodontitis.
2. **Prevalence of Periodontitis**
   a. The presence of periodontal disease is measured clinically in several ways. NHANES 2009–2010 used attachment loss and probing depths to assess periodontal disease. Loss of attachment is the term used to describe the destruction of periodontal ligament fibers and alveolar bone that support the teeth. The study found that attachment loss of 4 mm or more affects approximately half of adults aged 50 to 59.[12]
   b. By age 60 to 69, less than half of all adults in the United States have retained 21 teeth or more.[12]

f. **Public Health Surveillance of Periodontal Disease.** The most recent NHANES data suggests that the amount of periodontal disease may have been underestimated in the general population.[15] Both the AAP and the CDC describe periodontal disease as a public health concern and are working toward improved disease surveillance. The CDC's Division of Oral Health and the Association of State and Territorial Dental Directors collaborate to monitor the burden of oral disease and track state data sources. As the World Health Organization and oral health professionals have reported, periodontal disease impairs the quality of life and general well-being for individuals and efforts are ongoing to create awareness of periodontal disease as a public health issue.[16]

Chapter Summary Statement

Periodontal pathogenesis is the sequence of events that occurs during the development of periodontal disease. The two types of periodontal disease are gingivitis and periodontitis:

- Gingivitis is a reversible condition that is characterized by changes in the color, contour, and consistency of the gingiva. Apical migration of the junctional epithelium or bone loss does not occur in gingivitis.
- Periodontitis results in some extent of permanent tissue destruction characterized by pocket formation, destruction of the periodontal ligament fibers, and resorption of alveolar bone. The pattern of alveolar bone loss and periodontal ligament destruction depends on the pathway that the inflammatory process takes as it spreads from the gingiva into the alveolar bone. It is the destruction of periodontal ligament fibers and resorption of alveolar bone that leads to tooth mobility and the possibility of tooth loss.

Epidemiological research of periodontal disease has three objectives: (1) to determine the amount and distribution of periodontal disease in a population, (2) to investigate the causes of periodontal disease, and (3) to apply this knowledge to the control of periodontal disease.

Advances in research have led to many changes in the understanding, prevention, and treatment of periodontal disease. In the future, ideas about causes and treatment will continue to be refined and changed as researchers delve further into the mysteries of periodontal disease.

Section 6

Focus on Patients

Clinical Patient Care

CASE 1

Your patient has 6- to 7-mm attachment loss on all surfaces of the maxillary first molar. Which of the tissues of the periodontium have experienced tissue destruction surrounding this tooth?

CASE 2

Your dental team provides appropriate therapy for a patient with periodontitis. When you began treatment, your initial findings were redness and edema (swelling) of the gingiva, bleeding on probing, periodontal pockets, and attachment loss. Successful control of the periodontal disease in the patient should *not* be expected to result in elimination of which of these initial clinical findings?

CASE 3

You find a newspaper article that estimates that in your home state 73% of state residents have some form of periodontal disease. You would like to use this statistic in a homework assignment. When you include this information in your homework assignment should you describe this statistic as incidence or prevalence of periodontitis?

CASE 4

Your dental team has a new patient who has gingivitis. The patient has poor daily self-care, generalized calculus deposits, poorly controlled diabetes mellitus, a history of smoking cigarettes, and inadequate dietary intake of calcium. In your patient counseling, how would you characterize the likelihood that the patient will develop periodontitis in the future and what might you tell the patient about this?

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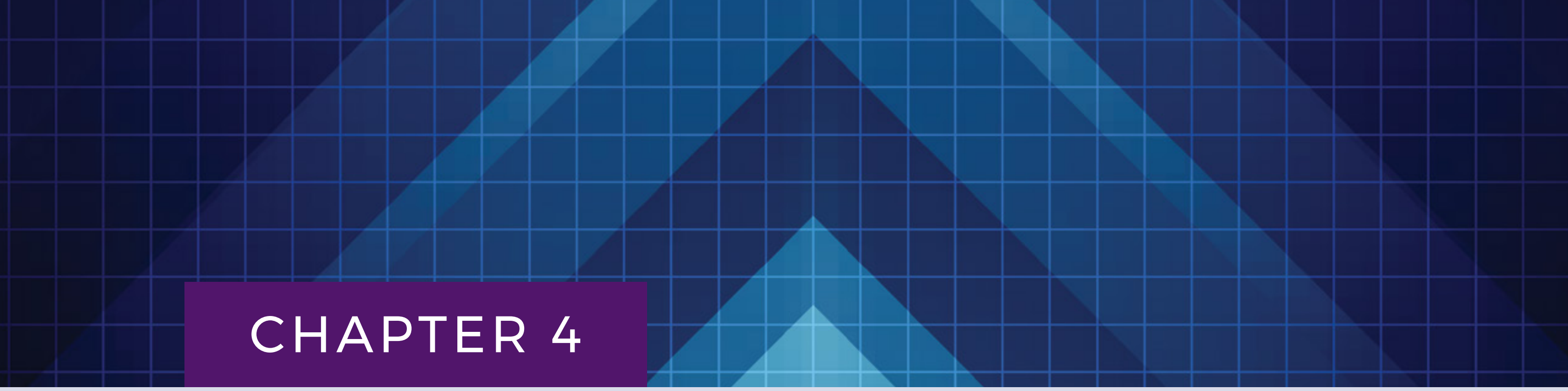

CHAPTER 4

Classification of Periodontal and Peri-Implant Diseases and Conditions

- Section 1 Disease Classification Systems
  - The 2017 AAP/EFP Classification of Periodontal and Peri-Implant Diseases and Conditions
- Section 2 The 2017 Classification of Periodontal and Peri-Implant Diseases and Conditions
  - Health, Gingivitis, and Gingival Diseases/Conditions
  - Forms of Periodontitis
  - Other Conditions Affecting the Periodontium
  - Peri-Implant Diseases and Conditions
  - Comparison of the 2017 and 1999 Classification Systems
- Section 3 Focus on Patients
  - Clinical Patient Care

CLINICAL APPLICATION

A periodontal disease classification system assists the clinician in diagnosing and treating periodontal and peri-implant diseases and conditions, communicating clinical findings accurately to other dental health care providers, and submitting information to dental insurance providers. In 2017, the American Academy of Periodontology and the European Federation of Periodontology developed a new classification scheme for periodontal and peri-implant diseases and conditions. This joint collaborative effort was undertaken to better align and update the classification system with our current understanding of periodontal and peri-implant diseases and conditions. Upcoming chapters will go into greater detail regarding the case definition and diagnostic criteria for each category. The purpose of this chapter is to provide the reader with a broad overview of the 2017 Classification of Periodontal and Peri-Implant Diseases and Conditions.

LEARNING OBJECTIVES

- Explain the rationale for a classification system for periodontal disease.
- List the three major categories of periodontal diseases and conditions.
- Explain why clinicians need to be familiar with terminology from the 1999 disease classification, such as *chronic periodontitis* and *aggressive periodontitis*.
- Compare and contrast the 2017 and the 1999 classification systems.
- List the four subcategories that fall under the Peri-Implant Diseases and Conditions category.

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KEY TERMS

Classification system
American Academy of Periodontology
European Federation of Periodontology
2017 AAP/EFP Classification of Periodontal and Peri-Implant Diseases and Conditions

Section 1

Disease Classification Systems

A **classification system** is a grouping of similar entities on the basis of certain differing characteristics. An everyday example of a classification system is grouping motor vehicles into categories such as passenger cars, crossover vehicles, sport utility vehicles, and trucks. Classification systems provide a tool to understanding the etiology, pathogenesis, and treatment of periodontal diseases in an orderly manner.

Clinicians and researchers have struggled to develop a classification of the various forms of periodontal disease. Over the years, classification systems have changed in response to new scientific knowledge about the etiology and pathogenesis of periodontal diseases and conditions. In recent years, several attempts have been made to classify periodontal diseases. *A disease classification system, however, should not be regarded as a permanent structure. Rather, it is ever-changing and evolves with the development of new evidence-based knowledge. Thus, it is expected that systems of classification will change over time.* As more is learned about the nature of a disease, the disease classification system is reassessed and redefined.

A periodontal classification system provides information necessary in (1) communicating clinical findings accurately to other dental health care providers, (2) formulating a diagnosis and devising an individualized treatment plan, (3) predicting treatment outcomes (prognosis), and (4) submitting information to dental insurance providers/carriers.

The 2017 AAP/EFP Classification of Periodontal and Peri-Implant Diseases and Conditions

The **American Academy of Periodontology (AAP)** and the **European Federation of Periodontology (EFP)** initiated the currently accepted classification system by organizing the World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions held in November 2017. This workshop brought together more than a hundred experts from across the world to review the scientific evidence and develop a new disease classification.

The previous classification of periodontal disease was developed in 1999. Since then, periodontics has made notable advancements in the treatment and diagnosis of periodontal disease. Furthermore, peri-implantitis has emerged as a major new problem that was not accounted for in the 1999 classification system. The 2017 disease classification accounts for scientific advances in recent years and strives to standardize the definition of the diseases and conditions. The **2017 AAP/EFP Classification of Periodontal and Peri-Implant Diseases and Conditions** is the most recent, internationally accepted classification system.[1]

Figure 4-1 presents a visual overview of the 2017 Classification of Periodontal and Peri-Implant Diseases and Conditions. **Box 4-1** provides the link to the complete proceedings of the World Workshop.

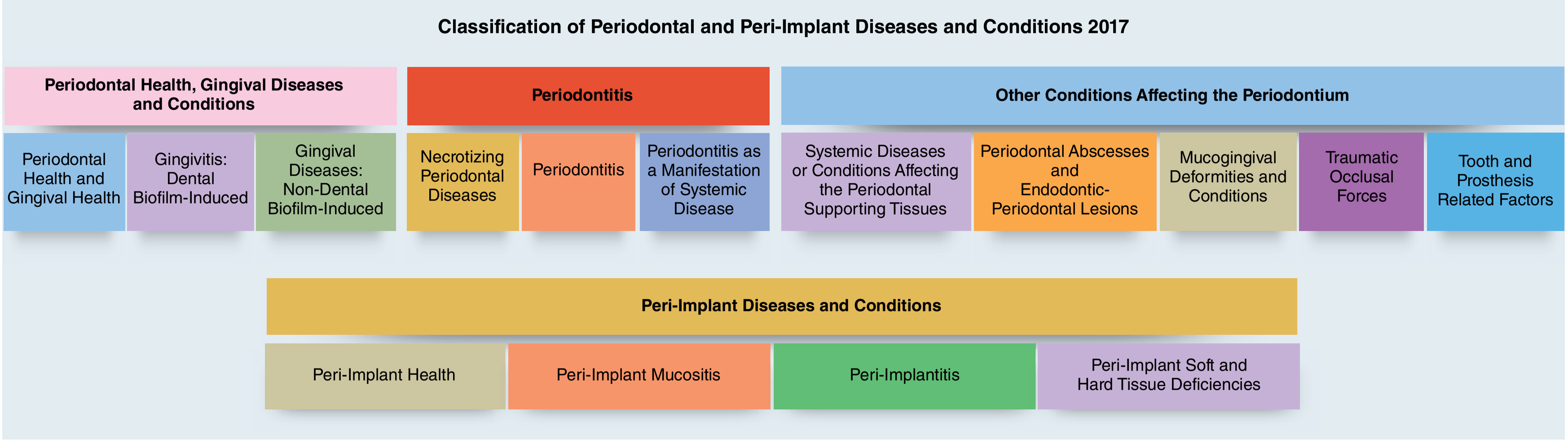
Classification of Periodontal and Peri-Implant Diseases and Conditions 2017
Periodontal Health, Gingival Diseases and Conditions
Periodontitis
Other Conditions Affecting the Periodontium
Periodontal Health and Gingival Health
Gingivitis: Dental Biofilm-Induced
Gingival Diseases: Non-Dental Biofilm-Induced
Necrotizing Periodontal Diseases
Periodontitis
Periodontitis as a Manifestation of Systemic Disease
Systemic Diseases or Conditions Affecting the Periodontal Supporting Tissues
Periodontal Abscesses and Endodontic-Periodontal Lesions
Mucogingival Deformities and Conditions
Traumatic Occlusal Forces
Tooth and Prosthesis Related Factors
Peri-Implant Diseases and Conditions
Peri-Implant Health
Peri-Implant Mucositis
Peri-Implantitis
Peri-Implant Soft and Hard Tissue Deficiencies

Figure 4-1 **The 2017 AAP/EFP Classification of Periodontal and Peri-Implant Diseases and Conditions.** An overview of the 2017 classification system.

Modified from Caton JG, Armitage G, Berglundh T, et al. A new classification scheme for periodontal and peri-implant diseases and conditions—introduction and key changes from the 1999 classification. *J Periodontol*. 2018;89(S1):S1–S8. doi:10.1002/JPER.18-0157. Used with permission by John Wiley & Sons.

Box 4-1 Link to the Complete Proceedings of World Workshop

The complete proceedings of the World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions is available free of charge online at https://onlinelibrary.wiley.com/toc/19433670/2018/89/S1.

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Section 2

The 2017 Classification of Periodontal and Peri-Implant Diseases and Conditions

Table 4-1 presents the 2017 Disease Classification for periodontal health, gingivitis, and gingival diseases and conditions. As shown in **Figure 4-2**, this broad category is subdivided into three subcategories: (1) periodontal and gingival health, (2) dental biofilm–induced gingivitis, and (3) nondental biofilm–induced gingival diseases. Each of these three major subcategories has several conditions listed under it (refer to Table 4-1).

Health, Gingivitis, and Gingival Diseases/Conditions

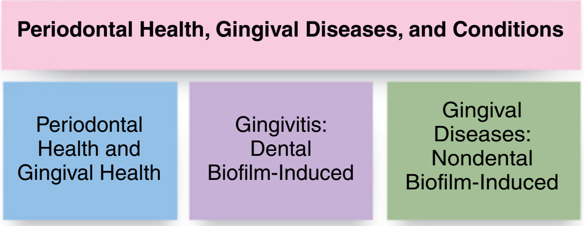
Periodontal Health, Gingival Diseases, and Conditions
Periodontal Health and Gingival Health
Gingivitis: Dental Biofilm-Induced
Gingival Diseases: Nondental Biofilm-Induced

Figure 4-2 Classification of Periodontal Health, Gingivitis, and Gingival Diseases/Conditions. The 2017 Disease Classification of Periodontal and Peri-Implant Diseases and Conditions organization of periodontal and gingival health, dental biofilm–induced gingivitis, and nondental biofilm–induced gingival diseases.

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Table 4-1 Periodontal Health, Gingivitis, and Gingival Diseases/Conditions

1. Periodontal health and gingival health
    a. Clinical gingival health on an intact periodontium
    b. Clinical gingival health on a reduced periodontium
        i. Stable periodontitis patient
        ii. Non-periodontitis patient
2. Gingivitis—dental biofilm–induced
    a. Associated with dental biofilm alone
    b. Mediated by systemic or local risk factors
    c. Drug-influenced gingival enlargement
3. Gingival diseases—nondental biofilm–induced
    a. Genetic/developmental disorders
    b. Specific infections
    c. Inflammatory and immune conditions
    d. Reactive processes
    e. Neoplasms
    f. Endocrine, nutritional, and metabolic diseases
    g. Traumatic lesions
    h. Gingival pigmentation

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Forms of Periodontitis

As shown in **Figure 4-3**, the major diagnostic category of periodontitis is subdivided into three forms. **Table 4-2** presents the 2017 classification framework for periodontitis.

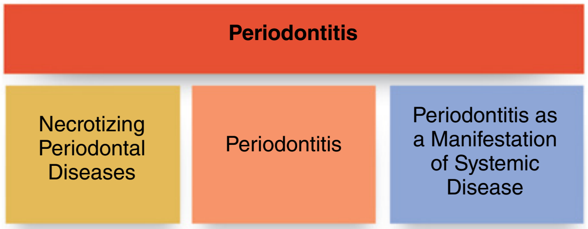
Periodontitis
Necrotizing Periodontal Diseases
Periodontitis
Periodontitis as a Manifestation of Systemic Disease

Figure 4-3 Three Major Forms of Periodontitis. The 2017 Disease Classification of Periodontal and Peri-Implant Diseases and Conditions subdivides the category of periodontitis into three major forms. Each of the three forms has two or more subcategories.

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Table 4-2 Forms of Periodontitis

1. Necrotizing periodontal diseases
    a. Necrotizing gingivitis
    b. Necrotizing periodontitis
    c. Necrotizing stomatitis
2. Periodontitis as manifestation of systemic diseases. Classification of these conditions should be based on the primary systemic disease according to the International Statistical Classification of Diseases and Related Health Problems (ICD) codes.
3. Periodontitis
    a. Stages: Based on severity[a] and complexity of management[b]
        Stage I: Initial periodontitis
        Stage II: Moderate periodontitis
        Stage III: Severe periodontitis with potential for additional tooth loss
        Stage IV: Severe periodontitis with potential for loss of the dentition
    b. Extent and distribution[c]: Localized, generalized; molar-incisor distribution
    c. Grades: Evidence or risk of rapid progression,[d] anticipated treatment response[e]
        Grade A: Slow rate of disease progression
        Grade B: Moderate rate of disease progression
        Grade C: Rapid rate of disease progression

[a] Severity: Interdental clinical attachment level (CAL) at site with greatest loss; Radiographic bone loss and tooth loss.
[b] Complexity of management: Probing depths, pattern of bone loss, furcation lesions, number of remaining teeth, tooth mobility, ridge defects, masticatory dysfunction.
[c] Add to stage as descriptor: Localized <30% teeth involved, generalized ≥30% teeth involved.
[d] Risk of progression: Direct evidence by periapical radiographs of CAL loss, or indirect (bone loss:age ratio).
[e] Anticipated treatment response: Case phenotype, smoking, hyperglycemia (elevated levels of glucose in blood).
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Other Conditions Affecting the Periodontium

As shown in **Figure 4-4**, this broad diagnostic category is subdivided into five conditions. **Table 4-3** presents the 2017 classification framework for other conditions affecting the periodontium.

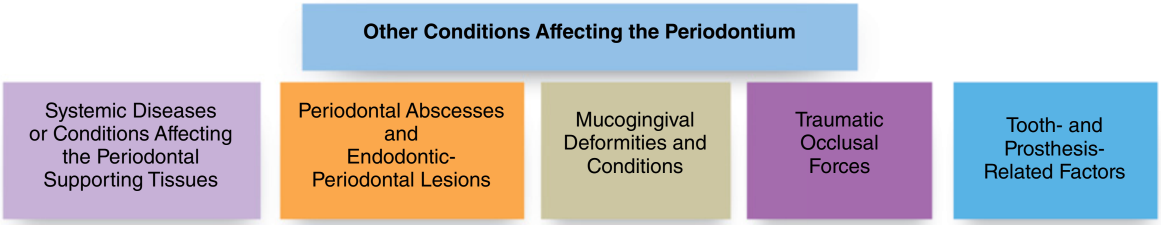
Other Conditions Affecting the Periodontium
Systemic Diseases or Conditions Affecting the Periodontal Supporting Tissues
Periodontal Abscesses and Endodontic-Periodontal Lesions
Mucogingival Deformities and Conditions
Traumatic Occlusal Forces
Tooth- and Prosthesis-Related Factors

Figure 4-4 **Other Conditions Affecting the Periodontium.** The 2017 Disease Classification of Periodontal and Peri-Implant Diseases and Conditions includes a category for other conditions that may affect the health of the periodontium.

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Table 4-3 **Other Conditions Affecting the Periodontium**

1. Systemic diseases or conditions affecting the periodontal supporting tissues
2. Periodontal abscesses and endodontic-periodontal lesions
3. Mucogingival deformities and conditions around teeth
    a. Gingival phenotype
    b. Gingival/soft tissue recession
    c. Lack of gingiva
    d. Decreased vestibular depth
    e. Aberrant frenum/muscle position
    f. Gingival excess
    g. Abnormal color
    h. Condition of exposed root surface
4. Traumatic occlusal forces
    a. Primary occlusal trauma
    b. Secondary occlusal trauma
    c. Orthodontic forces
5. Prostheses and tooth-related factors that modify or predispose to plaque-induced gingival diseases/periodontitis
    a. Localized tooth-related factors
    b. Localized dental prostheses–related factors

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Peri-Implant Diseases and Conditions

As shown in **Figure 4-5**, this broad diagnostic category is subdivided into four subcategories. **Table 4-4** presents the 2017 classification framework for peri-implant diseases and conditions.

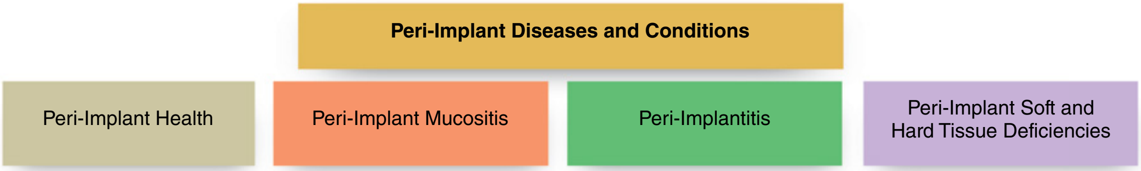
Peri-Implant Diseases and Conditions
Peri-Implant Health
Peri-Implant Mucositis
Peri-Implantitis
Peri-Implant Soft and Hard Tissue Deficiencies

Figure 4-5 **Peri-Implant Diseases and Conditions.** The 2017 Disease Classification of Periodontal and Peri-Implant Diseases and Conditions subdivides the category of peri-implant diseases and conditions into four subcategories.

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Table 4-4 **Peri-Implant Diseases and Conditions**

1. Peri-implant health
2. Peri-implant mucositis
3. Peri-implantitis
4. Peri-implant soft and hard tissue deficiencies

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Comparison of the 2017 and 1999 Classification Systems

Much of the periodontal literature found in periodontal journals and textbooks is based on the earlier 1999 disease classification. For example, the classifications of chronic and aggressive periodontitis will appear in journal articles printed before June 2018. For this reason, readers need to be somewhat familiar with both the 1999 and 2017 classification systems. The 1999 disease classification is outlined in **Table 4-5**.[2]

Table 4-5 Main Categories: 1999 Classification of Periodontal Diseases and Conditions

1. Gingival Diseases
    a. Plaque-Induced Gingival Diseases
    b. Non-Plaque-Induced Gingival Lesions
2. Chronic Periodontitis
3. Aggressive Periodontitis
4. Periodontitis as a Manifestation of Systemic Diseases
5. Necrotizing Periodontal Diseases
6. Abscesses of the Periodontium
7. Periodontitis Associated with Endodontic Lesions
8. Developmental or Acquired Deformities and Conditions

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The 2017 classification system differs from the 1999 classification system in several critical ways that are briefly outlined below:

1. The updated classification system introduces a new category: “Periodontal/Gingival Health.” This is valuable because some healthy patients may present with the distinctive clinical and radiographic features that are more consistent with a diagnosis of periodontal health, rather than periodontal disease.
2. Inclusion of a case definition of an “intact periodontium” (no previous loss of periodontal tissues) versus a “reduced periodontium” (previous or ongoing loss of periodontal tissues). A reduced periodontium may be observed in either a successfully stable periodontitis patient or in a non-periodontitis patient. Under previous classification systems, any site with clinical attachment loss or bone loss was deemed to be a periodontitis site regardless of the cause. The current classification system, however, accounts for attachment loss or bone loss that could be attributed to reasons other than periodontitis, such as traumatic toothbrushing; history of orthodontic therapy; or previous history of periodontal surgery, such as surgical crown lengthening.
3. Forms of the disease that were previously recognized as “chronic” or “aggressive” are now grouped into the single disease entity known as “periodontitis.” The 2017 classification system no longer has a chronic periodontitis category and a separate aggressive periodontitis category because there is little consistent evidence that aggressive and chronic periodontitis are different diseases. Furthermore, there is no evidence of a specific pathophysiology that is distinct for either form of periodontitis.
4. A classification system for periodontitis that is based on a multidimensional staging and grading system, much like a system used for cancer classification. Using the combined staging and grading system, the provider can individualize patient-specific treatment modalities and treatment sequences that will enable him or her to optimize individual patient management, and thus serve as the launchpad for personalized patient care.
5. A new classification category that accounts for peri-implant diseases and conditions.

Chapter Summary Statement

This chapter presents an overview of the updated classification system of periodontal diseases and conditions and a new classification of peri-implant diseases and conditions. The 2017 classification represents the work of the worldwide community of scholars and clinicians in periodontology and implant dentistry.

Classification systems, such as the one for periodontal diseases and conditions, group similar diseases and conditions into general categories. Classification systems provide a tool to study the etiology, pathogenesis, and treatment of periodontal diseases in an orderly manner. From the clinician’s point of view, the classification system is a framework that provides a starting point for formulating a diagnosis and an individualized treatment plan. In addition, periodontal disease classifications assist the clinician in communicating with other dental health care providers and dental insurance providers.

Section 3

Focus on Patients

Clinical Patient Care

CASE 1

During the assessment of the periodontium, you note the following: apical migration of the junctional epithelium and attachment loss. The patient's medical history is normal; he takes no medications. According to the 2017 disease classification, would this patient be classified as having gingivitis, periodontitis, or periodontitis as a manifestation of systemic disease?

CASE 2

During the assessment of the periodontium, you note the following: bleeding, redness, and swelling of the tissue; abundant plaque biofilm; no apical migration of the junctional epithelium; and no attachment loss. The patient's medical history is normal; he takes no medications. According to the 2017 disease classification, would this patient be classified as having gingivitis, periodontitis, or periodontitis as a manifestation of systemic disease?

CASE 3

During the assessment of the periodontium, you note the following: bleeding, redness, and swelling of the tissue; abundant plaque biofilm; apical migration of the junctional epithelium; and attachment loss. The patient takes medication for diabetes. According to the 2017 disease classification, would this patient be classified as having gingivitis, periodontitis, or periodontitis as a manifestation of systemic disease?

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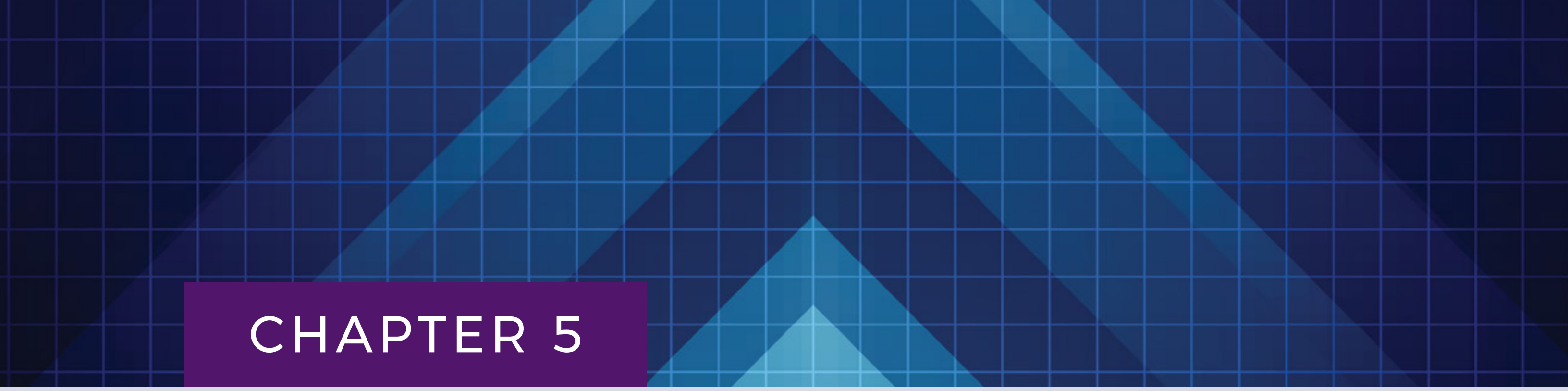

CHAPTER 5

Periodontal Health and Gingival Diseases/Conditions

- Section 1 Periodontal Health/Gingival Health
  - Case Definitions and Diagnostic Considerations
  - 2017 AAP/EFP Subcategories of Periodontal Health
- Section 2 Dental Biofilm-Induced Gingivitis (also commonly referred to as dental plaque-induced gingivitis)
  - Case Definitions and Diagnostic Considerations of Dental Biofilm-Induced Gingivitis
  - Extent and Distribution of Dental Biofilm-Induced Gingivitis
  - 2017 AAP/EFP Subcategories of Dental Biofilm-Induced Gingivitis
  - Modifying Factors of Dental Biofilm-Induced Gingivitis
- Section 3 Non–Dental Biofilm-Induced Gingival Diseases and Conditions (also commonly referred to as non-dental plaque-induced gingival diseases)
  - Description of Selected Disease Disorders
  - Description of Selected Inflammatory and Immune Conditions and Lesions
- Section 4 Focus on Patients
  - Clinical Patient Care
  - Ethical Dilemma

CLINICAL APPLICATION

Careful assessment of the periodontium is part of each routine dental examination. In this context, a thorough visual, clinical, and radiographic evaluation of the patient's periodontal tissues provides the dental practitioner with invaluable insight that is critical to determining the health status of the periodontium. As a critical member of the dental team, dental hygienists are often the first to detect the early signs of periodontal disease. In 2017, the American Academy of Periodontology (AAP) and the European Federation of Periodontology (EFP) developed a new worldwide classification scheme for periodontal and peri-implant diseases and conditions. Included in the new classification scheme is the category "periodontal health, gingival diseases/conditions." The focus of this chapter will primarily be on underscoring the key characteristics and features that define this broad category. Appreciating what constitutes as periodontal health and what constitutes as gingival diseases/conditions serves as the basis for the dental provider to have a foundational understanding of the signs and symptoms consistent with the different categories of gingival diseases and conditions that are commonly encountered in clinical practice. This chapter will also serve as a springboard for highlighting more advanced forms of periodontal disease—such as periodontitis and peri-implant diseases—in upcoming chapters.

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LEARNING OBJECTIVES

- Define *periodontal health* and be able to describe the clinical features that are consistent with periodontal health.
- Compare and contrast the etiologic factors associated with dental biofilm-induced gingivitis and non–dental biofilm-induced gingival diseases and conditions.
- List the conditions that are classified under the non-dental biofilm-induced gingival diseases and conditions category.
- Describe the differences between an intact periodontium and a reduced periodontium.
- Differentiate between papillary gingivitis, marginal gingivitis, and diffuse gingivitis.
- Describe the clinical signs which are characteristic of dental biofilm-induced gingivitis.
- List the systemic factors that modify the host response to plaque biofilm and contribute to gingival inflammation.

KEY TERMS

Periodontal health
Intact periodontium
Reduced periodontium
Periodontal health on an intact periodontium
Periodontal health on a reduced periodontium in a non-periodontitis patient
Periodontal health on a reduced periodontium in a successfully treated stable periodontitis patient
Dental biofilm-induced gingivitis (may also be referred to as dental plaque-induced gingivitis)
Non-dental biofilm-induced gingival diseases and conditions (may also be referred to as non-dental plaque-induced gingival diseases and conditions)
Extent of inflammation
Distribution of inflammation
Papillary gingivitis
Marginal gingivitis
Diffuse gingivitis
Gingivitis on an intact periodontium
Gingivitis on a reduced periodontium in a non-periodontitis patient
Gingival inflammation on a reduced periodontium in a successfully treated stable periodontitis patient
Pregnancy-associated pyogenic granuloma
Hyperglycemia
Leukemia
Drug-influenced gingival enlargements
Lichen planus

Section 1

Periodontal Health/Gingival Health

Case Definition and Diagnostic Considerations

The 2017 World Workshop defines **periodontal health** as simply "*a state free from inflammatory periodontal disease that allows an individual to function normally and avoid consequences (mental or physical) due to current or past disease.*"[1]

1. **Characteristics of Periodontal Health.** Clinically, periodontal health is characterized by the *absence* of the following: bleeding on probing, erythema, edema, attachment loss, and alveolat bone loss.
   a. **Tissue Color in Health**
      1. Healthy keratinized gingival tissue has a uniform, pink color. The shade of pink depends on the number and size of blood vessels in the connective tissue and the thickness of the gingival epithelium.
      2. The shade of pink may also be influenced by an individual's skin complexion.
         a. In individuals with fair complexions, the gingiva has a pale pink appearance (**Figure 5-1A**).
         b. In individuals with darker complexions, the gingiva may appear more pigmented and have a more diffuse dark brownish-blackish appearance. This is known as physiologic pigmentation (**Figure 5-1B**).
            i. Physiologic pigmentation is usually symmetric and can occur on the gingiva, buccal mucosa, hard palate, lips, and even the tongue.
            ii. Physiologic pigmentation results from an increase in melanin production, not an increase in the number of cells that produce melanin.

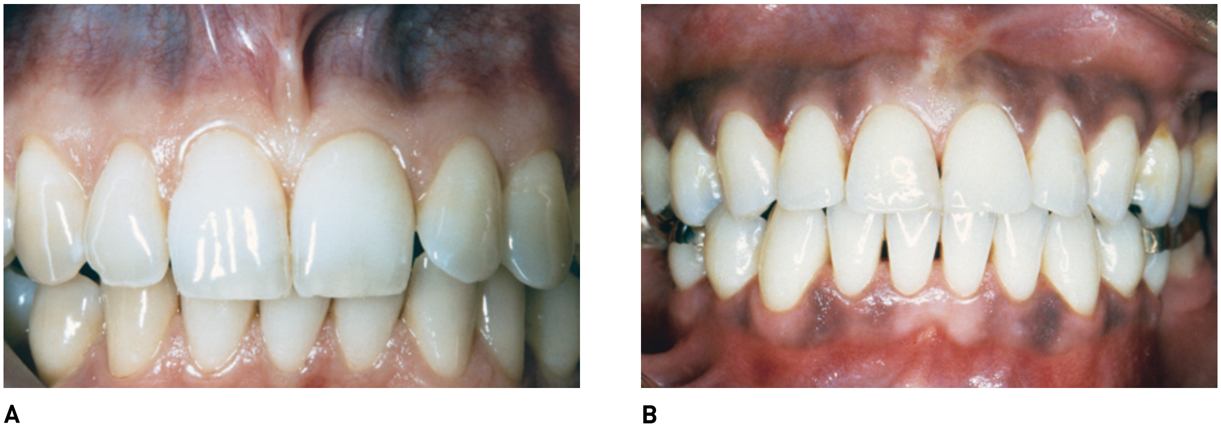

Figure 5-1 Periodontal Health and Pigmentation of the Gingiva. A. Periodontal health showing coral pink gingiva. Note the distinct difference in appearance between the keratinized gingiva and the nonkeratinized alveolar mucosa. **B.** Pigmentation of the gingiva. Note the light brown to dark brownish/blackish macules on the gingiva.

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  3. The coral pink of the gingiva is easily distinguished from the dark red alveolar mucosa.

b. **Tissue Contour (Size and Shape)**
  1. In health, the gingival margin tightly encircles the cervical region of the tooth.
  2. The contour of the healthy gingival margin is smoothly scalloped (arched) as it flows from one tooth to the other and conforms to the outline of the cementoenamel junctions (CEJ) (**Figure 5-2**).

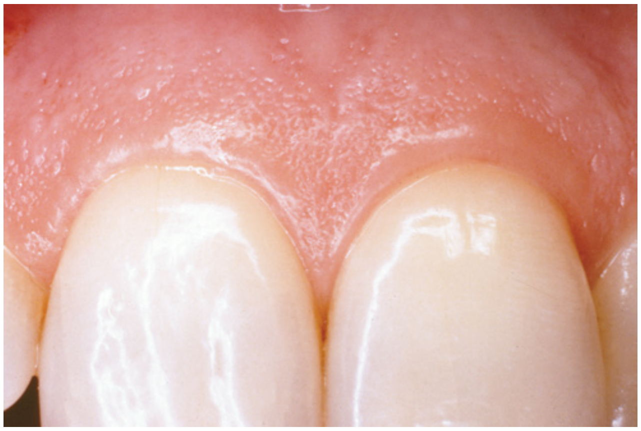

Figure 5-2 Contours of Healthy Gingiva. The gingiva in this image exhibits all the characteristics of health, including smooth scalloped, tapered gingival margins and pointed triangular papillae that completely fill the embrasure spaces (interdental spaces).

Courtesy of Dr. Don Rolfs, Periodontal Foundations, Wenatchee, WA.

  3. The healthy gingival margin meets the cervix of the tooth with a tapered (knife-edged), flat, or slightly rounded edge.
  4. Within the interdental space, the healthy gingival papilla has a pyramidal (triangular) appearance with the point of the papilla just apical to the contact point and completely fill the embrasure space (Figure 5-2). Papillae are less pointed in the posterior region because the buccal-lingual aspect is wider.
     a. Papillae are flat in areas where there is no contact between teeth.
     b. Papillae may appear narrow in areas where teeth overlap.

c. **Tissue Consistency in Health**
   1. Like the free gingiva, the attached gingiva is keratinized. However, unlike the free gingiva, the attached gingiva is firmly bound to the underlying cementum and bone.
   2. When probed, healthy tissue is firm and resilient (elastic). If gentle pressure is applied to the free gingiva with the tip of a periodontal probe, the free gingiva can be slightly retracted away from the cervix of the tooth, but it will resist compression and spring back to its normal form and shape quickly. In contrast, the attached gingiva is immovable and cannot be retracted due to its tight attachment to the underlying cementum and alveolar bone.

d. **Surface Texture of the Tissue in Health**
   1. In health, the surface of the gingiva may have a dimpled appearance similar to the skin of an orange peel.
   2. This dimpled appearance—known as stippling—appears as minute elevations and depressions of the surface of the gingiva due to connective tissue projections within the epithelial tissue (connective tissue papillae). The presence of stippling is best viewed by drying the tissue with compressed air (**Figure 5-3**).

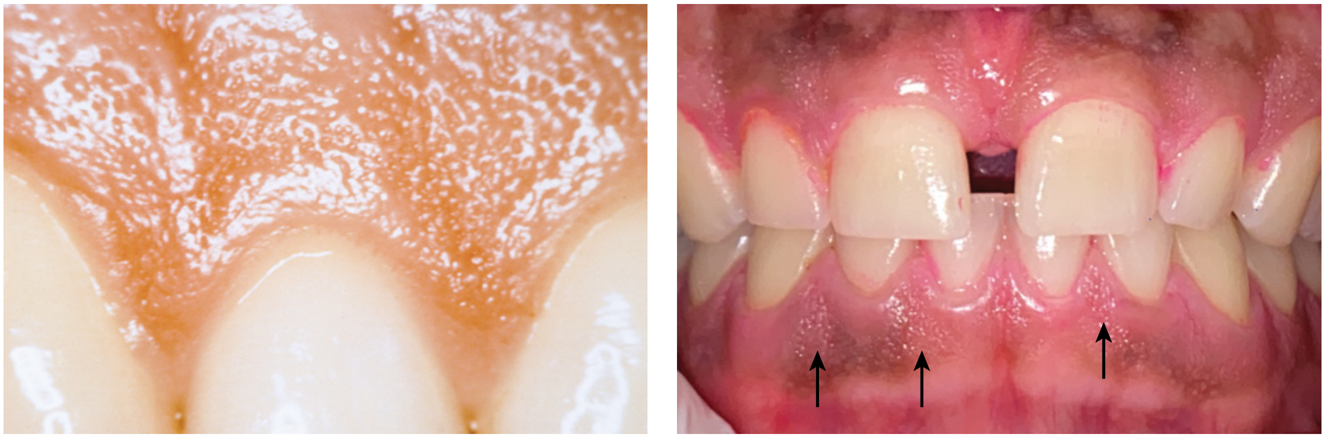

Figure 5-3 **Stippling of the Gingival Tissue.** Healthy gingival tissue showing a stippled appearance in both photographs. Stippling varies greatly from individual to individual. Note the physiologic pigmentation on the attached gingiva (right image).

Left: © Wolters Kluwer

   3. Healthy tissue may or may not exhibit a stippled appearance because the presence of stippling varies greatly from individual to individual.

e. **Position of Gingival Margin in Health**
   1. In health, the gingival margin coincides with the level of the CEJ or is ever so slightly coronal to the CEJ (**Figures 5-4** and **5-5**).
   2. A patient with a previous history of periodontitis who is currently under periodontal maintenance may present with a gingival margin that is apical to the CEJ (**Figure 5-6**) due to a prior history of disease activity.
   3. Nonetheless, the clinician should be aware that there is always the possibility that the position of the gingiva may appear apical to the CEJ due to non-periodontitis reasons, such as history of traumatic toothbrushing or a high frenum.

f. **Absence of Bleeding in Health.** Healthy tissue does not bleed when stimulated by clinical procedures such as gentle probing of the sulcus.

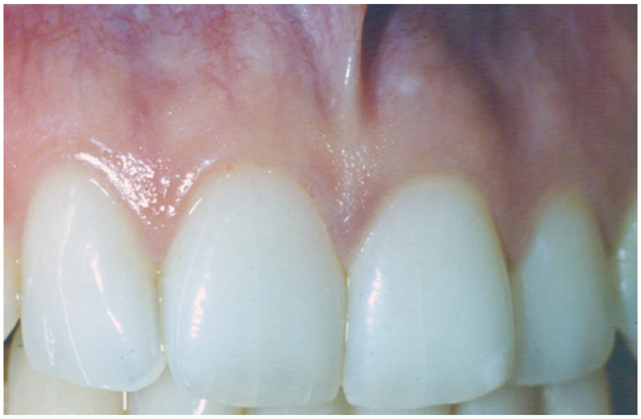

Figure 5-4 Position of the Margin in Health. In health, the gingival margin is either at the same level or ever so slightly coronal to the CEJ. In anterior sextants, the margin is characterized by pronounced scalloping of the margin and pointed interdental papillae.

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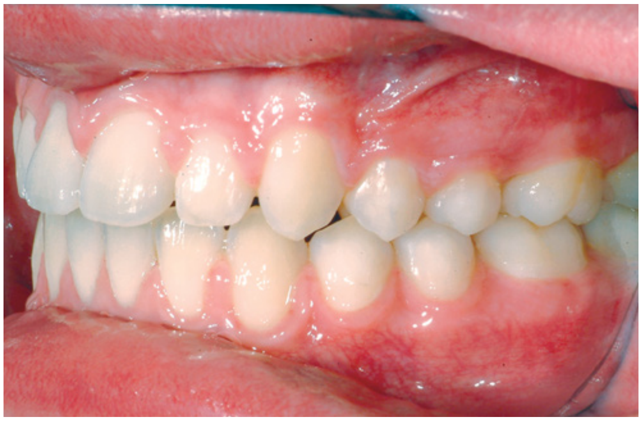

Figure 5-5 Gingiva in Posterior Sextants in Health. In posterior sextants, the healthy tissue is characterized by a gently scalloped margin that coincides with the CEJ level and gingival papillae that completely fill the embrasure spaces.

Used with permission from Langlais RP. *Color Atlas of Common Oral Diseases*. Wolters Kluwer; 2003. © Wolters Kluwer.

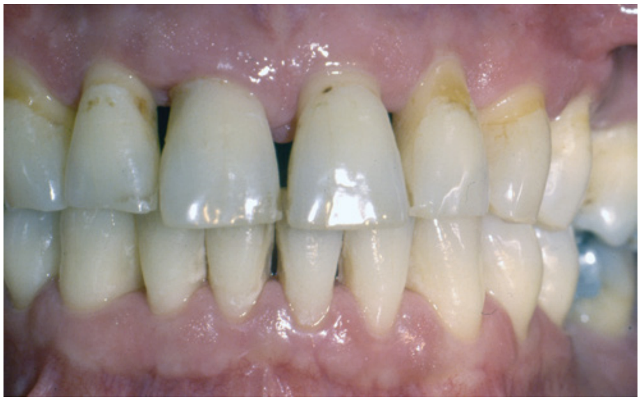

Figure 5-6 Health or Disease? This individual received periodontal treatment for periodontitis several years ago. The assessment at today's appointment reveals no inflammation and no additional attachment loss since beginning periodontal maintenance several years ago. Thus, despite the prior history of clinical attachment loss, the tissue at today's appointment is considered healthy. The attachment loss present is simply an indicator of previous disease activity.

Courtesy of Dr. Ralph Arnold, San Antonio, TX.

2017 AAP/EFP Subcategories of Periodontal Health/Gingival Health

1. **Normal (Intact) Versus Reduced Periodontium.** The 2017 AAP/EFP classification system recognizes that periodontal health can occur in a patient with either an intact periodontium or a reduced periodontium.
   a. An **intact periodontium**—a periodontium with no loss of periodontal tissue (no loss of connective tissue or alveolar bone).
   b. A **reduced periodontium**—a periodontium with preexisting loss of periodontal tissue but that is not currently undergoing loss of connective tissue/alveolar bone (periodontitis in remission).
2. **Three Subcategories of Periodontal Health.** Taking into consideration the possibility of an intact periodontium or a reduced periodontium, the current classification system subdivides periodontal health into three subcategories:
   a. **Periodontal health on an intact periodontium**—clinical signs characteristic of periodontal health coupled with an intact periodontium (**Box 5-1**, **Figure 5-7**).

Box 5-1 Periodontal Health on an Intact Periodontium

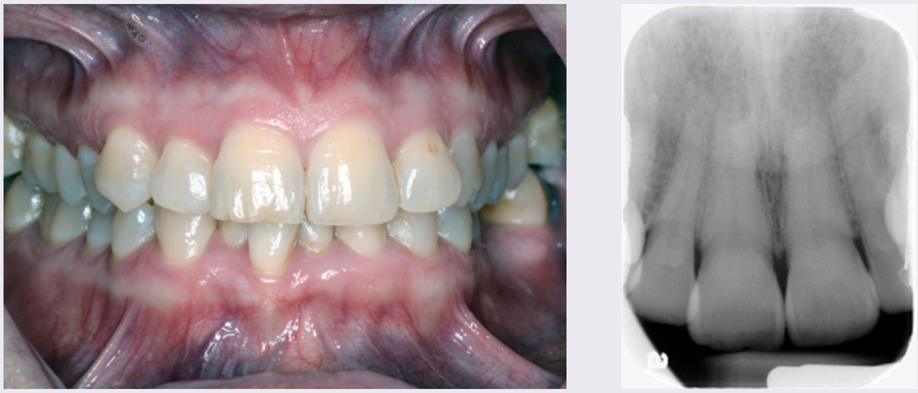

Figure 5-7 Periodontal Health on an Intact Periodontium. This patient exhibits no clinical signs of gingival inflammation and no previous loss of periodontal tissues. The accompanying radiograph show normal crestal bone levels and an absence of pathology.

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b. **Periodontal health on a reduced periodontium in a non-periodontitis patient**—clinical signs of periodontal health on a periodontium with a preexisting loss of connective tissue and/or loss of alveolar bone that is attributed to non-periodontitis reasons (**Box 5-2**, **Figure 5-8**). For example, recession of the gingival margin that occurs from a history of traumatic toothbrushing or the deliberate removal of alveolar bone that occurs during a surgical crown lengthening procedure.

Box 5-2 Periodontal Health on a Reduced Periodontium in a Non-Periodontitis Patient

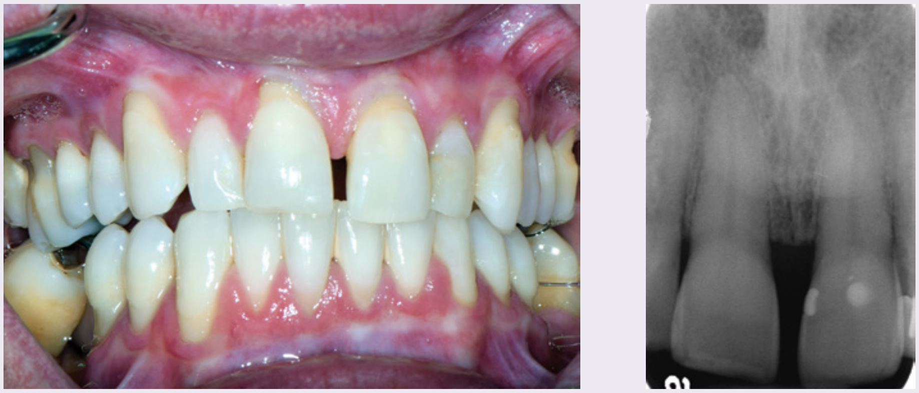

Figure 5-8 Periodontal Health on a Reduced Periodontium in a Non-Periodontitis Patient. This is a 54-year-old male patient with a history of excessive frequent daily toothbrushing (he brushed five to six times a day with a hard-bristled toothbrush).

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- Note the generalized recession of the gingival margin. However, there are no evident signs of clinical inflammation.
- The accompanying radiograph does not reveal any changes in either the alveolar bone height or the architecture and morphology of the alveolar bone.
- Treatment objectives for this case are to (1) address the patient's self-care habits and reinforce proper oral hygiene and (2) periodically monitor the patient's periodontal status to prevent further loss of tissue.

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c. **Periodontal health on a reduced periodontium in a successfully treated stable periodontitis patient**—clinical signs of periodontal health on a periodontium with a preexisting loss of connective tissue and alveolar bone that is attributed to periodontitis but that has been successfully treated and is currently stable (**Box 5-3**, **Figure 5-9**).

Box 5-3 Periodontal Health on a Reduced Periodontium in a Successfully Treated Periodontitis Patient

- The accompanying radiograph reveals loss of alveolar bone height that occurred due to previous history of active disease.
- Future treatment objectives should be targeted to (1) maintain the reduced periodontium in a healthy and stable state and (2) prevent the reactivation of periodontitis.

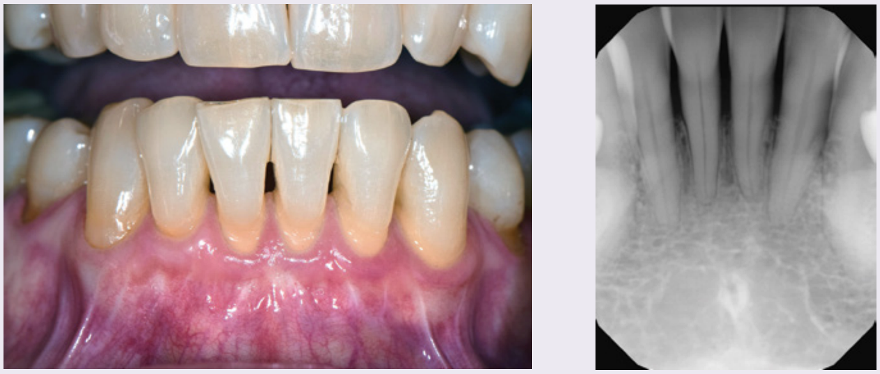

Figure 5-9 Periodontal Health on a Reduced Periodontium in a Successfully Treated Stable Periodontitis Patient. In 2016, this generalized periodontitis patient underwent nonsurgical and surgical periodontal therapy to control the disease. Eighteen months later, the above photo was taken to illustrate how healthy the gingiva appears following therapy and meticulous home care.

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Section 2

Dental Biofilm-Induced Gingivitis

Case Definitions and Diagnostic Considerations of Dental Biofilm-Induced Gingivitis

As illustrated in **Figure 5-10**, gingival diseases and conditions are broadly classified as (1) gingivitis that is dental biofilm-induced or (2) gingival diseases that are non-dental biofilm-induced. This section discusses the first and most common of these: **dental biofilm-induced gingivitis** (the reader should take note that this condition may be commonly referred to as dental plaque-induced gingivitis).[2]

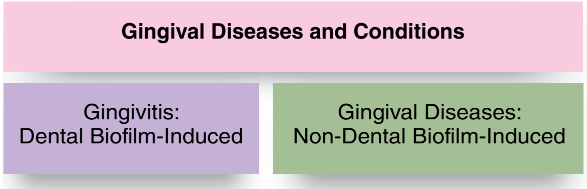
Gingival Diseases and Conditions
Gingivitis: Dental Biofilm-Induced
Gingival Diseases: Non-Dental Biofilm-Induced

Figure 5-10 Gingival Diseases and Conditions. Gingival diseases and conditions are classified as either dental biofilm-induced or non-dental biofilm-induced.

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Features of Dental Biofilm-Induced Gingivitis

1. **The Most Common Form of Periodontal Disease.** Dental biofilm-induced gingivitis is by far the most common type of periodontal disease. It does not directly cause tooth loss; however, managing gingivitis is the primary strategy for preventing periodontitis.[3] Epidemiologic data show that biofilm-induced gingivitis is prevalent at all ages in the population.[4,5]
2. Although gingivitis does not cause tooth loss, it must be managed as it is an active disease process which can potentially lead to periodontitis.[3]
    a. Dental biofilm-induced gingivitis is an inflammatory response of the gingival tissues resulting from plaque biofilm accumulation located at and below the gingival margin. Löe, Theilade, and Jensen first described gingivitis in their landmark research in 1965.[6]
    b. Gingival inflammation is the body's initial response to the invasion of the gingival tissues by periodontal pathogens. The host's ensuing response to the bacterial invasion manifests as changes to gingival color, contour, consistency, and surface texture.
    c. *The intensity of the clinical signs and symptoms of gingivitis may vary between individuals and within the dentition of an individual.*[7]
3. Epidemiologic data show that dental biofilm-induced gingivitis is prevalent in all age groups within the population.[4,5]
    a. Dental biofilm-induced gingivitis differs between children and adults (for more detailed information regarding the periodontium of a child patient, please refer to Chapter 35 which is an online chapter.).
    b. Inflammation is not as intense in children versus young adults with the same quantity of plaque biofilm.[8,9] Children may have fewer pathogenic bacteria in their plaque biofilm, a thicker junctional epithelium, and a less developed immune response.[10,11]
    c. Adolescents may have elevated levels of certain bacteria: *Actinomyces*, *Capnocytophaga*, *Leptotrichia*, and *Selenomonas* species.[12]
    d. Gingival inflammation in adults is more pronounced even when similar amounts of plaque biofilms are present, perhaps attributed to age-related differences in cellular inflammatory response to plaque biofilm.[13,14]
4. **Characteristics of Dental Biofilm-Induced Gingivitis**
    a. A clinician with a trained eye can discern subtle differences in color, contour, and consistency even in gingival tissues that appear relatively healthy at first glance. The phrase "tissue talks" is a good phrase to remember when assessing the gingival tissue. Indications that the tissue is not healthy include such clinical observations as red, swollen tissue or papillae that do not fill the interdental space. **Table 5-1** contrasts the characteristics of healthy versus inflamed gingival tissue.

Table 5-1 Characteristics of Healthy Versus Inflamed Gingival Tissue

	Healthy Tissue	Gingivitis
Color	Uniform pink color Pigmentation may be present	Acute: bright red Chronic: bluish red to purplish red
Contour	Marginal gingiva: meets the tooth in a tapered or slightly rounded edge Interdental papillae: pointed papilla fills the space between the teeth	Marginal gingiva: meets the tooth in a rolled, thickened edge Interdental papillae: bulbous, blunted, cratered
Consistency	Firm Resilient under compression	Spongy, flaccid (soft movable) Indents easily when pressed lightly Compressed air deflects the tissue
Texture	Smooth and/or stippled	Tissue appears "shiny" Stretched appearance
Margin	Slightly coronal to the CEJ	Coronal to the CEJ (due to swelling)
Bleeding	No bleeding upon probing	Bleeding upon probing

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b. **Tissue Color in Gingivitis**
   1. Gingivitis is an inflammation of the gingiva that causes the tissue to appear red (erythematous) and swollen (edematous) and bleed easily.[15] An increase in crevicular fluid also occurs in proportion to the severity of inflammation.[1]
   2. Inflammation results in increased blood flow to the gingiva, causing the tissue to appear bright red. **Figures 5-11** and **5-12** show examples of common clinical presentations of gingivitis.

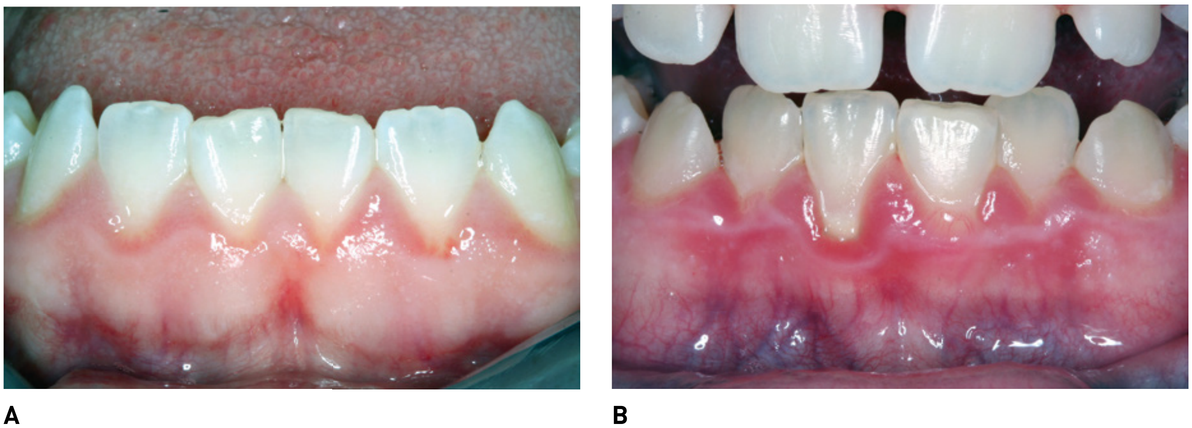

Figure 5-11 Color Changes in Gingivitis. A. Slight marginal redness is a clinical sign of early gingivitis. **B.** The gingival tissue in this image shows more inflammation than seen in photograph A. The marginal and papillary gingival tissues are bright red in color. Also note the swelling of the marginal gingiva and papillae in this example.

Courtesy of Dr. Richard J. Foster, Educational Solutions and Design, Greensboro, NC.

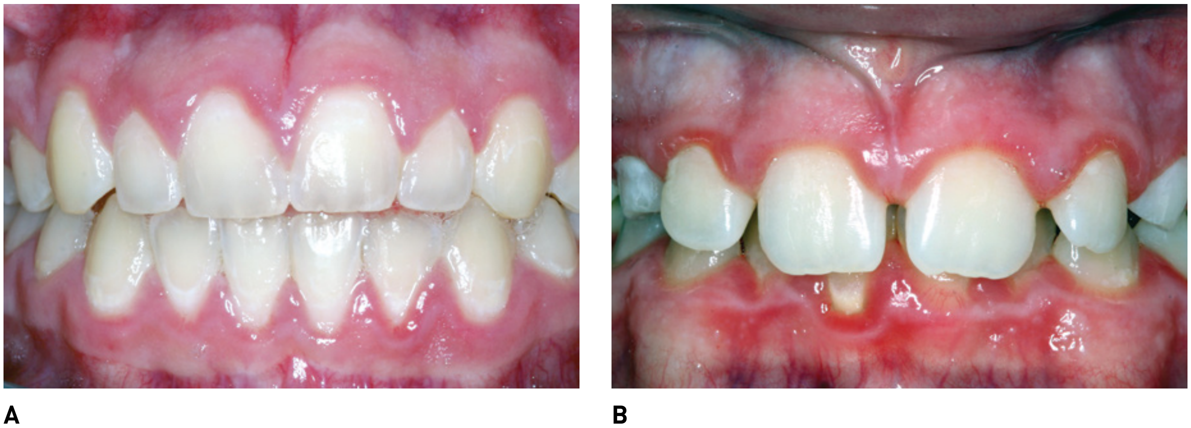

Figure 5-12 Color Changes in Gingivitis. A. This example shows subtle color changes in the marginal and papillary gingival tissues. **B.** In this example, the color changes are pronounced with fiery red marginal gingiva and papillae.

Courtesy of Dr. Richard J. Foster, Educational Solutions and Design, Greensboro, NC.

c. **Tissue Contour (Size and Shape) in Gingivitis**
   1. An increase of fluid within the tissue spaces—edema—causes enlargement of the gingival tissues. The normal scalloped appearance of the gingiva is lost if the gingival papillae and/or margins are swollen.
   2. Examples of types of changes in the appearance of the papillae are listed below.
      a. **Bulbous papilla**—a papilla that is enlarged and appears to bulge out of the interproximal space (**Figure 5-13**).
      b. **Blunted papilla**—a papilla that is flat and does not fill the interproximal space (**Figure 5-14**).
      c. **Cratered papilla**—a papilla that appears to have been "scooped out," leaving a concave depression in the midproximal area. Cratered papillae are associated with necrotizing gingivitis (**Figure 5-15**).
   3. As inflammation progresses, the gingival margin—the border where the gingiva contacts the cervical region of the anatomic crown—undergoes a transformation, changing from a knife-edge border to a bulbous and swollen border. Consequently, the gingival margin appears rolled and thickened (**Figure 5-16**).

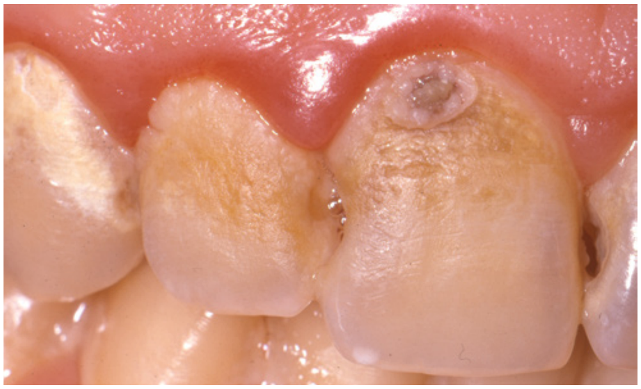

Figure 5-13 **Bulbous Papillae.** In gingivitis, the papillae may be enlarged and appear to bulge out of the interproximal space as seen in the papilla between the central and lateral incisors in this clinical photograph.

Courtesy of Dr. Ralph Arnold, San Antonio, TX.

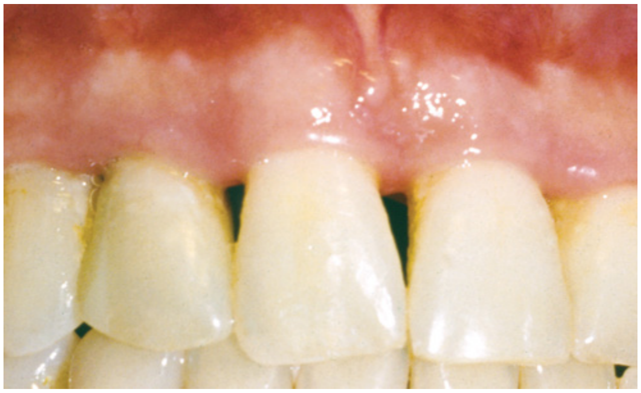

Figure 5-14 **Blunted Papillae.** In gingivitis, the papillae may be blunted and missing as seen in the papillae between the central and lateral incisors.

Courtesy of Dr. Don Rolfs, Periodontal Foundations, Wenatchee, WA.

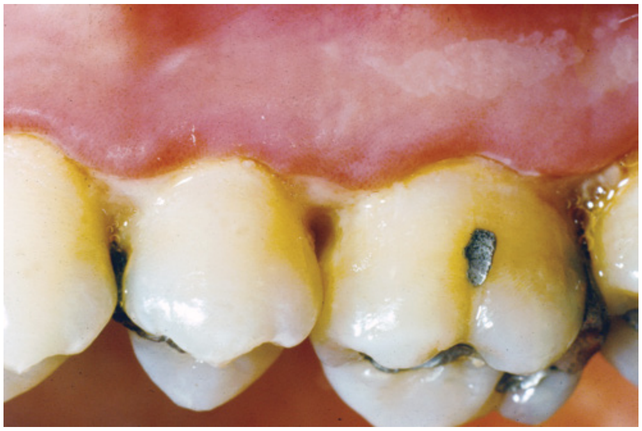

Figure 5-15 **Cratered Papillae.** The papillae may have a concave appearance in the midproximal area as seen in the papillae between the second premolar and molar in this clinical photo.

Courtesy of Dr. Don Rolfs, Periodontal Foundations, Wenatchee, WA.

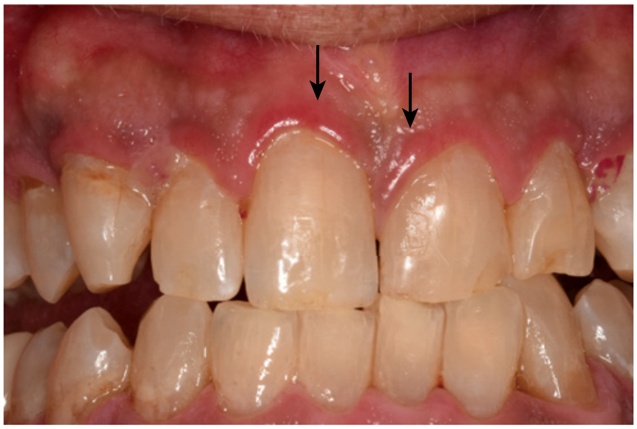

Figure 5-16 **Rolled, thickened gingival margins.** Note the enlarged and swollen gingival margins, especially around the maxillary central incisors (arrows).

d. **Tissue Consistency and Texture in Gingivitis**
  1. **Tissue Consistency in Gingivitis**
    a. Inflamed free gingiva loses its firm consistency, feels flaccid (soft, movable), and is easily retractable (**Figure 5-17**).
    b. When light pressure is applied to the inflamed gingiva with the side of a probe, the tissue feels spongy and can retain an imprint of the probe for several seconds.
    c. Inflamed gingival tissue loses its firm consistency becoming flaccid (soft, movable). When compressed air is directed into the sulcus, it readily deflects the gingival margin and papillae away from the neck of the tooth.
  2. **Surface Texture in Gingivitis**
    a. The increase in fluid due to the inflammatory response can cause the gingival tissues to appear smooth and very shiny (**Figure 5-18**).
    b. The tissue almost has a "stretched" appearance that resembles plastic wrap that has been pulled tightly.
    c. An individual with stippled gingiva prior to the onset of gingivitis may lose the gingival stippling following the onset of gingivitis.

e. **Position of Margin in Gingivitis**
  1. In gingivitis, the position of the gingival margin may appear more coronally (further above the CEJ) compared to its position in periodontal health.
  2. This change in the position of the gingival margin is due to tissue swelling and enlargement (**Figure 5-19**).

f. **Presence of Bleeding in Gingivitis**
   1. Bleeding upon gentle probing is seen clinically before changes in color are clinically detectible (**Figure 5-20**).
   2. In gingivitis, the epithelial lining of the sulcus becomes ulcerated, and the blood vessels become engorged. The tissues thus bleed easily from anything that involves soft tissue manipulation, such as probing and even toothbrushing.
   3. A direct relationship exists between inflammation and bleeding: the more severe the inflammation, the heavier the bleeding.

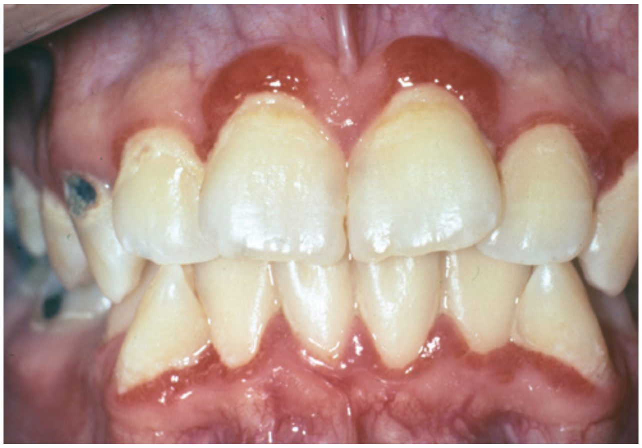

Figure 5-17 **Soft, Spongy Tissue.** Inflamed gingival tissue may be soft and spongy.

Courtesy of Dr. Ralph Arnold, San Antonio, TX.

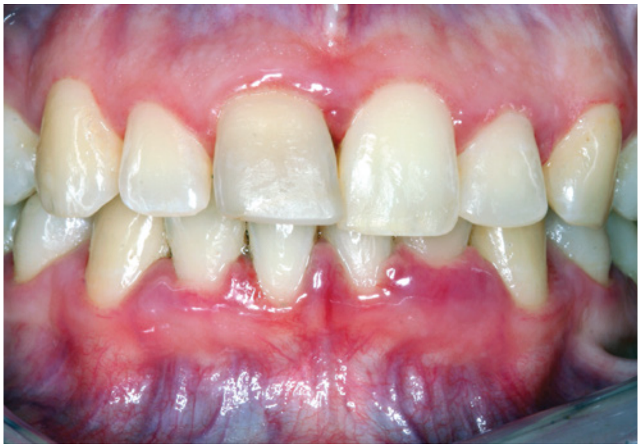

Figure 5-18 **Smooth, Shiny Tissue.** In gingivitis, fluid in the tissue can cause the tissue to appear smooth and shiny with a stretched appearance.

Courtesy of Dr. Richard J. Foster, Educational Solutions and Design, Greensboro, NC.

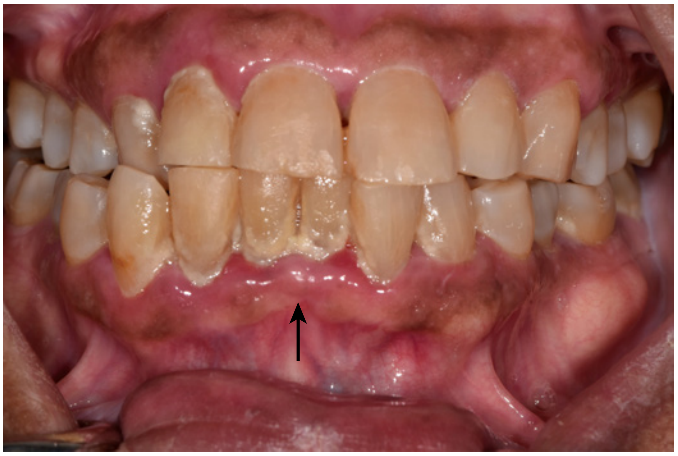

Figure 5-19 **Tissue Margin in Gingivitis.** The tissue swelling in gingivitis may cause the position of the gingival margin to move coronally—further above the CEJ—than in health. There is no destruction of periodontal ligament fibers or alveolar bone in gingivitis.

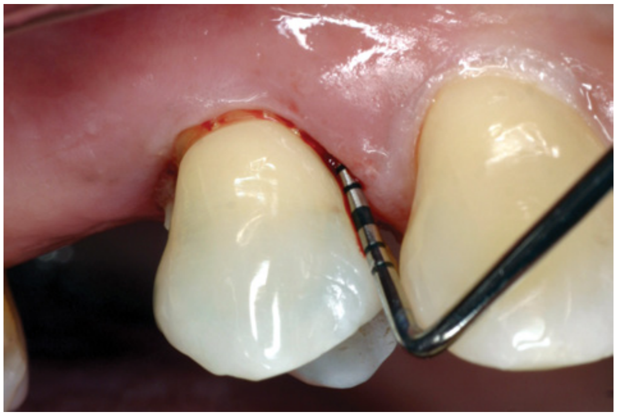

Figure 5-20 **Bleeding on Probing.** Bleeding is an important clinical indicator of inflammation. Inflammation results in ulceration of the sulcus/pocket wall, causing the gingival tissues to bleed easily during gentle probing. In spite of having a healthy color (coral pink), the gingival tissues easily bleed upon probing. This illustrates that evidence of bleeding often precedes any distinct change in gingival color.

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Extent and Distribution of Dental Biofilm-Induced Gingivitis

In documenting dental biofilm-induced gingivitis, it is important to note both the extent and distribution of the inflammation. Simply stating that gingivitis is present is too vague and does not indicate the level of concern. Identifying the severity of inflammation provides the information needed to accurately establish a diagnosis and appropriate treatment plan.

1. **Gingival Inflammation**
    a. **Extent of Inflammation.** The **extent of inflammation** is the degree to which the inflammation has spread throughout the mouth. The extent of inflammation is described as localized or generalized in the mouth.
    b. The primary parameter to determine the extent of inflammation is a percentage score based on the number of bleeding sites (that occur when probed by a standardized periodontal probe with a controlled force of 0.25N) divided by the total number of six sites on a tooth (mesio-buccal, mid-buccal, disto-buccal, mesio-lingual, mid-lingual, and disto-lingual) of all teeth present.
        1. If the bleeding upon probing affects less than 10% of sites, and there is an absence of bone loss or clinical attachment loss attributed to periodontitis, then the patient is given a diagnosis of gingival health.
        2. If the bleeding upon probing affects 10–30% of sites, and there is an absence of bone loss and/or clinical attachment loss attributed to periodontitis, then the extent of gingivitis is localized.
        3. If the bleeding upon probing affects more than 30% of sites, and there is an absence of bone loss and/or clinical attachment loss attributed to periodontitis, then the extent of gingivitis is generalized.
    c. **Distribution of Inflammation.** The **distribution of inflammation** describes how spread out the inflammation is within the gingival unit.
        1. The distribution of inflammation (a) may be limited to affect only the interdental papilla (known as **papillary gingivitis**), (b) may affect the gingival margin and the papilla (known as **marginal gingivitis**), or (c) may be more extensive to include the gingival margin, papilla, and the attached gingiva (known as **diffuse gingivitis**).
        2. **Table 5-2** summarizes the parameters that define the extent and distribution of inflammation of the gingival tissue. **Figures 5-21** to **5-25** illustrate the application of this descriptive terminology.

Table 5-2 Gingival Inflammation

Extent	■ Localized—bleeding upon probing affects 10–30% of sites, and there is an absence of bone loss and/or clinical attachment loss attributed to periodontitis. ■ Generalized—bleeding upon probing affects more than 30% of sites, and there is an absence of bone loss and/or clinical attachment loss attributed to periodontitis.
Distribution	■ Papillary—inflammation of the interdental papilla only. ■ Marginal—inflammation of the gingival margin and papilla. ■ Diffuse—inflammation of the gingival margin, papilla, and attached gingiva.
Descriptions	Descriptive terms may be combined to create a verbal picture of the inflammation, such as: ■ "Localized marginal inflammation in the mandibular anterior sextant" ■ "Localized papillary inflammation on the maxillary right canine" ■ "Localized diffuse inflammation between mandibular central incisors" ■ "Generalized marginal inflammation" ■ "Generalized diffuse inflammation"

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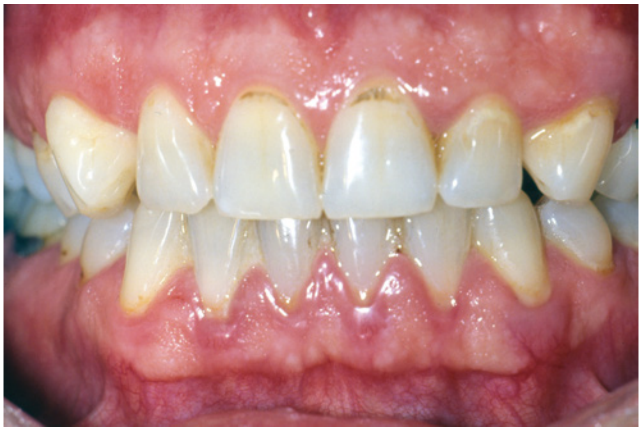

Figure 5-21 Localized Marginal Inflammation. Note the erythema and edema of the marginal and papillary gingival tissues localized to the mandibular anterior sextant.

Courtesy of Dr. Ralph Arnold, San Antonio, TX.

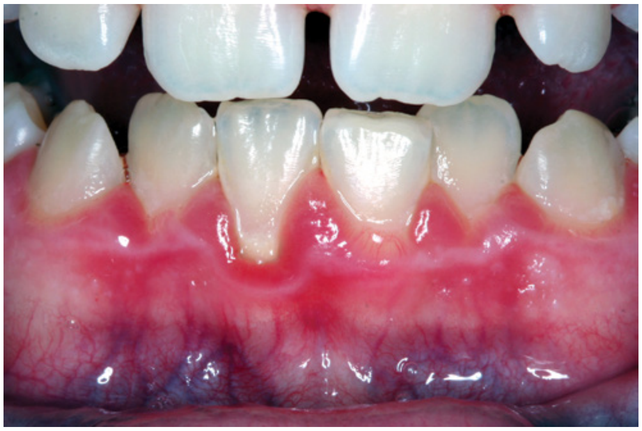

Figure 5-22 Localized Diffuse Inflammation. Erythema and edema of the gingival margin, papillae, and attached gingiva in the mandibular anterior sextant.

Courtesy of Dr. Richard J. Foster, Educational Solutions and Design, Greensboro, NC.

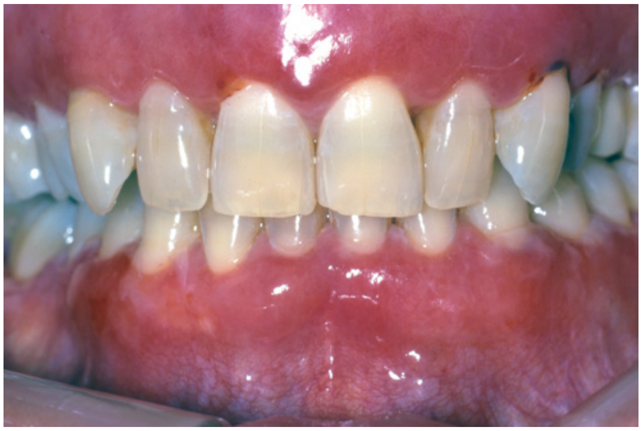

Figure 5-23 Generalized Diffuse Inflammation. Diffuse inflammation of the gingival margin, papillae, and attached gingiva throughout the entire mouth.

Courtesy of Dr. Ralph Arnold, San Antonio, TX.

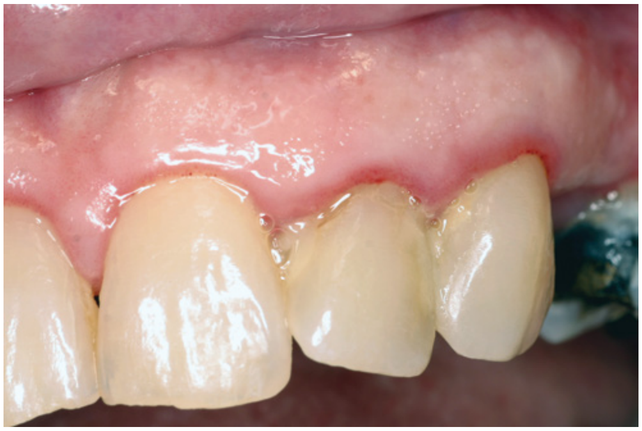

Figure 5-24 Localized Marginal Inflammation. Note the erythema along the gingival margin, extending down into the papillae on these maxillary anterior teeth.

Courtesy of Dr. Richard J. Foster, Educational Solutions and Design, Greensboro, NC.

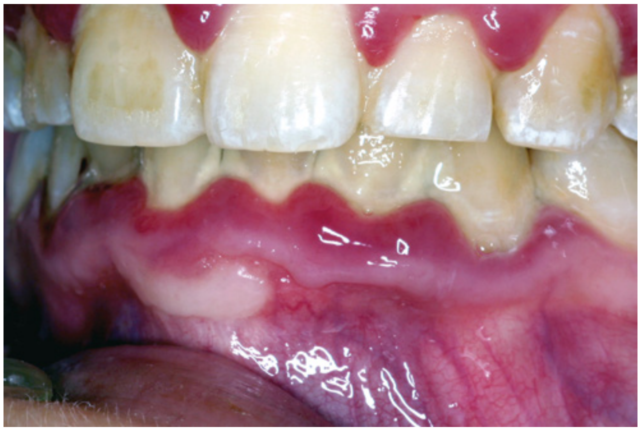

Figure 5-25 Localized Diffuse Inflammation. Inflammation involving the gingival margin, papillae, and attached gingiva of the mandibular anterior sextant.

Courtesy of Dr. Richard J. Foster, Educational Solutions and Design, Greensboro, NC.

2017 AAP/EFP Subcategories of Dental Biofilm-Induced Gingivitis

The 2017 classification system recognizes that dental biofilm-induced gingivitis can occur either on an intact periodontium or on a reduced periodontium and can occur in a non-periodontitis patient or a successfully treated stable periodontitis patient. **Table 5-3** summarizes the distinguishing clinical features of each category.

Table 5-3 Key Features of Periodontal Health and Dental Biofilm-Induced Gingivitis

Intact Periodontium	**Health**	**Gingivitis**
Clinical attachment loss	No	No
Radiological bone loss	No	No
Reduced Periodontium, Non-Periodontitis Patient	**Health**	**Gingivitis**
Clinical attachment loss	Yes	Yes
Radiological bone loss	Possible[a]	Possible[a]
Successfully Treated Stable Periodontitis Patient	**Health**	**Gingivitis**
Clinical attachment loss	Yes	Yes
Radiological bone loss	Yes	Yes

[a] Clinical attachment loss would be present on a reduced periodontium; however, bone loss may not be detectible on a radiograph.

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1. Subcategories of Dental Biofilm-Induced Gingivitis
   a. **Dental biofilm-induced gingivitis on an intact periodontium** (**Box 5-4**, **Figure 5-26**)—the presence of dental biofilm-induced inflammation on an intact periodontium.
   b. **Dental biofilm-induced gingivitis on a reduced periodontium in a non-periodontitis patient** (**Box 5-5**, **Figure 5-27**)—the presence of dental biofilm-induced inflammation on a periodontium with pre-existing loss of connective tissue and/or loss of alveolar bone that can be attributed to non-periodontitis reasons.
   c. **Gingival inflammation on a reduced periodontium in a successfully treated stable periodontitis patient** (**Box 5-6**, **Figure 5-28**).

Box 5-4 Dental Biofilm-Induced Gingivitis on an Intact Periodontium

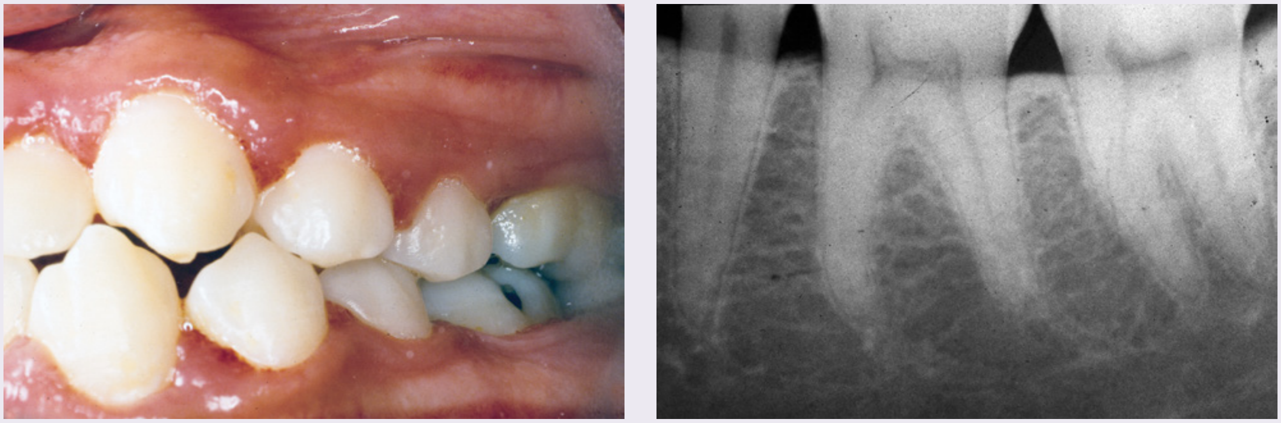

Figure 5-26 **Dental Biofilm-Induced Gingivitis.** Clinical and radiographic images of patient who presents with dental biofilm-induced gingivitis on an intact periodontium. Note the rolled gingival papilla and enlarged papilla. The accompanying radiograph does not reveal any changes in either the alveolar bone height or the architecture and morphology of the alveolar bone.

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Box 5-5 Dental Biofilm-Induced Gingivitis on a Reduced Periodontium in a Non-Periodontitis Patient

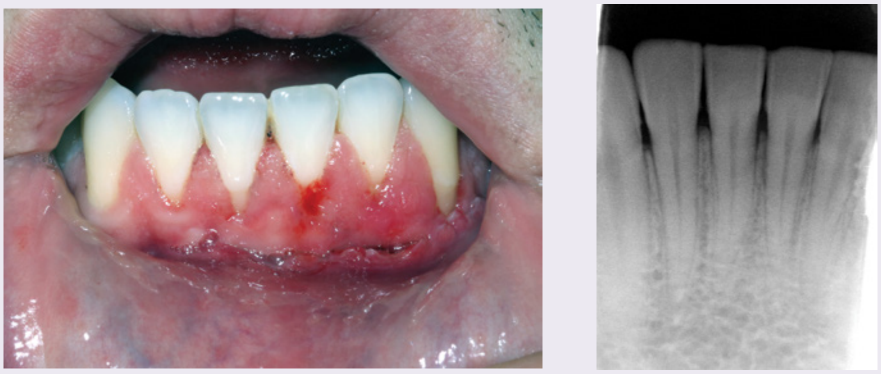

Figure 5-27 Dental Biofilm-Induced Gingivitis on a Reduced Periodontium in a Non-Periodontitis Patient. On the clinical photograph, observe all the visual signs of gingival inflammation: bleeding, swelling, and redness. This is a 17-year-old male patient who just completed orthodontic therapy to straighten teeth that were previously malaligned (crowded). The accompanying radiograph does not reveal any changes in either the alveolar bone height or the architecture and morphology of the alveolar bone.

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- Orthodontists use different strategies to move teeth to an ideal alignment and position. However, one undesired consequence is that changes in the periodontal tissues may occur as a result of orthodontically induced movement of the teeth. This is apparent in cases where orthodontic therapy displaces the teeth in a buccal direction, causing resorption of the overlying thin, delicate outer cortical plate. In the case above, post-orthodontic movement of the lower right central incisor and both canines have led to recession of the gingival margin (permanent loss of the marginal gingiva).
- Because the recession of the gingival margin is not due to periodontitis, the treatment objectives are to (1) remove the etiologic factors responsible for gingival inflammation, (2) reinforce oral hygiene, (3) avoid further loss of periodontal tissues (i.e., recession of the gingival margin), and (4) minimize the risk of the gingivitis converting into periodontitis.

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Box 5-6 Dental Biofilm-Induced Gingivitis on a Reduced Periodontium in a Successfully Treated Periodontitis Case

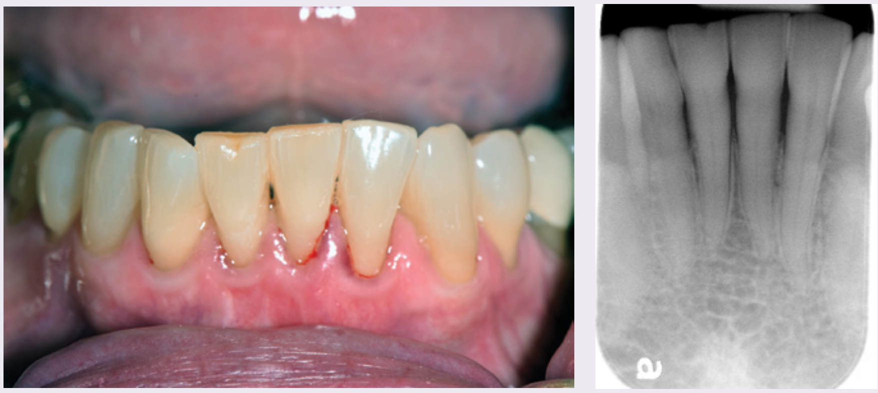

Figure 5-28 Dental Biofilm-Induced Gingivitis on a Reduced Periodontium in a Successfully Treated Stable Periodontitis Patient. This is a 68-year-old female patient who was successfully treated in the past for periodontitis. The dental radiograph reveals loss of alveolar bone height.

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(*continues*)

Box 5-6 Dental Biofilm-Induced Gingivitis on a Reduced Periodontium in a Successfully Treated Periodontitis Case *(continued)*

- By performing a comprehensive periodontal examination and comparing the present information with previous periodontal findings (such as past probing depths, clinical attachment loss [CAL], bleeding upon probing scores, etc.), it is apparent that the periodontitis is not ongoing, but rather arrested (no additional loss of alveolar bone or connective tissue).
- However, note the visual signs of gingival inflammation such as thickened marginal gingiva and slight gingival bleeding from the lower left incisors.
- In such a case, the treatment objectives are to (1) maintain the reduced periodontium in a healthy and stable state by removing the etiologic agents and (2) reinforce patient self-care and professional care to prevent the reactivation of periodontitis.

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2. **Dental Biofilm-Induced Gingivitis on a Reduced Periodontium After Successful Treatment**
   a. In someone successfully treated for periodontitis, if the extent of attachment loss present remains stable over months or years, then the presence of attachment loss is *not* an indication of active periodontitis.
   b. Dental biofilm-induced gingivitis on a reduced periodontium in a successfully treated periodontitis case is characterized by the return of bacteria-induced inflammation to the gingival margin on a reduced periodontium with *no evidence of progressive attachment loss.*[2] If a clinician is going to make the diagnosis of "gingivitis on a reduced but stable periodontium," it is necessary to demonstrate that attachment loss (loss of connective tissue and alveolar bone) is not ongoing.
   c. The common clinical findings on a reduced periodontium are the same as dental biofilm-induced gingivitis except for the presence of preexisting attachment loss, and therefore a higher risk of periodontitis relapse. Due to this higher risk for recurrence of periodontitis, individualized professional care is of utmost importance for the patient with gingival inflammation on a reduced periodontium.[16]
   d. Although a successfully treated periodontitis patient exhibits an absence of ongoing, progressive periodontitis, the patient cannot revert to being classified as a "dental biofilm-induced gingivitis on an intact periodontium."
      1. Periodontitis is an irreversible condition that results in a *permanent* loss of attachment and alveolar bone.
      2. A periodontitis patient remains a periodontitis patient for life—even following successful periodontal therapy—and requires lifelong supportive care to prevent recurrence of disease.[1]

Modifying Factors of Dental-Biofilm-Induced Gingivitis

Although the primary etiology of dental biofilm-induced gingivitis is plaque biofilm, several modifying factors may play a role in exacerbating the host inflammatory response to the plaque biofilm. These modifying factors can be subdivided into (1) systemic factors that alter the host response to plaque biofilm or (2) oral factors that contribute to increased plaque retention. **Table 5-4** outlines the various conditions that modify biofilm-induced gingivitis but are not considered diagnoses in and of themselves.

1. **Systemic Conditions as Modifying Factors**
   a. **Sex and Steroid Hormones.** The maintenance of a healthy balance within the periodontium involves a complex relationship with the endocrine system. Evidence suggests that tissue responses in the periodontium are influenced by the levels of sex hormones present at one time or another throughout a person's life cycle.[17,18] *The following conditions may modify biofilm-induced gingivitis but are not considered diagnoses in and of themselves*:
      1. **Puberty**
         a. The dramatic rise in steroid hormone levels during puberty has a temporary effect on the inflammatory status of the gingiva.[17,18]
         b. Studies demonstrate an increase in gingival inflammation around the time of puberty in both genders.[19,20]

Table 5-4 Classification of Dental Biofilm-Induced Gingivitis and Modifying Factors

A. Associated with bacterial dental biofilm only
B. Potential modifying factors of biofilm-induced gingivitis
    1. Systemic conditions
        a. Sex and steroid hormones
            1. Puberty
            2. Menstrual cycle
            3. Pregnancy
            4. Oral contraceptives
        b. Hyperglycemia
        c. Leukemia
        d. Smoking
        e. Malnutrition
    2. Oral factors enhancing plaque biofilm accumulation
        a. Prominent subgingival restoration margins
        b. Hyposalivation (reduced saliva production)
C. Drug-influenced gingival enlargements

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c. Puberty-associated gingivitis has many of the clinical features of biofilm-induced gingivitis; however, *it is distinguished by clinical signs of gingival inflammation in the presence of relatively small amounts of plaque biofilm*.[2] Thus, puberty-associated gingivitis is characterized by an exaggerated inflammatory response of the gingiva to a relatively small amount of plaque biofilm around the time of puberty. **Figure 5-29** shows one example of puberty-associated gingivitis.

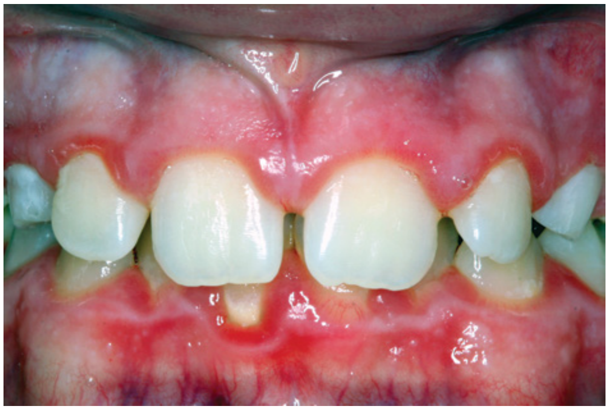

Figure 5-29 Gingivitis Associated with Puberty. Gingivitis associated with puberty is an exaggerated inflammatory response of the gingiva to a relatively small amount of plaque biofilm. The exaggerated response is modulated by hormones released during puberty.

Courtesy of Dr. Richard J. Foster, Educational Solutions and Design, Greensboro, NC.

2. **Menstrual Cycle**
    a. Most clinical studies show there to be only modest observable inflammatory changes in the gingiva during ovulation.[19,20]
    b. Although there may be a few women who are extremely sensitive to hormonal changes in the gingiva during the menstrual cycle, in most women there will be no clinically evident inflammatory changes to the gingiva.[21–23]

3. **Pregnancy**
    a. During pregnancy, the levels of estrogen and progesterone continue to rise and reach their peak in the eighth month of gestation. High levels in both blood and saliva cause an exaggerated tissue response to plaque biofilm. Increased quantities of hormones also trigger gingival crevicular fluid flow, which may precipitate gingival inflammation.
    b. Gingival inflammation is significantly higher in the pregnant patient in response to even relatively small amounts of plaque biofilm and bleeding on probing or with toothbrushing is also increased.[6,24–27] **Figure 5-30** shows a case of gingivitis associated with pregnancy.
    c. Gingivitis associated with pregnancy can spontaneously resolve postpartum.
    d. In some cases, a gingival papilla can react so strongly to plaque biofilm that a large, localized overgrowth of gingival tissue called a **pregnancy-associated pyogenic granuloma** (pregnancy tumor) may form on the interdental gingiva or on the gingival margin.[28]
        1. This condition is the result of an exaggerated tissue response to plaque biofilm or other irritants that usually occurs after the first trimester of pregnancy.
        2. The gingival mass is characterized by a mushroom-like tissue mass that most commonly occurs in the maxilla and interproximally (**Figure 5-31**).

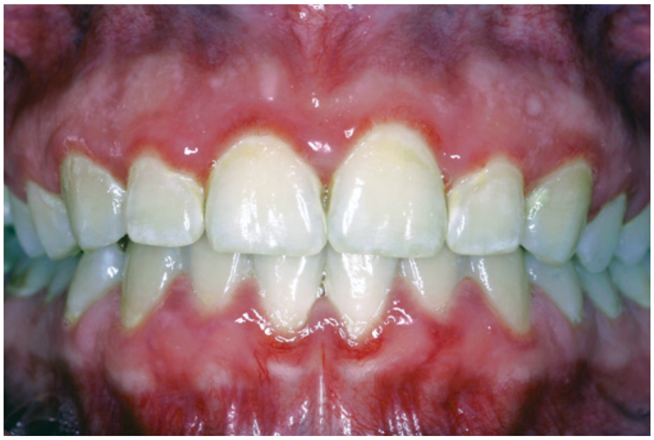

Figure 5-30 Gingivitis Associated with Pregnancy. Note the red gingiva and bulbous interdental papilla on this patient with gingivitis that is associated with pregnancy.

Courtesy of Dr. Richard J. Foster, Educational Solutions and Design, Greensboro, NC.

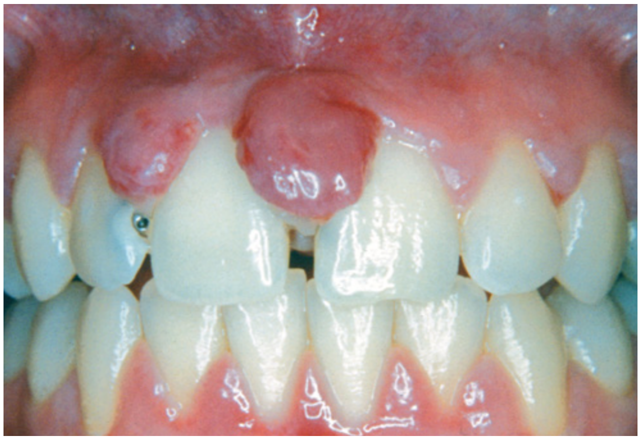

Figure 5-31 Pregnancy-Associated Pyogenic Granuloma. This mushroom-like mass of the gingiva bleeds easily if disturbed.

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        3. A pregnancy-associated pyogenic granuloma is painless and noncancerous. However, if it grows to interfere with occlusion, painful ulceration of the pyogenic granuloma may occur.
        4. The tissue mass bleeds easily if disturbed and may appear to be covered with dark red pinpoint markings.
        5. The growth usually resolves after childbirth. Even though the growth spontaneously resolves after childbirth, the granuloma can be eliminated during the pregnancy stage by removing the plaque biofilm responsible for the enlargement.
4. **Oral Contraceptives**
    a. Early conceptive agents had high hormone concentrations that were associated with gingival inflammation.
    b. Current oral contraceptive concentrations are much lower than the original doses and do not induce clinical changes in the gingiva.[17,18,29]

b. **Hyperglycemia. Hyperglycemia** is the presence of an abnormally high concentration of glucose in the circulating blood that occurs especially in individuals with diabetes mellitus.
    1. Gingivitis is often seen in children with poorly controlled diabetes mellitus.
    2. The level of glycemic control may be more important in determining the severity of the gingival inflammation than the amount of plaque biofilm present.[30,31] In other words, the inflammatory response of the gingiva to plaque biofilm is exacerbated by the high blood glucose levels.

c. **Leukemia. Leukemia** is cancer of the body's blood-forming tissues, including the bone marrow and the lymphatic system. Leukemia usually involves the white blood cells. In people with leukemia, the bone marrow produces abnormal white blood cells, which do not function properly. Many types of leukemia exist. Some forms of leukemia are more common in children. Other forms of leukemia occur mostly in adults.
   1. Gingivitis associated with leukemia is an exaggerated inflammatory response of the gingiva to plaque biofilm resulting in increased bleeding and tissue enlargement. Oral lesions may be the first clinical signs of leukemia; therefore, dental health care providers can be the first to suspect that a patient may have leukemia.
   2. Gingival tissues appear swollen, spongy, shiny, and red to deep purple in appearance.[32,33] **Figure 5-32** shows an example of gingivitis associated with leukemia. Typically, leukemia-associated gingivitis begins in the papillae and spreads to the marginal and then the attached gingiva.
   3. Tissues are very friable (tear easily) and tend to bleed with slight provocation.
   4. Although plaque biofilm can exacerbate the gingival response to leukemia, the presence of biofilm is *not* a prerequisite for gingivitis in patients with leukemia.[32]

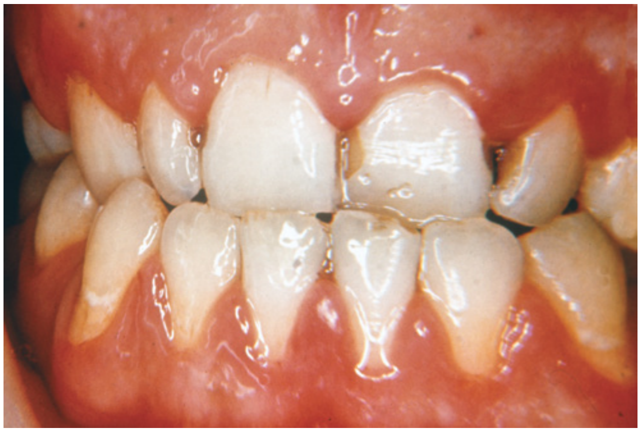

Figure 5-32 Gingivitis in an Adult with Leukemia. Note the red, swollen appearance of the gingiva in this patient with leukemia.

Courtesy of Dr. Ralph Arnold, San Antonio, TX.

d. **Smoking**
   1. Epidemiologic studies indicate that smoking is one of the major lifestyle risk factors for periodontal disease.[34]
   2. Plaque biofilm accumulation and disease progression are exacerbated in smokers. Gingival fibrosis—the formation of an abnormal amount of fibrous tissue—is often observed in smokers.[35]
   3. *Smokers have fewer clinical signs and symptoms of gingival inflammation than nonsmokers.*[35–38] Therefore, clinicians should be aware that smoking can mask gingivitis.
      a. Smoking as a risk factor is more fully discussed in Chapter 17, Tobacco, Smoking, and Periodontal Disease.

e. **Malnutrition**
   1. Even with our adequate food supply in North America, infants, institutionalized individuals, and alcoholics are all at risk for vitamin deficiencies. However, the precise relationship between nutrition and periodontal disease is not fully understood.
   2. The one nutritional deficiency that has well-documented effects on the periodontium involves the depletion of plasma ascorbic acid (vitamin C).
   3. Vitamin C—a substance found in many fruits and vegetables—is essential for the formation of collagen and fibrous tissue for normal intercellular matrices in teeth, bone, cartilage, connective tissue, and skin, and for the structural integrity of capillary walls.
      a. A lack of vitamin C can lead to scurvy, or less severe conditions, such as delayed healing of wounds.
      b. Gingivitis associated with declining ascorbic acid levels may be difficult to detect clinically, and when it is detected, it usually has characteristics similar to biofilm-induced gingivitis. **Figure 5-33** shows an example of gingivitis in a patient with scurvy.

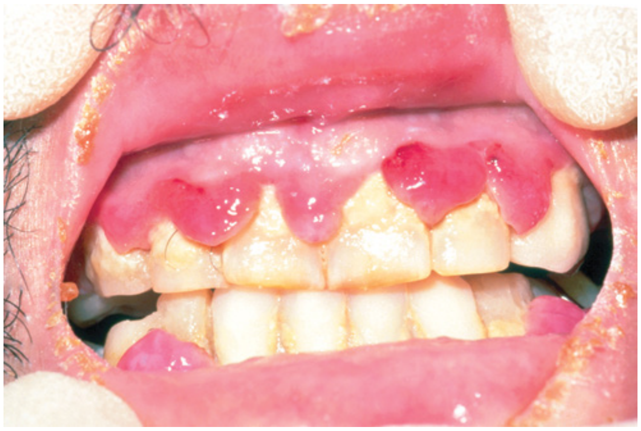

Figure 5-33 Ascorbic Acid-Deficiency Gingivitis. A photograph of a patient with scurvy. Scurvy arises from a dietary deficiency in vitamin C (ascorbic acid). Note the bright red, swollen, and ulcerated gingival tissue.

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2. **Oral Factors That Enhance Plaque Biofilm Accumulation**
   a. **Prominent Subgingival Restoration Margins**
      1. A 26-year longitudinal study confirms that dental restorations margins placed apical to the gingival margin are detrimental to gingival health.[39]
      2. The margins of prominent subgingival restorations promote gingivitis by increasing the local accumulation of bacterial plaque biofilms. Therefore, subgingival restoration margins need to be carefully designed to minimize biofilm retention.

   b. **Hyposalivation**
      1. Hyposalivation—a decreased flow of saliva—may be caused by some health conditions/diseases such as Sjögren syndrome, anxiety, and poorly controlled diabetes. In addition, it is frequently observed as a side effect of medications, such as antihistamines, decongestants, antidepressants, and antihypertensive medications.
      2. Hyposalivation may cause progressive dental caries, taste disorders, halitosis, and inflammation of the oral mucosa, tongue, and gingiva.[40,41] Dryness of the mouth may make patient self-care more difficult and worsen gingival inflammation.

3. **Drug-Influenced Gingival Enlargements**
   a. **Drug-influenced gingival enlargements** are an increase in size of the gingiva associated with certain systemic medications, most commonly anticonvulsants, calcium channel blockers, and immunosuppressants.

   b. **Medications Associated with Gingival Enlargement**
      1. **Anticonvulsants** (e.g., phenytoin, methsuximide, divalproex sodium). Anticonvulsants are used to manage epileptic seizures. In addition, some anticonvulsants are now used in the treatment of bipolar disorder.
      2. **Immunosuppressants** (e.g., cyclosporine). Immunosuppressant drugs suppress the natural immune responses. Immunosuppressants are given to transplant patients to prevent organ rejection or to patients with autoimmune diseases. The immunosuppressant stimulates fibroblast proliferation with excessive extracellular matrix accumulation in gingival tissues.[19]
      3. **Calcium Channel Blocking Agents** (e.g., amlodipine, nifedipine, verapamil). Calcium channel blocking agents relax the blood vessels and increase the supply of blood and oxygen to the heart while reducing its workload. Some of the calcium channel blocking agents are used to relieve and control angina pectoris (chest pain). Some are also used to treat high blood pressure (hypertension). These drugs affect gingival connective tissues by stimulating an increase of fibroblasts which increases the production of connective tissue matrix.

c. **Etiology**
   1. For drug-influenced gingival conditions, plaque biofilms in conjunction with the drug are necessary to produce gingival enlargements.
   2. However, not all individuals who take these medications will develop enlargements of the gingival tissues.

d. **Common Characteristics of Drug-Influenced Gingival Enlargements**
   1. The onset of tissue enlargement usually occurs within 3 months of medication use. The severity of overgrowth is directly affected by level of patient self-care; scrupulous patient self-care can reduce the severity of the overgrowth but may not eliminate it.
   2. There is a higher prevalence of drug-influenced gingival enlargements in younger age groups.

e. **Clinical Appearance**
   1. Gingival tissues in anterior sextants are most commonly affected; however, tissue enlargements can occur in posterior sextants (**Figures 5-34** and **5-35**).
   2. The pattern of tissue enlargement is irregular, usually first observed in the papillae, beginning as a painless area of enlargement on the papilla and then proceeding to the marginal gingiva.
   3. Gingival enlargement is characterized by an increased flow of crevicular fluid from the sulcus and bleeding upon probing with no attachment loss.

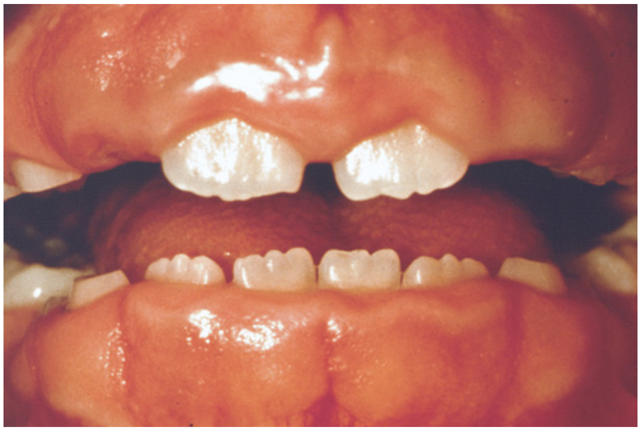

Figure 5-34 Phenytoin-Induced Gingival Enlargement. Massive-tissue overgrowth may be seen in phenytoin-induced gingival enlargement.

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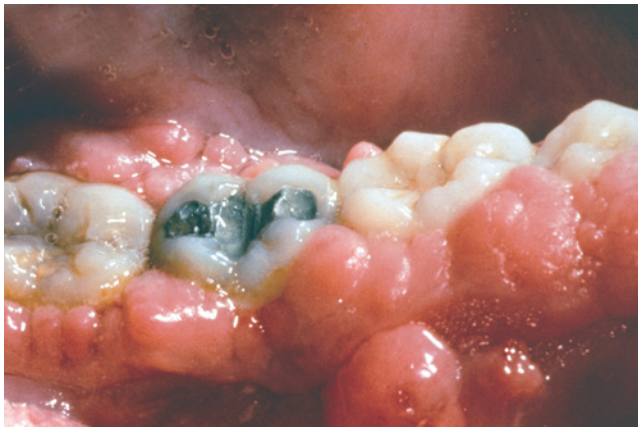

Figure 5-35 Cyclosporine-Induced Gingival Enlargement. Gingival changes seen in cyclosporine-induced gingival enlargement.

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Section 3

Non–Dental Biofilm-Induced Gingival Diseases and Conditions

Less common, but equally significant, is another form of gingival diseases known as **non–dental biofilm-induced gingival diseases and conditions** (also commonly referred to as non-dental plaque-induced gingival diseases and conditions). *In contrast to dental biofilm-induced gingivitis, plaque biofilm is not the primary etiologic factor for non–dental biofilm-induced gingival diseases and conditions.* Nevertheless, although dental biofilm is not necessary to trigger non–dental biofilm-induced gingival diseases and conditions, its presence can certainly exacerbate the severity of inflammation. Additionally, unlike dental biofilm-induced gingivitis, non–dental biofilm-induced gingival diseases and conditions can be painful and can be of major significance for patients. This section presents some examples of the small percentage of gingival disease in which plaque biofilm does not play a primary etiologic role. The 2017 classification scheme for non–dental biofilm-induced gingival diseases and conditions is outlined in **Table 5-5**.

Table 5-5 Classification of Non–Dental Biofilm-Induced Gingival Diseases and Conditions

1. **Genetic/developmental disorders**
    - 1.1 Hereditary gingival fibromatosis (HGF)
2. **Specific infections**
    - 2.1 Bacterial origin
        - Necrotizing periodontal diseases (*Treponema* spp., *Selenomonas* spp., *Fusobacterium* spp., *Prevotella intermedia*, and others)
        - *Neisseria gonorrhoeae* (gonorrhea)
        - *Treponema pallidum* (syphilis)
        - *Mycobacterium tuberculosis* (tuberculosis)
        - Streptococcal gingivitis (strains of streptococcus)
    - 2.2 Viral origin
        - Coxsackie virus (hand-foot-and-mouth disease)
        - Herpes simplex types 1 and 2 (primary or recurrent)
        - Varicella-zoster virus (chicken pox or shingles affecting cranial nerve V)
        - Molluscum contagiosum virus
        - Human papilloma virus (squamous cell papilloma, condyloma acuminatum, verruca vulgaris, and focal epithelial hyperplasia)
    - 2.3 Fungal
        - Candidosis
        - Other mycoses (e.g., histoplasmosis, aspergillosis)
3. **Inflammatory and immune conditions and lesions**
    - 3.1 Hypersensitivity reactions
        - Contact allergy
        - Plasma cell gingivitis
        - Erythema multiforme
    - 3.2 Autoimmune diseases of skin and mucous membranes
        - Pemphigus vulgaris
        - Pemphigoid
        - Lichen planus
        - Lupus erythematosus
    - 3.3 Granulomatous inflammatory conditions (orofacial granulomatosis)
        - Crohn's disease
        - Sarcoidosis
4. **Reactive processes**
    - 4.1 Epulides
        - Fibrous epulis
        - Calcifying fibroblastic granuloma
        - Pyogenic granuloma (vascular epulis)
        - Peripheral giant cell granuloma (or central)
5. **Neoplasms**
    - 5.1 Premalignant
        - Leukoplakia
        - Erythroplakia
    - 5.2 Malignant
        - Squamous cell carcinoma
        - Leukemia
        - Lymphoma
6. **Endocrine, nutritional, and metabolic diseases**
    - 6.1 Vitamin deficiencies
        - Vitamin C deficiency (scurvy)
7. **Traumatic lesions**
    - 7.1 Physical/mechanical insults
        - Frictional keratosis
        - Toothbrushing-induced gingival ulceration
        - Factitious injury (self-harm)

  7.2 Chemical (toxic) insults
    - Etching
    - Chlorhexidine
    - Acetylsalicylic acid
    - Cocaine
    - Hydrogen peroxide
    - Dentifrice detergents
    - Paraformaldehyde or calcium hydroxide

  7.3 Thermal insults
    - Burns of mucosa

8. **Gingival pigmentation**
   - Gingival pigmentation/melanoplakia
   - Smoker's melanosis
   - Drug-induced pigmentation (antimalarials; minocycline)
   - Amalgam tattoo

Description of Selected Disease Disorders

1. **Genetic/Developmental Abnormalities**
   a. **Hereditary Gingival Fibromatosis.** Hereditary gingival fibromatosis is a rare benign oral condition characterized by slow and progressive enlargement of both maxillary and mandibular attached gingiva. An example of a patient with hereditary gingival fibromatosis is provided in **Figure 5-36**.[42] It may develop as an isolated disorder but can feature along with a syndrome. Compared to drug-related gingival overgrowth, hereditary gingival fibromatosis is a rare condition.

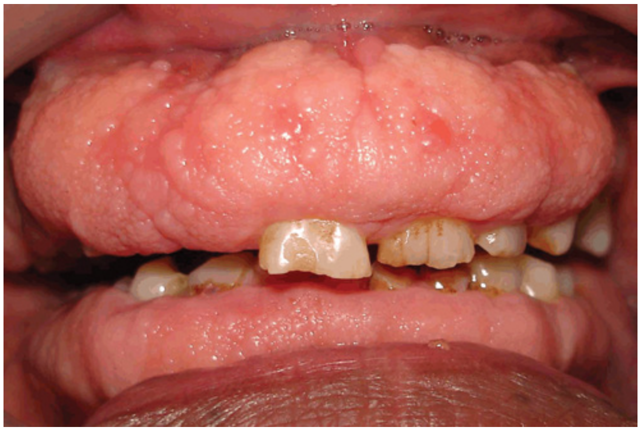

Figure 5-36 Hereditary Gingival Fibromatosis. A patient with gingival overgrowth of hereditary gingival fibromatosis.

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2. **Infections of Bacterial Origin**
   a. Non–dental biofilm-induced bacterial infections of the gingiva are uncommon. Gingival diseases in this category are characterized by a bacterial infection of the gingiva by a specific bacterium that is *not commonly found in a typical bacterial plaque biofilm*.
      1. Gingival diseases of specific bacterial origin occur on rare occasions when a bacterial infection overwhelms the host resistance. Examples include infections with *Neisseria gonorrhoeae*, *Treponema pallidum*, and *Streptococcal* species.[43]
      2. The gingival lesions manifest as painful ulcerations, chancres, or mucous patches with atypical gingival inflammation.

Description of Selected Inflammatory and Immune Conditions and Lesions

1. **Hypersensitivity Reactions: Intraoral Allergic Reactions**. Intraoral allergic reactions can be caused by ingredients in toothpastes, mouthwashes, or chewing gum.[44,45] These reactions are usually the result of flavor additives or preservatives in the product. Flavor additives known to cause gingival reactions are cinnamon and carvone.
   a. **Occurrence of Intraoral Allergic Reactions**
      1. Allergic reactions occur most commonly in patients who have a history of allergic conditions such as hay fever, allergic skin rashes, or asthma.
      2. Allergic patients seem to be particularly sensitive to the flavoring agent. The flavoring agent in toothpastes and mouthwashes is usually the most allergenic component.

   b. **Clinical Manifestations.** The clinical manifestations of allergy are a diffuse fiery red gingivitis sometimes with ulcerations (**Figure 5-37**).

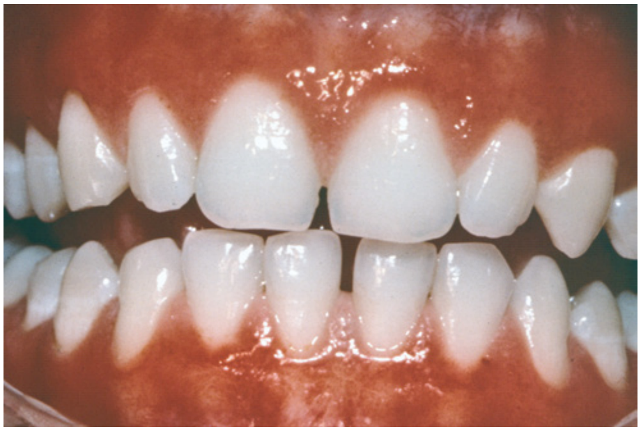

Figure 5-37 Allergic Reaction. Clinical signs of allergic reactions in the gingival tissues include redness extending from the gingival margin to the mucogingival junction.

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   c. **Recognition and Treatment of Allergic Reaction**
      1. The dental clinician might suspect an intraoral allergic reaction in a patient with good self-care who previously has had healthy gingiva (especially if the patient has a history of allergies). The dental clinician should inquire if the patient is using a new toothpaste, mouthwash, or chewing gum.
      2. Advise the patient to change brands or flavors of gum, toothpaste, or mouthwash. Cessation of the allergen-containing product should result in a resolution of gingival inflammation.
      3. If necessary, the diagnosis of allergic response can be confirmed by a biopsy with a diagnosis of plasma cell gingivitis.
      4. When the manufacturer becomes aware of allergic reactions, the flavoring agent or additive causing the problem is usually altered. For this reason, the patient may switch back to the original product (after 6 to 12 months) and use it without a problem.

2. **Erythema Multiforme**
   a. **Disease Characteristics**
      1. Erythema multiforme is an uncommon acute immune inflammatory disorder of the skin and/or oral mucosa.[46] The characteristic hallmarks of this condition are large, symmetrical erythematous papules, resembling a target, that appear on the skin in a circular pattern.
      2. On mucous membranes, it begins as blisters which progresses to ulcers (**Figure 5-38**). Oral involvement occurs in as many as 25–60% of cases and is sometimes the only involved site.
      3. The exact cause is unknown, though may involve a hypersensitivity reaction.

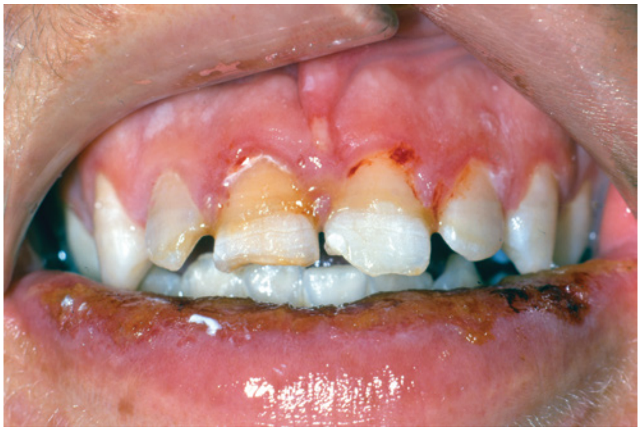

Figure 5-38 Erythema Multiforme. Erythema multiforme with ulcerations of the gingiva and crust formation of the lower lip.

Courtesy of Dr. Ralph Arnold, San Antonio, TX.

3. **Oral Lichen Planus**
   a. **Disease Characteristics**
      1. **Lichen planus** (LIE-kun play-nus) is a common inflammatory condition that can affect the skin, hair, nails, and mucous membranes.[47]
      2. On the skin, lichen planus usually appears as purplish, often itchy, flat-topped bumps, developing over several weeks. In the mouth and other areas covered by a mucous membrane, lichen planus forms lacy white patches, sometimes with painful sores. **Figure 5-39** shows two different presentations of lichen planus.
      3. The initial episode of oral lichen planus may last for weeks or months. Unfortunately, oral lichen planus is usually a chronic condition and can last for many years.
      4. Symptoms usually can be managed, but people who have oral lichen planus need regular monitoring because they may be at risk of developing oral cancer in the affected areas. It is important to diagnose, treat, and follow patients through regular oral examinations.[48,49]
      5. Good patient self-care can relieve the painful symptoms of the gingival lesions.[50]

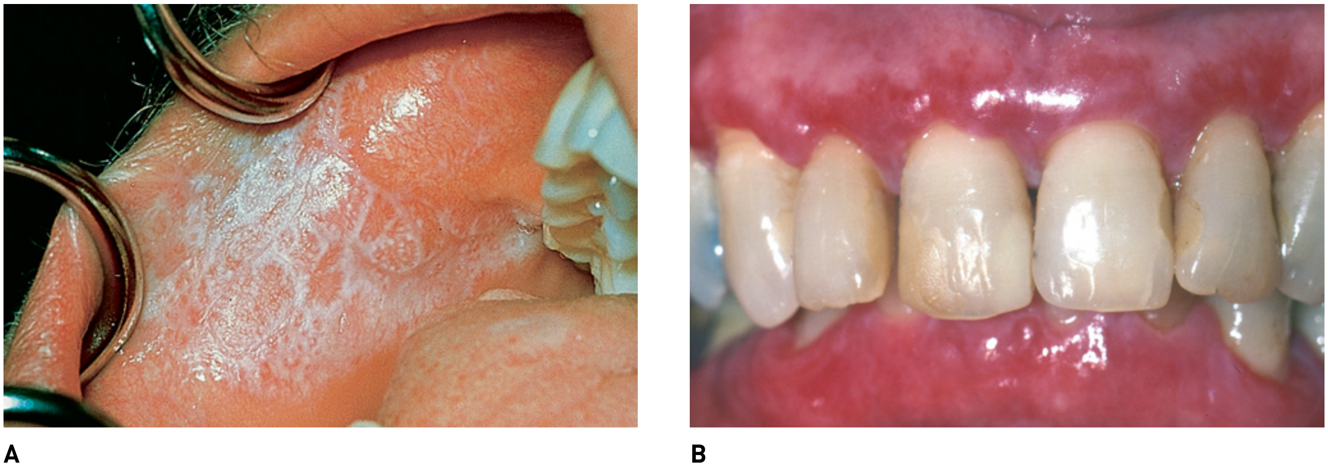

Figure 5-39 Lichen Planus of Buccal Mucosa. A. These delicate white lines arranged in a lacy web-like network are a characteristic feature of the reticular pattern of lichen planus, known as Wickham's striae. **B.** Oral lichen planus of the maxillary gingiva. The gingival tissues are erythematous, ulcerated, and painful.

A: Used with permission from Langlais RP, *Color Atlas of Common Oral Diseases*. Wolters Kluwer; 2016. © Wolters Kluwer; **B:** Courtesy of Dr. Ralph Arnold, San Antonio, TX.

b. **Clinical Manifestations**
   1. Six types of clinical manifestations of oral lichen planus have been described: papular, reticular, plaque type, erythematous, ulcerative, and bulbous lesions.
      a. The reticular pattern is commonly found bilaterally on the buccal mucosa as lacy web-like, white threads that are slightly raised. These is known as Wickham's striae, and is pathognomonic of oral reticular lichen planus (Figure 5-39A).
      b. The ulcerative pattern can affect any mucosal surface, including the buccal mucosa, tongue, and gingiva. Ulcerative lichen planus is painful and associated with intense erythema of the gingiva.
   2. Because the clinical appearance of lichen planus may mimic other types of non–dental biofilm-induced gingival lesions, a biopsy and an oral pathology consultation will be required to determine the definitive diagnosis and treatment.

Chapter Summary Statement

This chapter reviews the criteria for periodontal health and gingival diseases/conditions as developed by the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions.

Periodontal health is defined as the absence of any clinical signs of inflammation. Periodontal health can occur on an intact periodontium, a reduced periodontium due to non-periodontitis reasons, or a reduced periodontium due to periodontitis.

Dental biofilm-induced gingivitis is the most common of the periodontal diseases. Clinically, dental biofilm-induced gingivitis is characterized by gingiva that is red, swollen, bleeds easily, and is slightly tender. Gingival diseases are the mildest form of periodontal disease, and patients with gingivitis can revert to a state of health. Like periodontal health, dental-biofilm-induced gingivitis can occur on an intact periodontium, on a reduced periodontium due to non-periodontitis reasons, or on a reduced periodontium due to periodontitis. Dental biofilm-induced gingivitis may also be modified by systemic factors, medications, or malnutrition.

Non–dental biofilm-induced gingival diseases are a group of uncommon gingival lesions that are *not caused by plaque biofilm*. Non–dental biofilm-induced gingivitis can result from such diverse causes as bacterial, viral, or fungal infection, inflammatory conditions, allergic reactions, or trauma.

Section 5

Focus on Patients

Clinical Patient Care

CASE 1

A patient new to your dental team has been appointed with you for a dental prophylaxis. The patient has just relocated to your town. The patient tells you that he saw a dentist just before moving who told him that he has gingivitis. During your discussion with the patient, he asks if there is some way he can tell at home if he has gingivitis. How might you reply to this patient's question?

CASE 2

Reading through your patient's treatment notes from the previous visit, you notice the clinician documented "presence of gingival inflammation." Explain why this statement is not adequate documentation. What would you add to the description to provide another clinician with a clear verbal description of the clinical features of the patient's gingival tissues?

CASE 3

You are scheduled to perform nonsurgical periodontal instrumentation on a patient with a diagnosis of localized severe biofilm-induced gingivitis. At the time of the appointment the patient informs you that she is pregnant. With this information in hand, what would be the most appropriate periodontal diagnosis?

CASE 4

A patient who has been cared for by your dental team suddenly exhibits poor self-care with quite a bit of plaque biofilm accumulation. This is unusual for this patient. Discussions reveal that the patient is having difficulty with brushing and flossing due to soreness of the mouth. Examination reveals numerous small mucosal ulcers. Further discussions reveal that the patient has been experiencing this soreness since she began using tartar control toothpaste. How might your dental team manage this patient's diminished effectiveness of self-care?

CASE 5

Your patient is a 12-year-old male, who, despite good self-care, presents with generalized marginal redness and bleeding upon probing. His demonstration of toothbrushing and flossing indicates high dexterity and an ability to remove plaque biofilm. In talking with his mother, she confirms that he practices daily oral hygiene. How would you explain the presence of gingival disease to this patient and what would you recommend to improve his gingival health?

CASE 6

Your patient reports a change in his medical history from last visit; he is now taking Depakote as a mood stabilizer. The drug reference manual states it is an anticonvulsant and may cause gingival enlargement. How will this new information alter your plan for dental hygiene care and patient education?

Ethical Dilemma

Lily E., a 17-year-old high school senior and routine 6-month recall patient, is your first patient of the afternoon. She received her driver's license 3 months ago and has driven herself to today's appointment. You review her medical history, and she states there are *"no changes and that she has no chief complaint."*

As you are performing your intraoral examination, you notice that the tissue between the maxillary central incisors does not appear to be normal. You observe a mushroom-shaped gingival mass projecting from the gingival papilla. It appears red, and bleeds easily upon digital palpation. You ask Lily if it bothers her, but she denies any discomfort. She also states that she was not aware of any problem.

You are concerned that the lesion may be a pyogenic granuloma associated with pregnancy. You would like to discuss the possible implication of this lesion with Lily, but you are not sure how to proceed.

1. What is the best way for you to handle this ethical dilemma?
2. What is the best way to address/discuss Lily's treatment plan with her?
3. Under the ethical principle of confidentiality, can you discuss this with your employer dentist, without violating Lily's confidentiality?
4. Do you have the right to divulge your findings and concerns to her parents?
5. Can a 17-year-old consent to treatment, or must you receive parental consent?

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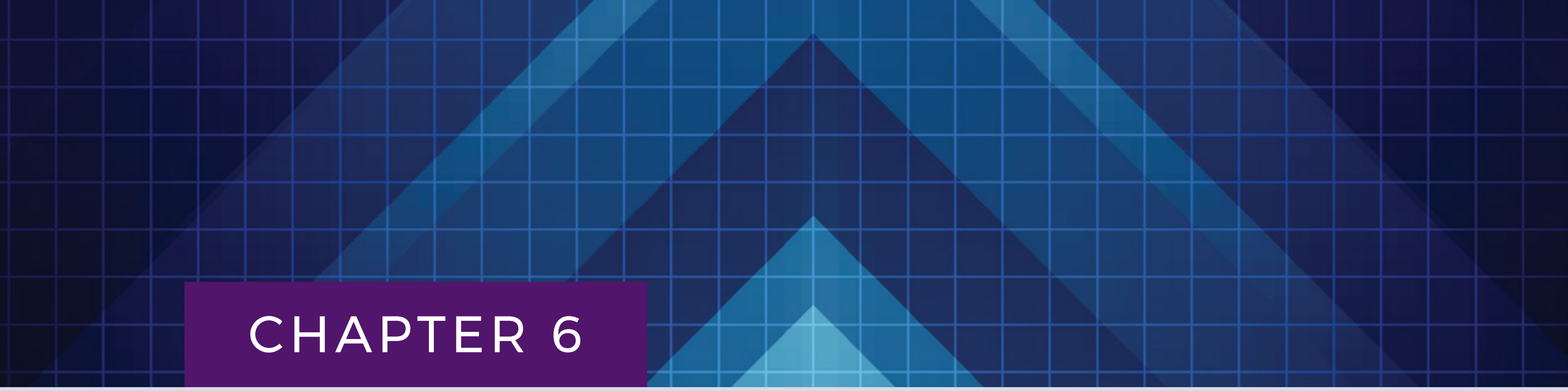

CHAPTER 6

Periodontitis

- Section 1 Periodontitis
  - Definition of Periodontitis
  - Characteristics of Periodontitis
  - Extent and Progression of Periodontitis
  - Therapeutic Endpoints and Goals
  - Recurrent and Refractory Forms of Periodontitis
- Section 2 Periodontitis Staging and Grading System
  - The Periodontitis Case Definition Criteria
  - Examples of Periodontitis Case Staging and Grading
- Section 3 Focus on Patients
  - Clinical Patient Care
  - Ethical Dilemma

CLINICAL APPLICATION

Periodontitis is a vast disease category that encompasses three conditions: periodontitis, necrotizing periodontal diseases, and periodontitis as a manifestation of systemic disease. All three conditions have a devastating impact on the periodontal tissues, and if left untreated, jeopardize the longevity of the tooth and may even have a long-lasting undesirable impact on systemic health. Consequently, all dental health care providers should be able to recognize the pathognomonic signs and symptoms of these conditions and be ready to render care designed to bring them under control. Of the three conditions listed above, periodontitis is the most prevalent. Thus, the focal point of this chapter centers on periodontitis. Other forms of periodontitis, specifically, necrotizing periodontal diseases and periodontitis as a manifestation of systemic diseases, will be discussed in later chapters.

LEARNING OBJECTIVES

- Describe the clinical signs and symptoms that are pathognomonic (*def*: specifically characteristic or indicative of a disease) of periodontitis.
- Define the term *clinical attachment loss* and explain its significance in staging and grading periodontitis.
- In the clinical setting, be able to explain to your patient the warning signs of periodontal disease and why these signs should not be ignored.
- Describe the radiographic hallmarks of periodontitis.
- Explain how disease severity and complexity of management play a role in determining the staging of periodontitis.
- Explain the differences in criteria of direct evidence of disease progression versus indirect evidence of disease progression and be able to describe the roles each plays in periodontitis grading.
- List the grade modifiers used to determine the grade of periodontitis.
- Define the meaning of the descriptors *recurrent* and *refractory* as they pertain to periodontitis.

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KEY TERMS

Periodontitis
Clinical attachment loss (CAL)
Extent
Localized periodontitis
Generalized periodontitis
Disease progression
Site-specific
Compromised periodontal maintenance
Recurrent form
Refractory form
Periodontitis case
Pathophysiology
Stage
Grade
Grade modifiers

Section 1

Periodontitis

Periodontitis is a bacterial-induced infection that triggers the host inflammatory response. This inflammatory response, in turn, results in irreversible pathologic destruction of all four tissues of the periodontium: gingiva, periodontal ligament, bone, and cementum. As depicted in **Figure 6-1**, the 2017 AAP/EFP Classification of Periodontal and Peri-Implant Diseases and Conditions separates the periodontitis category into three conditions: periodontitis, necrotizing periodontal diseases, and periodontitis as a manifestation of system disease.[1] This chapter focuses solely on the periodontitis subcategory. Content on the subcategory necrotizing periodontal diseases is presented in Chapter 9; content on the subcategory periodontitis as a manifestation of systemic disease is discussed in Chapters 14 and 15.

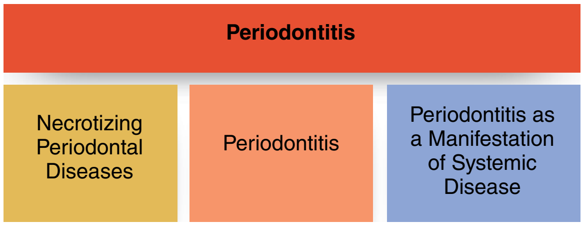
Periodontitis
Necrotizing Periodontal Diseases
Periodontitis
Periodontitis as a Manifestation of Systemic Disease

Figure 6-1 Three Forms of Periodontitis. The 2017 AAP/EFP Classification of Periodontal and Peri-Implant Diseases and Conditions seperates the category of periodontitis into three subcategories: necrotizing periodontal diseases, periodontitis, and periodontitis as a manifestation of systemic disease.

Modified from Caton JG, Armitage G, Berglundh T, et al. A new classification scheme for periodontal and peri-implant diseases and conditions—introduction and key changes from the 1999 classification. *J Periodontol*. 2018;89(S1):S1–S8. doi:10.1002/JPER.18-0157. Used with permission by John Wiley & Sons.

The clinician may encounter the terms *chronic periodontitis* and *aggressive periodontitis* when reviewing patient chart notes or reading journal articles predating 2017. However, over the past several decades (since the 1999 classification system was released), clinicians failed to reach a consensus as to whether chronic periodontitis and aggressive periodontitis were truly distinct diseases with the same pathophysiology. Consequently, because of a paucity of data to support the notion that chronic and aggressive periodontitis are indeed different diseases, these two disease forms were eliminated from the 2017 disease classification.[2,3] The clinician should be aware that these are historical terms which are no longer valid diagnostic terms under the current disease classification system.

Definition of Periodontitis

Periodontitis is a complex microbial infection that triggers a host-mediated inflammatory response within the periodontium, resulting in progressive irreversible destruction of the periodontal ligament and supporting alveolar bone.[4]

According to epidemiologic data, periodontitis is the number one cause of tooth loss in adults.[5] To illustrate, according to the U.S. Centers for Disease Control and Prevention, approximately 47.2% of adults older than 30 years suffer from periodontitis. As a result, periodontitis is a significant public health problem.[6]

- Periodontitis begins as biofilm-induced gingivitis. Biofilm-induced gingivitis is a reversible condition that can potentially revert to a state of health if managed appropriately and in a timely manner. If left untreated, however, gingivitis may progress to periodontitis.
- *When a patient is diagnosed with periodontitis, he or she will remain a periodontitis patient for life—even following successful periodontal therapy—and will require lifelong supportive care to maintain the stability of the periodontium and to prevent disease recurrence.*[4]
- **Figure 6-2** depicts the disease characteristics of periodontitis.

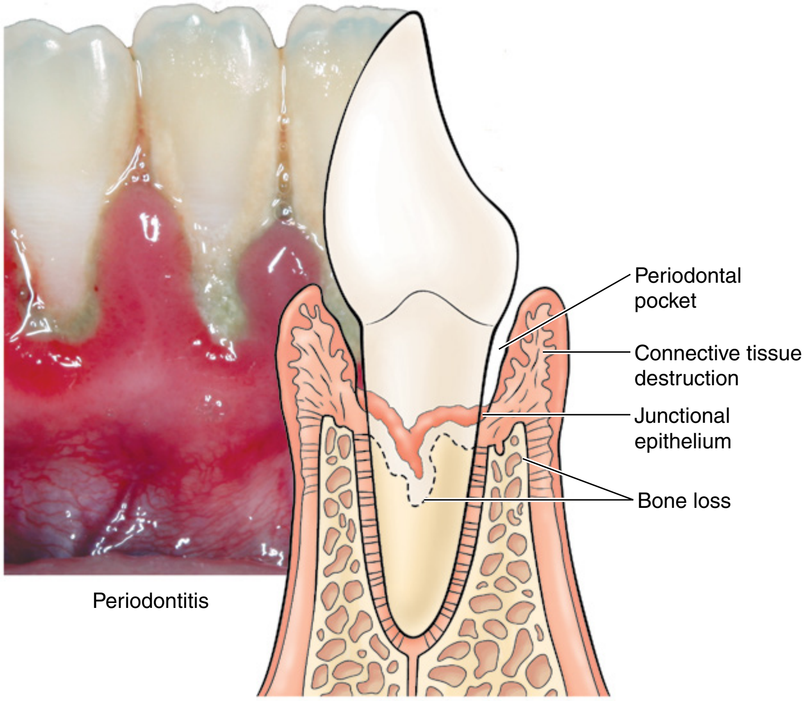

Figure 6-2 **Disease Characteristics of Periodontitis.** Periodontitis is initiated by the buildup of periodontal pathogens and the ensuing host-mediated inflammatory response within the supporting tissues of the teeth, leading to progressive irreversible destruction of the periodontal ligament and loss of supporting alveolar bone.

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Characteristics of Periodontitis

1. **Signs and Symptoms of Periodontitis.** Clinical signs and symptoms of periodontitis include accumulation of plaque biofilm and dental calculus, redness (erythema), swelling (edema), gingival bleeding, suppuration (pus), periodontal pockets, clinical attachment loss, and tooth mobility. The radiographic hallmark is the presence of crestal bone loss and/or interfurcal bone loss (in multirooted teeth).
   a. **Alterations in Color, Texture, and Size of the Marginal Gingiva**
      1. **Reddish or Purplish Tissue.** In periodontitis, the gingiva may appear bright red or purplish.
         a. In such cases, the clinical signs of periodontitis are very evident at the initial examination of the oral cavity. The gingiva appears swollen with the color ranging from pale red to magenta. Alterations in gingival contour and form are evident, such as rolled gingival margins and blunted or flattened papillae.
         b. An example of periodontitis exhibiting this type of appearance is shown in **Box 6-1**, **Figure 6-3A**.

2. **Pale Pink Tissue.** In periodontitis, the gingival tissue may also appear pale pink and have an almost healthy-looking appearance. An example of periodontitis exhibiting this type of appearance is shown in Box 6-1, **Figure 6-3B**.

Box 6-1 Periodontitis

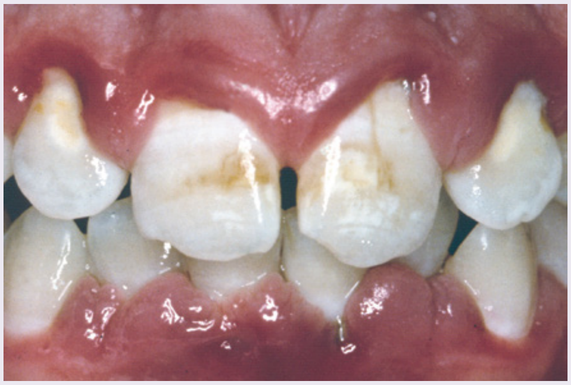

Figure 6-3A Overt Visible Changes in the Gingiva. In this example of periodontitis, the visual signs of inflammation are overt (obvious), such as erythematous gingiva, rolled gingival margins, and bulbous papillae.

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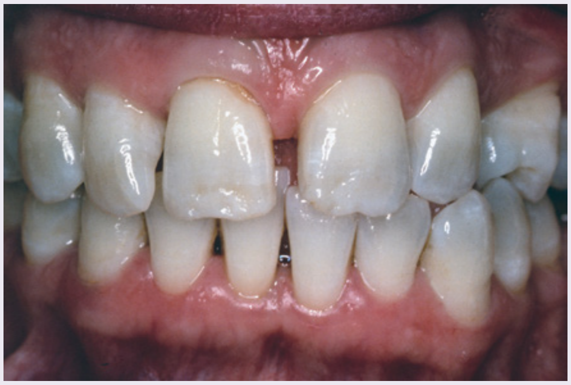

Figure 6-3B Minimal Visible Changes in the Gingiva. In this example of periodontitis, there are minimal visible tissue changes. Because periodontitis is a disease affecting the deeper tissues of the periodontium, the visual appearance of the gingival tissues often may be an unreliable indicator of disease onset and/or disease activity.

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Characteristics of Periodontitis

- Most commonly seen in adults, but can occur in children and adolescents.
- Initiated and continued by plaque biofilms, but host response plays an essential role in disease progression.
- Signs and symptoms of inflammation include moderate or heavy deposits of plaque biofilm and dental calculus, redness (erythema), swelling (edema), gingival bleeding, suppuration (pus), periodontal pockets, bone loss, and tooth mobility.
- Bone loss may be evident on radiographs (**Figure 6-3C**).
- Untreated periodontitis most commonly progresses slowly over time; however, in rare cases there is rapid disease progression.
- Attachment loss may occur in one site of a single tooth, on several teeth, or the entire dentition.
- It can be modified by other factors, such as smoking and poorly controlled diabetes.
- The clinical appearance of the tissues is not a reliable indicator of the presence or severity of periodontitis. Refer to **Figures 6-4** to **6-6**.

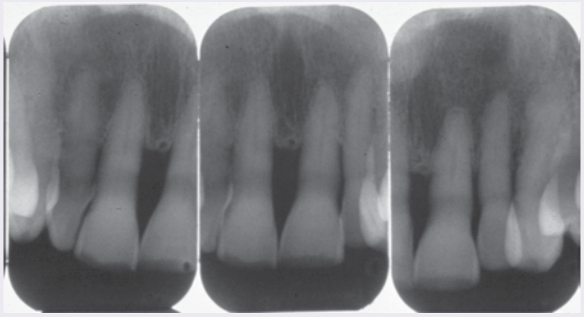

Figure 6-3C Radiographic Evidence of Periodontitis. Dental radiographs of a patient with periodontitis reveal a horizontal pattern of alveolar bone loss.

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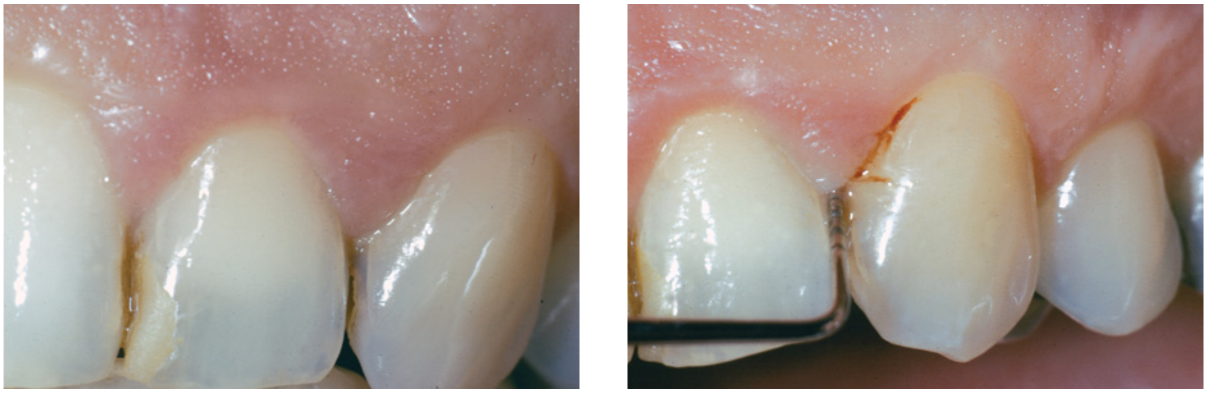

Figure 6-4 **Health or Disease?** The visual appearance of the tissue in the left image suggests health. However, when assessed with a periodontal probe (right image), it is evident that there is a deep 7-mm pocket on the mesiofacial of the canine. This example underscores the importance of a thorough periodontal assessment that includes a visual, clinical, and radiographic assessment of the periodontium.

Courtesy of Dr. Don Rolfs, Periodontal Foundations, Wenatchee, WA.

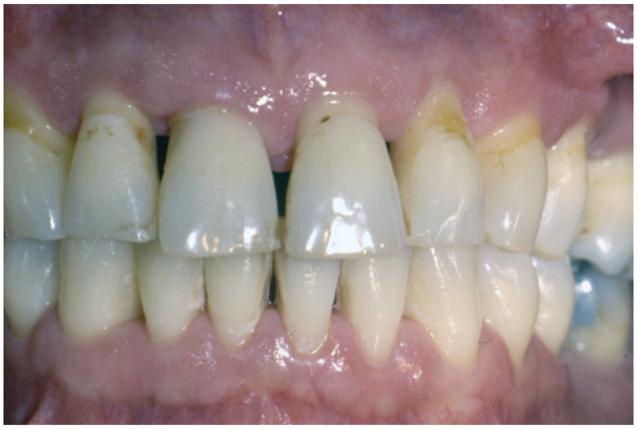

Figure 6-5 **Health or Disease?** This individual received periodontal treatment for periodontitis several years ago. The assessment at today's appointment reveals meticulous patient self-care with no additional attachment loss since being enrolled in a periodontal maintenance recall program several years ago. Therefore, this tissue is considered healthy. The attachment loss is simply an indicator of previous disease.

Courtesy of Dr. Ralph Arnold, San Antonio, TX.

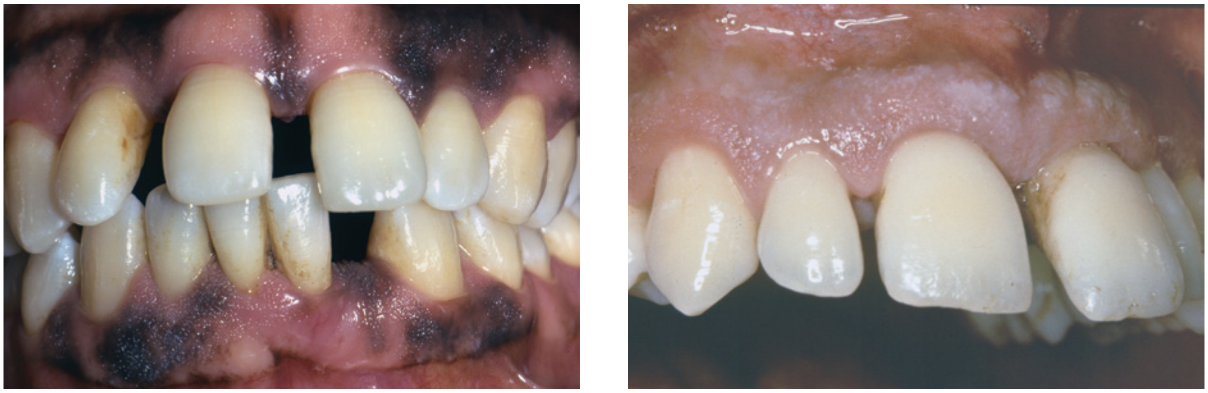

Figure 6-6 **Periodontitis.** Two examples of periodontitis showing firm, nodular (fibrotic) tissue changes.

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    a. In many patients, the changes in color, contour, and consistency may not be visible upon inspection. At first glance, an inexperienced clinician may mistake the clinical appearance of periodontitis for one of health. Closer examination, however, will reveal firm, rigid (fibrotic) tissue, the presence of pocketing, and bleeding upon probing. Periodontitis exhibiting a fibrotic appearance is shown in Figure 6-6.

b. **Bleeding, Crevicular Fluid, and Exudate**
  1. Gingival bleeding—either spontaneous bleeding or bleeding in response to probing—is common.
  2. Increased flow of gingival crevicular fluid or suppuration (pus) from periodontal pockets is common.

c. **Plaque Biofilm and Calculus Deposits**
  1. Periodontitis is initiated by mature supra- and subgingival plaque biofilms and calculus deposits. Teeth with periodontitis usually have very complex and thick deposits of plaque biofilm on affected root surfaces.
  2. Although periodontitis is initiated and sustained by plaque biofilm, host factors determine the pathogenesis and the progression of the disease.

d. **Loss of Attachment**
  1. **Clinical attachment loss (CAL)** is a measurement of the amount of destruction affecting tooth-supporting structures that have been destroyed around a tooth. Loss of attachment occurs in periodontitis and is characterized by (1) apical migration (relocation) of the junctional epithelium to the tooth root, (2) destruction of the fibers of the gingiva, (3) destruction of the periodontal ligament fibers, and (4) loss of alveolar bone support from around the tooth. The changes that occur in the alveolar bone in periodontitis are significant because loss of bone height eventually can result in tooth loss.
  2. Accompanying features of attachment loss include:
    a. Loss of alveolar bone support to the teeth.
    b. Periodontal pockets or apical migration of the gingival margin.
    c. Furcation involvement in multirooted teeth (**Figure 6-7**).

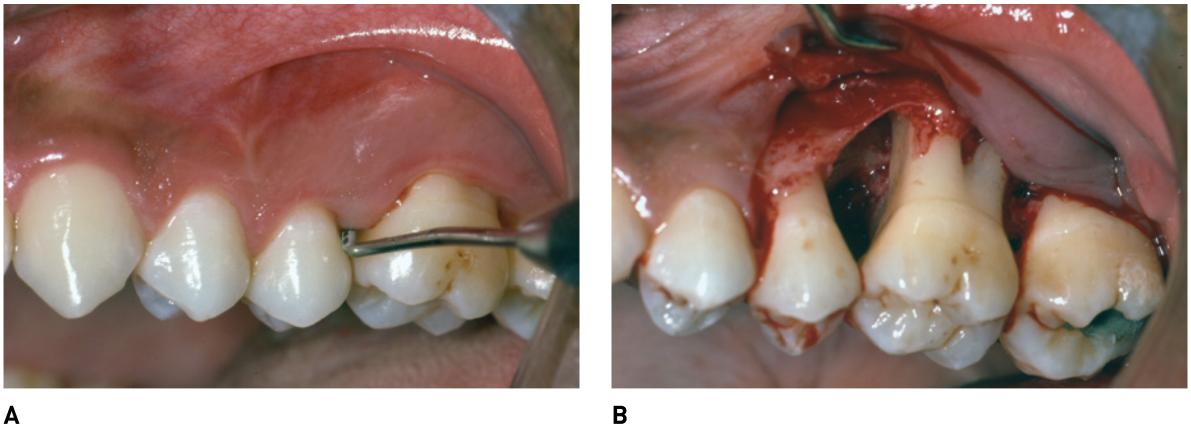

Figure 6-7 Attachment Loss. A. Assessment with a periodontal probe indicates severe loss of attachment on this molar tooth. **B.** Periodontal flap surgery reveals severe loss of alveolar bone and furcation involvement.

Courtesy of Dr. Ralph Arnold, San Antonio, TX.

d. Tooth mobility and/or drifting, known as pathologic tooth migration (**Figure 6-8**).

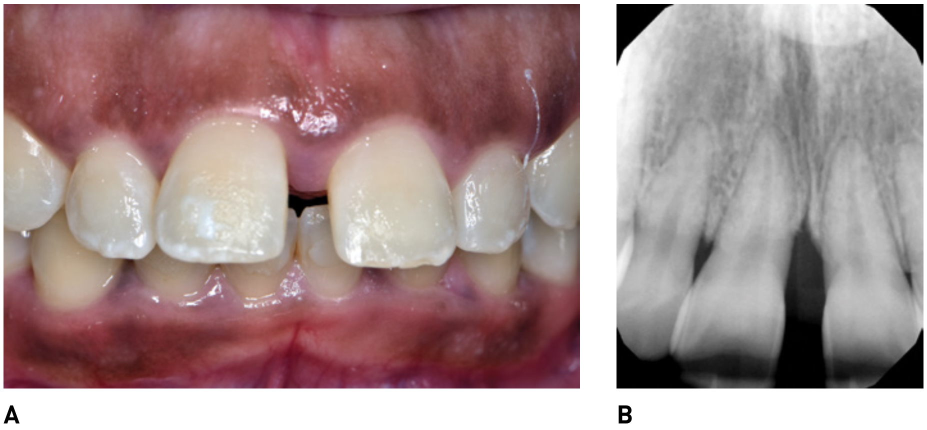

Figure 6-8 Pathologic Tooth Migration. An 18-year-old patient presented with increasing spacing between the maxillary incisors that have drifted apart over a 1-year period. **A.** Note the divergence of the two incisors. Deep probing depth of 6 mm was noted on the mesial aspects of both incisors with Class III mobility. **B.** Radiographic evidence of severe bone loss consistent with pathological migration or drifting of incisors due to periodontitis.

e. **Deeper Probing Measurements.** It is well established that the extent of probe penetration is influenced by the inflammatory status of the periodontal tissue.[7–20] **Figure 6-9** diagrams the relationship between the level of tissue inflammation and the depth of periodontal probe tip penetration.

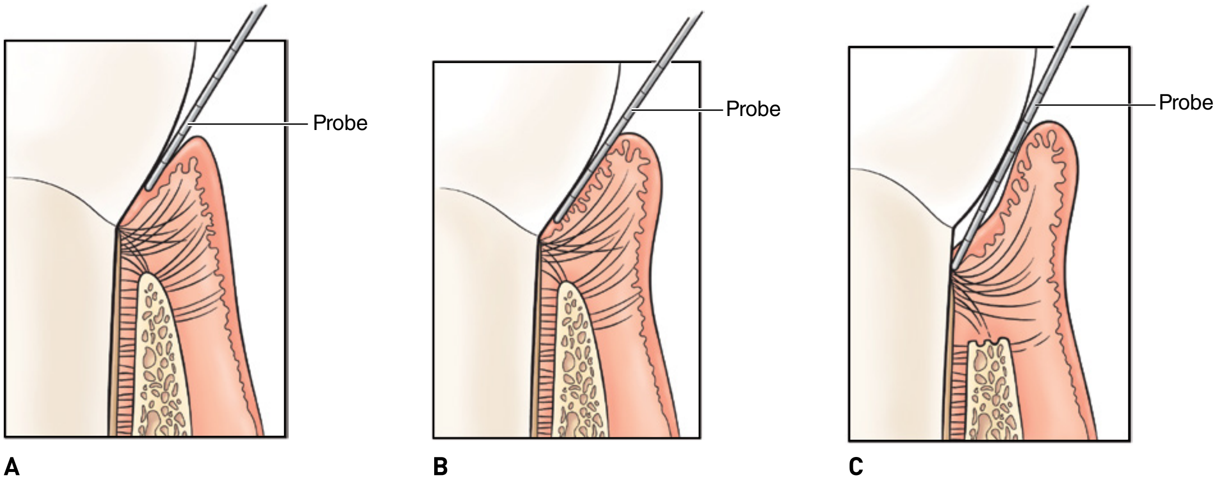

Figure 6-9 Diagrammatic Representation of Probe Tip Penetration Relative to the Periodontal Tissues. The depth of probe penetration is influenced by the inflammatory status of the periodontal tissues. **A.** In a healthy sulcus, the probe penetrates about one-third of the length of the junctional epithelium. **B.** In moderate inflammation, the probe tip penetrates approximately half the length of the junctional epithelium. **C.** In severe inflammation, the probe tip penetrates through the entire length of the junctional epithelium and only stops when it encounters the most coronal intact collagen fibers in the gingival connective tissue.

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f. **Localized or Generalized Inflammation.** In periodontitis, there is no consistent pattern to the number and the types of teeth involved.
    1. Localized inflammation may involve one site on a single tooth, several sites on a tooth, or several teeth, whereas generalized inflammation may involve the entire quadrant or the entire dentition.
    2. In some cases, the **site-specific** nature of periodontitis may be apparent. For instance, a patient may present with areas of health in most sites interspersed with a single site or localized sites exhibiting clinical attachment loss.

g. **Contributing Factors**
    1. Periodontitis may be modified by factors that increase the susceptibility of a patient to the disease. For instance, environmental factors (such as smoking), systemic factors (such as diabetes or HIV), and genetic factors can contribute to increasing an individual's susceptibility to periodontitis.
    2. Even local intraoral factors, such as tooth crowding or an overhanging restorative margin, may contribute to an increased susceptibility to plaque-induced inflammation.

h. **Symptoms**
    1. Periodontitis usually is painless. This is unfortunate because an individual with mild forms of periodontitis may be totally unaware of the disease until the clinical signs of periodontitis (such as profuse gingival bleeding or tooth mobility) become more apparent. As a result, some periodontitis patients may either delay interventional treatment or not seek treatment until the very last moment.
    2. In more advanced cases, however, individuals may first become aware that something is wrong when they notice that their gingiva bleed when brushing, that spaces occur between the teeth (pathologic tooth migration), or that teeth have become mobile.
    3. Patients may complain of food impaction, sensitivity to hot or cold due to exposed roots, or dull pain radiating into the jaw.

2. **Onset of Periodontitis**
    a. **Gingivitis as a Risk Factor for Periodontitis**
        1. Findings from epidemiologic studies and clinical trials indicate that the presence of gingivitis may be regarded as a risk factor for periodontitis.[21,22]
        2. Dental biofilm-induced gingivitis always precedes the onset of periodontitis. However, in most cases, dental biofilm-induced gingivitis may remain stable for many years, and will not progress to periodontitis. The level of host susceptibility and other contributing factors determine whether gingivitis progresses to periodontitis.
        3. Gingivitis manifests only after days or weeks of plaque biofilm accumulation. In most cases, periodontitis requires longer periods (years) of plaque biofilm and calculus exposure to develop.[23]

    b. **Age of Onset.** The onset of periodontitis may be at any age. It most commonly affects adults, but can also be seen in children and adolescents. The prevalence and severity of periodontitis increases with age.

3. **Patient Education: The Warning Signs of Periodontitis**
    a. **Pain Usually Not a Symptom of Periodontitis.** As discussed above, the absence of pain may explain why periodontitis advances unnoticed before the patient seeks treatment and why a patient may neglect treatment even after receiving the diagnosis of periodontitis.
        1. The warning signs of periodontitis are red or swollen gingiva, bleeding during brushing, a bad taste in the mouth, persistent bad breath, sensitive teeth, loose teeth, and pus around teeth and gingiva.
        2. Tools such as oral health self-evaluations distributed at health fairs or other events can be helpful in increasing the public's awareness of the signs and symptoms of periodontal disease.

Extent and Progression of Periodontitis

1. **Extent of Destruction in Periodontitis**
    a. **Extent** is the distribution of periodontitis throughout the entire oral cavity. It can be thought of as the degree to which the periodontitis has spread within the oral cavity. The extent is based on the percentage of affected teeth that exhibit periodontal breakdown divided by the total number of teeth present in the mouth at the time of diagnosis.
        1. Localized inflammation may involve one site (single site) on a single tooth, several sites on a tooth, or several teeth. A patient may simultaneously have areas of health adjacent to areas with periodontitis.
        2. Generalized inflammation may involve many teeth or the entire dentition.

   b. **Localized or Generalized Extent**
      1. **Localized periodontitis** is periodontitis in which less than 30% the teeth in the mouth exhibit attachment loss and/or bone loss.
      2. **Generalized periodontitis** is periodontitis in which 30% or more of the teeth in the mouth exhibit attachment loss and/or bone loss.

2. **Disease Progression**
   a. **Disease progression** refers to the changes that occur *over time* in the periodontal tissues due to the natural progression of the disease as it advances from its earliest stages to its peak. For example, how does the amount of attachment loss and bone destruction seen today compare with what was observed initially? Is it the same, somewhat worse, or much worse?
   b. **Progression of Periodontitis.** The exact natural course of untreated periodontitis has never been fully ascertained and is still in debate. However, three different views of disease progression have been proposed:
      1. The first view is known as the continuous disease hypothesis. Under this hypothesis, untreated periodontitis progresses as a continuous gradual process. Long-term longitudinal models had estimated that untreated periodontitis may progress anywhere between 0.05 mm to 0.3 mm of attachment loss per year.
      2. The second view, however, hypothesizes that untreated periodontitis progresses randomly and in episodic bursts of activity—some sites demonstrate no disease activity, whereas other sites in the same mouth experience short random bursts of disease activity interspersed by periods of no disease activity. This model of periodontitis disease progression is known as the random burst model.
         a. Some diseased sites may remain dormant for long periods of time.[23–25]
         b. Other diseased sites may progress more rapidly. More rapidly progressing disease sites occur most frequently in interproximal areas and may be associated with areas of greater plaque biofilm accumulation, specific subgingival pathogens, and inaccessibility to plaque biofilm control measures (e.g., sites of malposed teeth, restorations with overhanging margins, areas of food impaction, deep periodontal pockets, and furcation areas).[25,26]
      3. The third view holds that periodontitis progresses in relatively short bursts during an individual's life followed by periods of extended remission. In other words, bursts of destructive periodontal disease activity may occur with high frequency during certain periods of an individual's life, then be dormant for the rest of the individual's lifespan (this type of activity is similar to the progression of acne, which predominantly affects adolescents and teenagers, but wanes as one reaches adulthood). This third view is known as the asynchronous burst hypothesis.
      4. To date, it remains unclear if untreated periodontitis progresses gradually or in bursts of activity interspersed with periods of remission.
      5. Regardless of which disease progression model prevails, the desired outcome of any periodontal therapy is to halt the progression of the disease. **Figures 6-10** to **6-12** show the clinical features before and after treatment for three different patients.

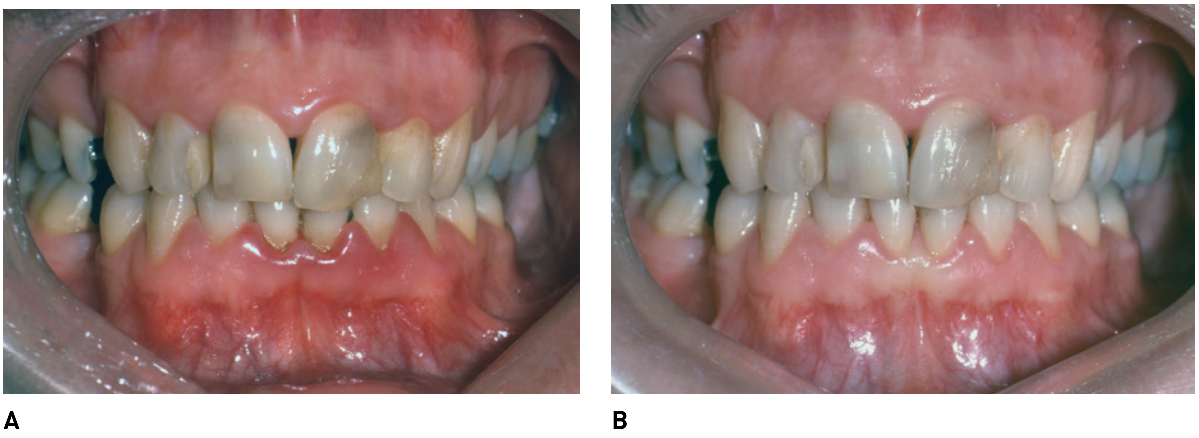

Figure 6-10 Periodontitis: Before and After Periodontal Therapy. A. Note the tissue appearance before periodontal therapy. Clinical inflammation is particularly pronounced on the mandibular anterior sextant. **B.** The same individual after treatment. Note the resolution of inflammation in the mandibular anterior sextant.

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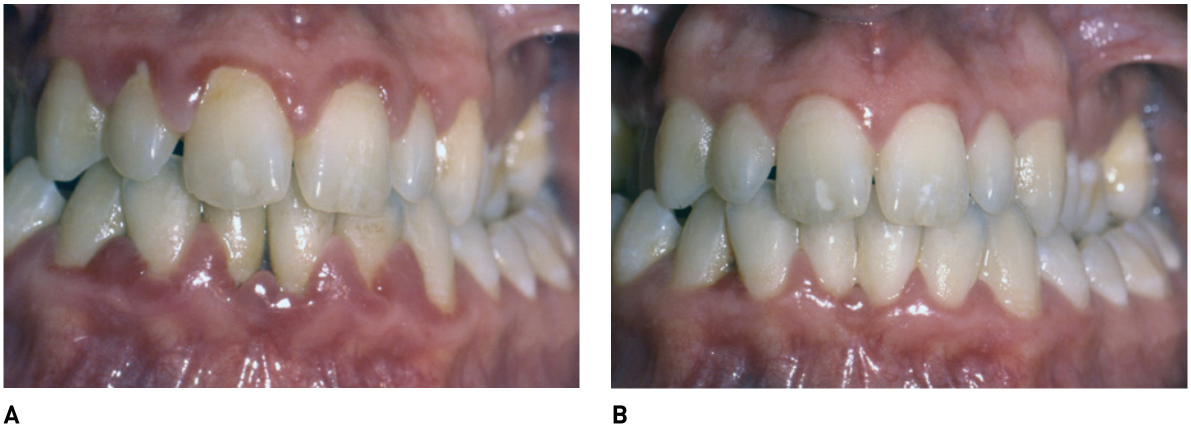

Figure 6-11 **Periodontitis: Before and After Periodontal Therapy. A.** Prior to therapy, very inflamed tissue is evident. **B.** Much improved clinical picture at 3-month follow-up appointment.

Courtesy of Dr. Ralph Arnold, San Antonio, TX.

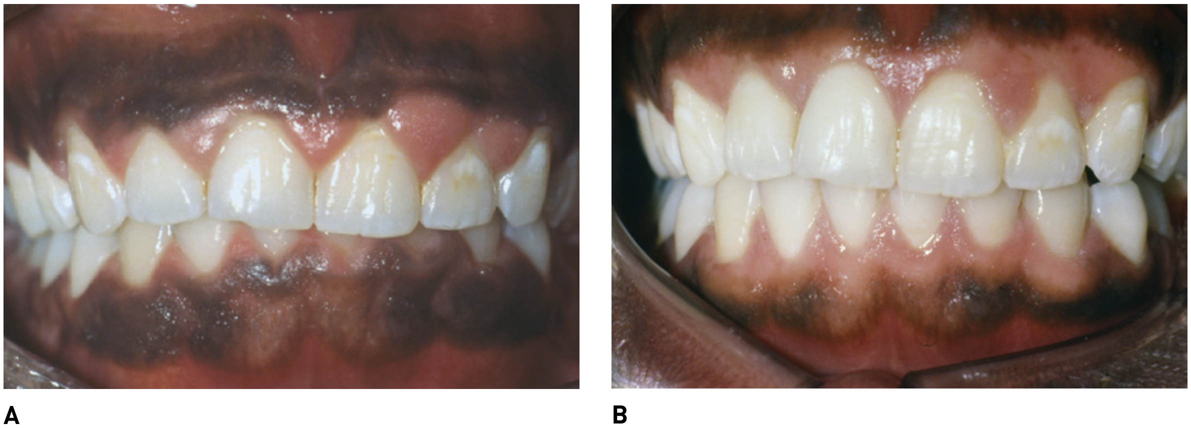

Figure 6-12 **Periodontitis: Before and After Periodontal Therapy. A.** Very swollen gingival tissues pre-treatment. **B.** The same individual after treatment.

Courtesy of Dr. Ralph Arnold, San Antonio, TX.

Therapeutic Endpoints and Goals

1. **Therapeutic Endpoints of Periodontal Therapy.** The therapeutic endpoints of periodontal therapy are (1) to eliminate the microbial etiology and contributing factors that perpetuate periodontal inflammation; (2) to preserve the current state of the teeth and periodontium in a state of health, function, and stability; (3) to prevent disease progression and reoccurrence; and (4) to reinforce behavioral modifications (i.e., better home care) that favor long-term compliance, improve oral health, and decrease the risk of disease reoccurrence. The last item is known as shared decision making. Not only is shared decision making a key component to the long-term success of periodontal therapy, but it also plays a critical role in patient-centered health care. Chapter 22 details the critical role shared decision making plays in balancing clinical evidence on risks and expected outcomes with patient preferences and values.
2. **Treatment Goals.** The goals of periodontal treatment are met if the clinician can (1) reinforce daily self-care; (2) perform effective periodontal instrumentation to remove the microbial etiology; (3) eliminate local intraoral factors contributing to the disease; (4) perform periodontal surgery (if there is still persistent periodontal inflammation following nonsurgical therapy); and (5) set a plan with the patient to adhere to a periodontal maintenance regimen after the disease is controlled. In certain cases, treatment that is not intended to attain optimal results may be warranted. This type of treatment is known as **compromised periodontal maintenance** and is designed to slow down (not eliminate or fully arrest) the progression of periodontitis in patients who may not respond favorably to active conventional periodontal therapy (such as nonsurgical scaling and root planing or periodontal surgery) due to serious health conditions, poor motivation/compliance, advanced age, or severe periodontal disease.

Recurrent and Refractory Forms of Periodontitis

In previous classification systems, recurrent periodontitis and refractory periodontitis were thought to be distinct and separate forms of periodontitis. However, because current evidence fails to support this contention, recurrent periodontitis and refractory periodontitis are excluded from the 2017 classification system. Nevertheless, it is important for dental providers to understand that there are numerous and complex contributing factors that can stymie the successful management of the disease in the long term. In such cases, two forms of periodontitis may arise: a recurrent form or a refractory form of periodontitis. (Chapter 31, Section 3 goes into greater detail of these two forms of periodontitis.)

1. **Recurrent form**—a return of destructive periodontitis that had been *previously arrested* by conventional periodontal therapy.
    a. Any individual with a prior history of periodontitis may be at risk of developing the recurrent form of the disease in the future.
    b. Recurrence of periodontitis is a common event, especially in patients with poor self-care or who are noncompliant with routine professional care.
    c. With recurrent disease, the patient and the dental practitioner must refocus their attention in arresting the disease and re-establishing patient adherence to the periodontal treatment, meticulous self-care, and frequent professional maintenance.
2. **Refractory form**—the form of periodontitis seen in a patient who has been previously treated and monitored over time, but exhibits *continuing* attachment loss despite the following conditions: (a) the patient receives appropriate and continuous professional periodontal therapy, (b) the patient practices satisfactory self-care, and (c) the patient follows the recommended program of periodontal maintenance visits. In other words, the patient previously underwent treatment and currently maintains adequate home care, but the periodontium, for whatever reason, remains unresponsive to treatment. **Box 6-2**, **Figure 6-13** shows an example of a refractory form of periodontitis.

Box 6-2 Refractory Form of Periodontitis

Ongoing and continued attachment loss in a patient despite all the following:

- Appropriate periodontal therapy was rendered.
- The patient practices satisfactory self-care.
- The patient has an appropriate program of periodontal maintenance visits.

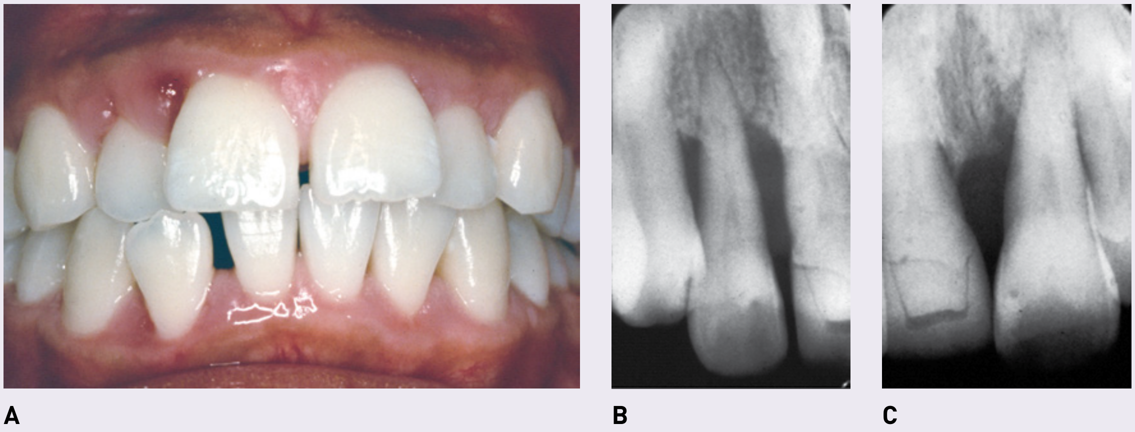

Figure 6-13 Refractory Form of Periodontitis. Periodontitis is considered refractory when the disease is not controlled by conventional periodontal therapy normally recommended for patients with periodontitis. In a refractory case, the patient experiences ongoing attachment loss despite undergoing effective periodontal therapy and performing satisfactory self-care. The dental radiographs of a patient with refractory form of periodontitis reveals evidence of continuing bone loss over time despite appropriate therapy.

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- a. The etiology of the refractory form of periodontitis remains unknown but may be due to a complexity of unknown factors such as the emergence of opportunistic pathogens or compromised host response to the bacterial attack.
- b. The designation "refractory" is applied to cases of periodontitis that do not respond favorably to conventional treatment.
- c. Because this form of periodontitis does not respond favorably to conventional therapy, periodontal therapy should be designed to slow down and control the progression of the disease.
- d. Treatment includes, but would not be limited to, patient education and behavior modification, periodontal instrumentation, use of systemic and/or local antibiotics, removal of periodontally hopeless teeth, correction of restorations that may be contributing to plaque retention, surgical therapy, and strict adherence to a periodontal maintenance regimen.
- e. Note that despite the patient's and the dental clinician's best efforts, control of periodontitis may not be possible in some refractory cases.

Section 2

Periodontitis Staging and Grading System

The Periodontitis Case Definition System

1. **Definition of a Patient as a Periodontitis Case**
   - a. According to the 2017 classification system, a patient is a **periodontitis case** if:
     1. Interdental clinical attachment loss is detectible at two or more nonadjacent teeth, *OR*
     2. Facial or lingual clinical attachment loss of 3 mm or more with pocketing greater than 3 mm is detectible at two or more teeth.
   - b. **Clinical Attachment in the Periodontitis Case Definition**
     1. To be defined as a periodontal case, the observed CAL cannot be attributed to non-periodontitis reasons, such as gingival recession of traumatic origin, dental caries extending to or apical to the cementoenamel junction (CEJ), presence of CAL on the distal aspect of a second molar associated with malposition or extraction of a third molar, an endodontic lesion draining through the marginal gingiva, or the occurrence of a vertical root fracture.
2. **Identification of the Form of Periodontitis**
   - a. **Pathophysiology**
     1. As related to periodontal disease, **pathophysiology** is the study of the pathological manifestations of periodontitis. Pathophysiology does not deal directly with the treatment of disease; rather, it explains the processes within the body that result in the signs and symptoms of periodontitis.
     2. Based on pathophysiology, three clearly different forms of periodontitis have been identified.[1]
        - a. Necrotizing periodontitis
        - b. Periodontitis as a manifestation of systemic diseases
        - c. Periodontitis
   - b. Content on periodontitis is explained in this chapter. Content on necrotizing periodontitis and periodontitis as a manifestation of systemic disorders are discussed in later chapters.
3. **The Periodontitis Staging and Grading System**
   - a. **Stage at Presentation.** An individual periodontitis case is categorized as **Stage** I, II, III, or IV.[3] The staging category of periodontitis is defined by (1) the disease severity (severity factors) and (2) the complexity of management of the case (complexity factors), which are summarized in **Table 6-1**.

Table 6-1 Periodontitis Stage

	Interdental CAL[a]	Radiographic Bone Loss	Tooth Loss	Probing Depth	Bone Loss	Other
Stage I	1 to 2 mm	Coronal third (> 15% loss)	No tooth loss due to periodontitis	4 mm or less	Mostly horizontal bone loss	
Stage II	3 to 4 mm	Coronal third (15–33% loss)	No tooth loss due to periodontitis	5 mm or less	Mostly horizontal bone loss	
Stage III	5 mm or more	Extending to the mid-third of root or beyond	4 or less teeth lost due to periodontitis	6 mm or more	Vertical bone loss 3 mm or greater	
Stage IV	5 mm or more	Extending to the mid-third of root and beyond	5 or more teeth lost due to periodontitis	6 mm or more	Vertical bone loss 3 mm or greater	Need for complex rehabilitation

[a] Interdental clinical loss of attachment at the site of greatest loss.

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b. When determining an individual's periodontitis staging category, the clinician should keep three things in mind. First, the severity score is primarily based on the interdental CAL at the *worst* affected site in the dentition. Second, there may be individual complexity factors and/or severity factors which shift the stage to a higher category. For instance, if a periodontitis patient has a maximum probing depth of 5 mm but a molar tooth with a Class II furcation, then that would shift the stage to either Stage III or Stage IV irrespective of other severity factors, such as CAL, radiographic bone loss, or tooth loss. Lastly, staging is a patient-based attribute and only a *single stage* is ascribed to an individual patient at a given time.

1. **Stage I Periodontitis.** Stage I represents the initial stages of periodontitis.
    a. **Disease Severity**
        1. Interdental CAL of 1 to 2 mm at the site of greatest loss.
        2. Radiographic bone loss extending to the coronal one-third of the root.
        3. No tooth loss due to periodontitis.
    b. **Complexity of Management**
        1. Maximum probing depths of 4 mm or less.
        2. Mostly horizontal bone loss.
2. **Stage II Periodontitis.** Stage II represents established periodontitis.
    a. **Disease Severity**
        1. Interdental CAL of 3 to 4 mm at the site of greatest loss.
        2. Radiographic bone loss extending to the coronal one-third of the root.
        3. No tooth loss due to periodontitis.
    b. **Complexity of Management**
        1. Maximum probing depths of 5 mm or less.
        2. Mostly horizontal bone loss.

3. **Stage III Periodontitis.** Stage III represents severe periodontitis with significant destruction to the attachment apparatus and potential tooth loss.
   a. **Disease Severity**
      1. Interdental CAL 5 mm or greater at the site of greatest loss.
      2. Radiographic bone loss extending to the mid-third of the root and beyond.
      3. Tooth loss due to periodontitis of four or fewer teeth.
   b. **Complexity of Management**—in addition to the Stage II complexity factors, one or more of the following complexity factors are characteristic of Stage III.
      1. Maximum probing depths of 6 mm or greater.
      2. Vertical bone loss 3 mm or greater.
      3. Class II or III furcation involvement.
      4. Moderate alveolar ridge defect that complicates implant placement.
4. **Stage IV Periodontitis.** Stage IV represents advanced periodontitis with extensive tooth loss and potential for loss of dentition.
   a. **Disease Severity**
      1. Interdental CAL of 5 mm or greater at the site of greatest loss.
      2. Radiographic bone loss extending to the middle one-third of the root and beyond.
      3. Tooth loss due to periodontitis of five or more teeth.
   b. **Complexity of Management**—in addition to the Stage III complexity factors, one or more of the following complexity factors are characteristic of Stage IV.
      1. Maximum probing depths of 6 mm or greater.
      2. Masticatory dysfunction.
      3. Secondary occlusal trauma; tooth mobility greater than Class II.
      4. Severe ridge defect of alveolar bone.
      5. Bite collapse; drifting, flaring of teeth.
      6. Fewer than 20 remaining teeth (10 opposing pairs).

c. **Grade. Grade** is an estimate of the *future* rate of progression of periodontitis (i.e., slow, moderate, or rapid). It measures the responsiveness to conventional periodontal therapy and the potential impact on systemic health.
   1. The primary criteria for grading are defined by direct or indirect evidence of disease progression.
   2. Direct evidence is based on a *longitudinal* evaluation of the progression of periodontitis by comparing the rate of attachment loss and/or bone loss over a period of 5 years or more.
   3. Indirect evidence, on the other hand, is based on:
      a. The percent of bone loss at the *worst* affected tooth in the dentition as a function of the patient's age (percent of bone loss at the site of greatest loss/patient age), *OR*
      b. The case phenotype, which is defined by whether the level of destruction is commensurate with the level of plaque biofilm in the patient's mouth.
   4. Although less precise and less exacting than direct evidence, indirect evidence provides a more practical means of assessing an individual patient's periodontitis grade (especially in cases where previous records such as past radiographs or past periodontal chartings are unavailable) and is a more feasible approach in quickly identifying highly susceptible or relatively resistant individuals.
   5. Note that in many cases longitudinal data is unavailable, which precludes using direct evidence to assign the periodontitis grade. In such cases, the clinician must rely on indirect evidence of disease progression when assigning a grading category.
   6. As in staging, when determining the grading category for an individual with periodontitis, the clinician should keep in mind that there may be individual risk factors that may shift the grade to a higher level. For instance, a periodontitis patient who smokes fewer than 10 cigarettes per day but exhibits no evidence of CAL over the last 5 years would be classified as Grade B.
   7. **Grade modifiers** are risk factors that may impact the rate of disease progression. Smoking and diabetes are systemic risk factors that have the greatest impact in disease progression. Grade as an indication of disease progression is summarized in **Table 6-2**.

Table 6-2 Periodontitis Grade

	Disease Progression/Characteristics	Grade Modifiers
Grade A	**Direct Evidence** No additional bone or attachment loss over past 5 years **Indirect Evidence** Percent of bone loss/patient age < 0.25 Low levels of tissue destruction not commensurate with the heavy biofilm deposits	Nonsmoker No history of diabetes
Grade B	**Direct Evidence** Evidence of less than 2 mm additional bone or attachment loss over a 5-year period **Indirect Evidence** Percent of bone loss to patient age is 0.25 to 1.0 Tissue destruction in line with the biofilm deposits	Smoking fewer than 10 cigarettes a day and/or An HbA1c of less than 7% in patients with diabetes
Grade C	**Direct Evidence** Evidence of 2 mm or more of bone or attachment loss over a 5-year period **Indirect Evidence** Percent of bone loss to patient age is > 1.0 Tissue destruction exceeds expectations given biofilm deposits	Smoking 10 or more cigarettes a day and/or An HbA1c of 7% or greater in patients with diabetes

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8. **Grade A: Slow Rate of Disease Progression**
   a. The primary criteria for a Grade A designation are:
      1. Direct evidence: no evidence of CAL or radiographic bone loss over a 5-year period.
      2. Indirect evidence: heavy biofilm deposits with low levels of tissue destruction *OR* percent of bone loss at the worst affected site/patient age is less than 0.25.
   b. The modifiers for Grade A are:
      1. Nonsmokers.
      2. Normoglycemic or nondiabetic patient.
9. **Grade B: Moderate Rate of Disease Progression**
   a. The primary criteria for a Grade B designation are:
      1. Direct evidence: less than 2 mm of CAL or radiographic bone loss over a 5-year period.
      2. Indirect evidence: tissue destruction is in line with expectations given the amount of biofilm deposits *OR* percent of bone loss at the worst affected site/patient age between 0.25 to 1.0.
   b. The grade modifiers for Grade B are:
      1. Smoking fewer than 10 cigarettes a day and/or
      2. An HbA1c of less than 7% in patients with diabetes. (The HbA1c level is a measurement of the average blood glucose level over the past 2 to 3 months. A normal HbA1C level is below 5.6%.)
10. **Grade C: Rapid Rate of Disease Progression**
    a. The primary criteria for a Grade C designation are:
       1. Evidence of 2 mm or more of attachment loss over a 5-year period.
       2. Tissue destruction exceeds expectations given the amount of biofilm deposits *OR* percent of bone loss at the worst affected site/patient age greater than 1.0.
    b. The grade modifiers for Grade C are:
       1. Smoking 10 or more cigarettes a day and/or
       2. An HbA1c of 7% or greater in patients with diabetes.

d. **Periodontal Diagnosis.** The periodontal diagnosis for an individual patient should include:
   1. Confirmation that patient is a periodontitis case based on detectable clinical attachment loss at two nonadjacent teeth.
   2. Identification of the form of periodontitis as necrotizing periodontitis, periodontitis as a manifestation of systemic disease, or periodontitis.
   3. Description of the presentation and aggressiveness of the disease by stage and grade.

Examples of Periodontitis Case Staging and Grading

Boxes 6-3 to **6-12** (**Figures 6-14** to **6-23**) provide examples of staging and grading for periodontitis cases.

Box 6-3 Case Example: Stage I, Grade A Periodontitis

Clinical Findings

- 45 years of age
- Interdental CAL of 1 to 2 mm
- No tooth loss due to periodontitis
- No probing depths over 4 mm
- Horizontal bone loss
- No additional bone loss over last 5 years
- Nonsmoker
- No history of diabetes

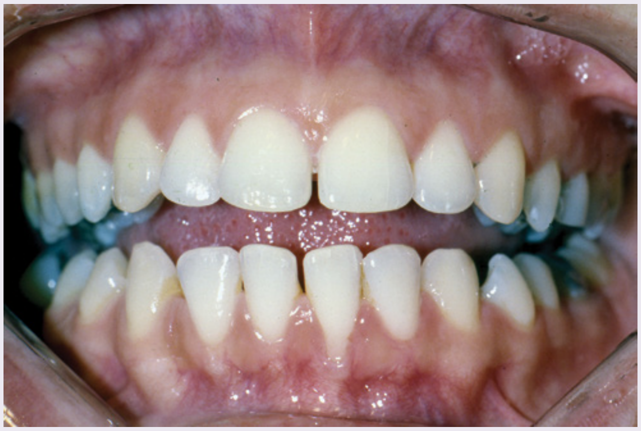

Figure 6-14 **Clinical Findings.** Clinical attachment readings indicate that this previously diagnosed periodontitis patient has not experienced any additional attachment loss over the past 5 years, indicating that a combination of good self-care and professional maintenance has stabilized the periodontium and re-established good periodontal health.

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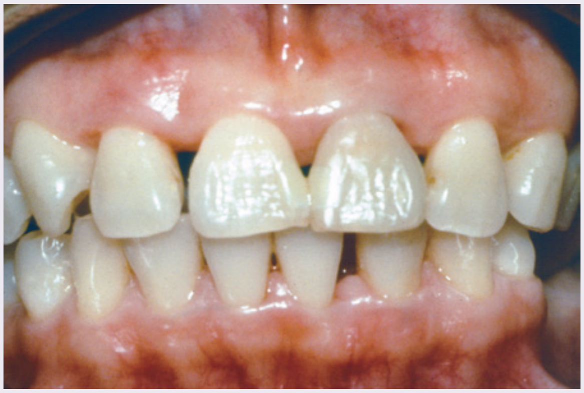
Box 6-4 Case Example: Stage III, Grade C Periodontitis

Clinical Findings

- 28 years of age
- Interdental CAL of 3 to 4 mm
- No tooth loss due to periodontitis
- Probing depths of 5 mm or less
- Mostly horizontal bone loss extending past the middle one-third of the root
- Evidence of 2 mm or more attachment loss over a 5-year period
- Tissue destruction exceeds expectations based on amount of biofilm present
- Smokes a pack of cigarettes a day (20 or more per day)
- Preserved masticatory function



A

Figure 6-15A **Clinical Findings.** This 28-year-old patient has experienced a rapid rate of loss of attachment over a 5-year period.

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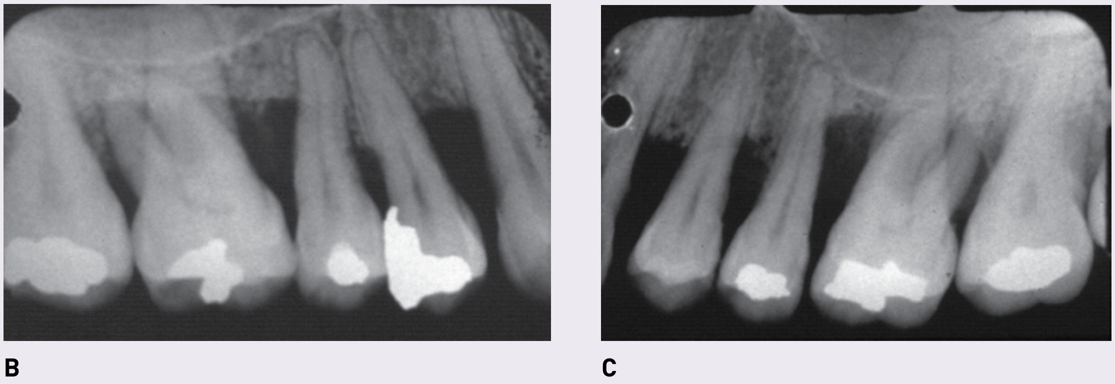

Figure 6-15B,C Radiographic Characteristics. The patient's radiographs reveal severe horizontal bone loss around most teeth.

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Box 6-5 Case Example: Stage III, Grade C Periodontitis

Clinical Findings

- 20 years of age
- Interdental CAL of 3 to 4 mm
- No tooth loss due to periodontitis
- Probing depths of 4 mm or less
- Radiographic bone loss extends to the coronal third of root (1/3) of the first molars
- Evidence of 2 mm or more attachment loss over a 5-year period
- Grade II furcation present on the mandibular molars
- Tissue destruction exceeds expectations based on amount of biofilm present
- Non-smoker, non-diabetic

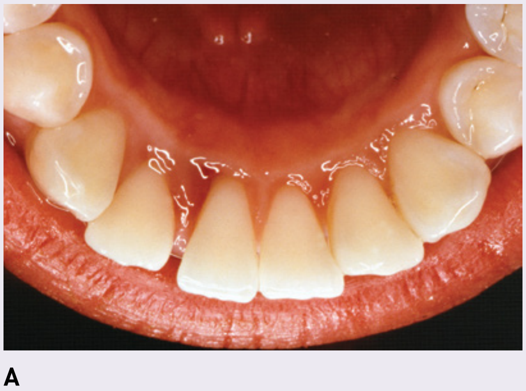

Figure 6-16A Clinical Findings. The photo shows a patient with Stage III, Grade C periodontitis. Note that there are not any supragingival calculus deposits evident; thus, the tissue destruction exceeds expectations.

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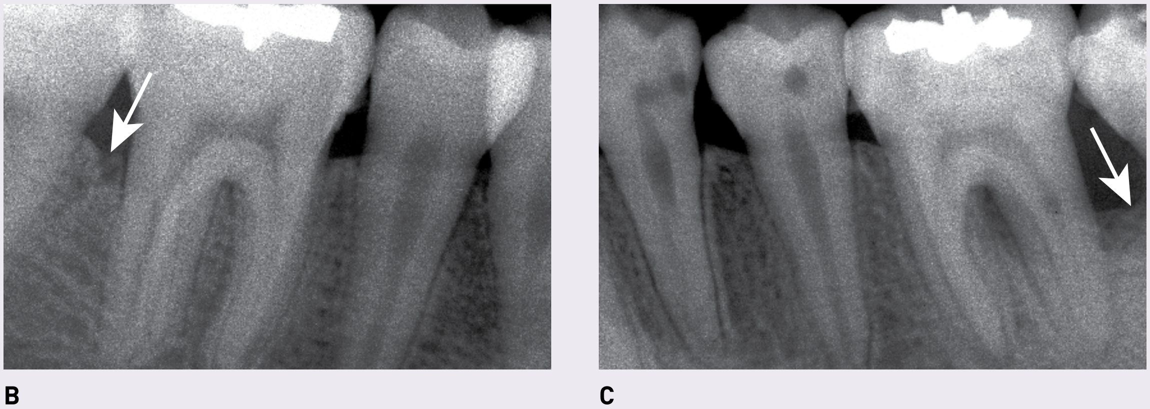

Figure 6-16B,C Radiographic Characteristics. The patient's radiographs reveal a pattern of bone loss on the first molars that is similar on both sides of the mandibular arch. Note the angular defects (arrows) on the distal surfaces of the molars. Also note radiographic evidence of furcation involvement on both molars.

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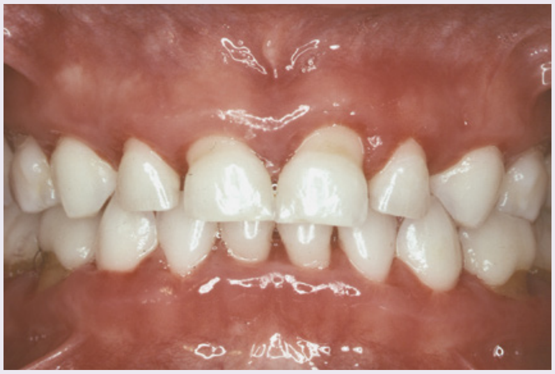
Box 6-6 Case Example: Stage III, Grade C Periodontitis

Clinical Findings

- 7 years of age
- Interdental CAL of 3 to 4 mm
- No tooth loss due to periodontitis
- Probing depths of 4 mm or less
- Tissue destruction exceeds expectations based on amount of biofilm present

Due to the absence of longitudinal radiographic data on this 5-year-old patient, using direct evidence of disease progression was not possible. Instead, the following indirect evidence of progression was used to assign a grade to this patient's periodontitis: (1) amount of periodontal destruction exceeded given biofilm deposits, (2) early onset of disease, and (3) rapid progression that is not characteristic of the patient's age.



A

Figure 6-17A Child with Stage III, Grade C Periodontitis. Although very rare, children may exhibit periodontitis.

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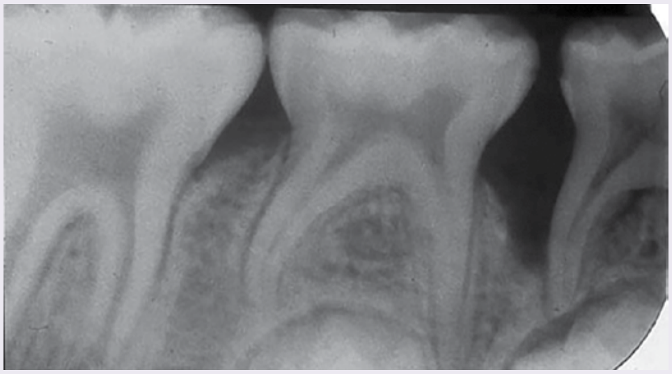

B

Figure 6-17B Radiographic Characteristics. Radiographic evidence of vertical bone loss in the patient shown in Figure 6-17A.

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Box 6-7 Case Example: Stage IV, Grade C Periodontitis

Clinical Findings

- 44 years of age
- Interdental CAL 5 mm or more
- Four teeth lost due to periodontitis
- Probing depths 6 mm or more
- Radiographic bone loss extends to the mid-third of root
- Vertical bone loss
- Evidence of 2 mm or more additional attachment loss over a 5-year period
- Smokes a pack of cigarettes a day
- Bite collapse with drifting of teeth and multiple teeth with Degree 2 mobility

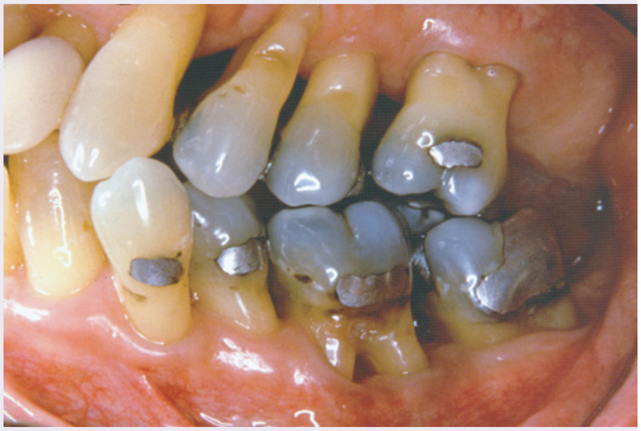

Figure 6-18 Patient with Stage IV, Grade C Periodontitis. An example of rapid disease progression despite good daily self-care by the patient.

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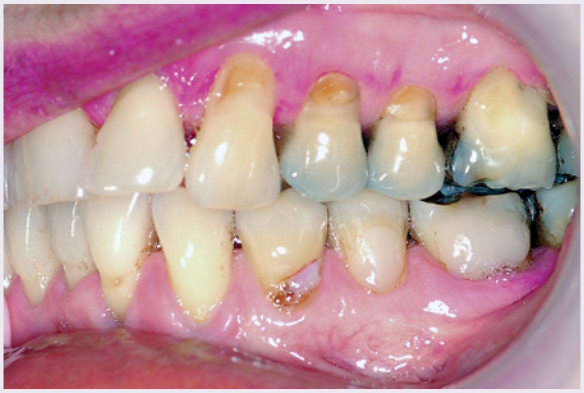
Box 6-8 Case Example: Stage II, Grade B Periodontitis

Clinical Findings

- 50 years of age
- Interdental CAL of 3 to 4 mm
- No tooth loss due to periodontitis
- Probing depths 5 mm or less
- Radiographic bone loss extends to the coronal third of root
- Mostly horizontal bone loss
- Evidence of less than 2 mm additional attachment loss over a 5-year period
- Nonsmoker
- No history of diabetes



A

Figure 6-19A **Clinical Findings.** The photo shows a patient with Stage II, Grade B periodontitis. At her maintenance (recall) visit, she exhibits excellent self-care.

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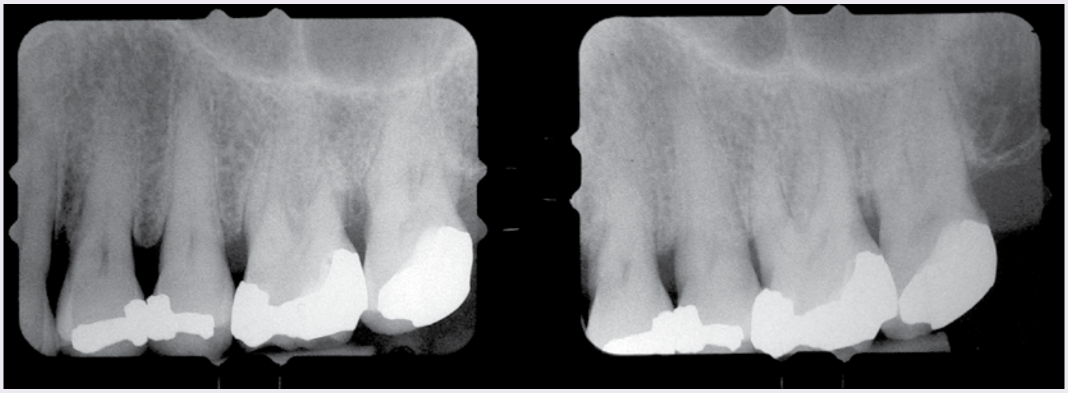

B

Figure 6-19B **Radiographic Characteristics.** The patient's radiographs reveal bone loss that extends to the coronal thirds of the roots. However, over the past 5 years, she experienced less than 2 mm additional clinical attachment loss.

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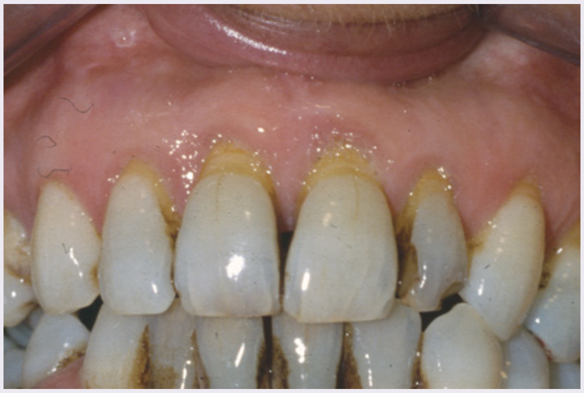
Box 6-9 Case Example: Stage III, Grade C Periodontitis

Clinical Findings

- 47 years of age
- Interdental CAL 5 mm or more
- Four teeth lost due to periodontitis
- Probing depths 6 mm or more
- Radiographic bone loss extends to the mid-third of root
- Some vertical bone loss
- Nonsmoker
- History of diabetes with a long-term HbA1c levels over 7%



A

Figure 6-20A Clinical Findings. The photo shows a patient with Stage III, Grade C periodontitis. The patient's HbA1c and the percent of bone loss as a function of the patient's age places her in the Grade C category.

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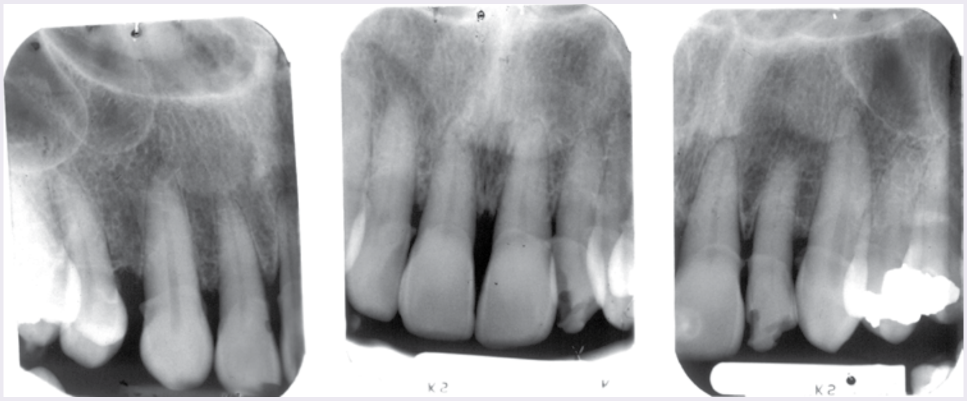

B

Figure 6-20B Radiographic Characteristics. The patient's radiographs reveal bone loss that extends to the mid-thirds of the roots.

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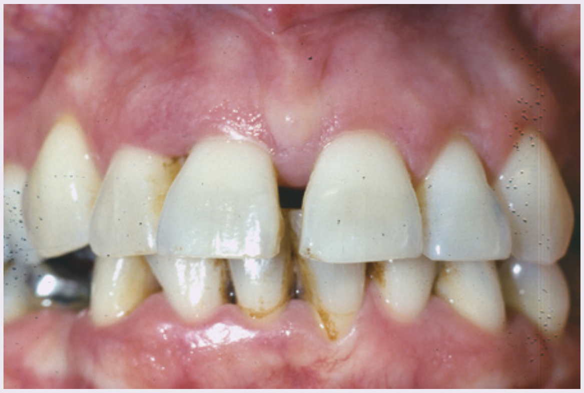
Box 6-10 Case Example: Stage IV, Grade C Periodontitis

Clinical Findings

- 53 years of age
- Interdental CAL 5 mm or more
- More than five teeth lost due to periodontitis
- Probing depths 6 mm or more
- Radiographic bone loss extends to the mid-third of root
- Vertical bone loss
- Nondiabetic
- Smokes more than 10 cigarettes a day



A

Figure 6-21A Clinical Findings. The photo shows a patient with Stage IV, Grade C periodontitis.

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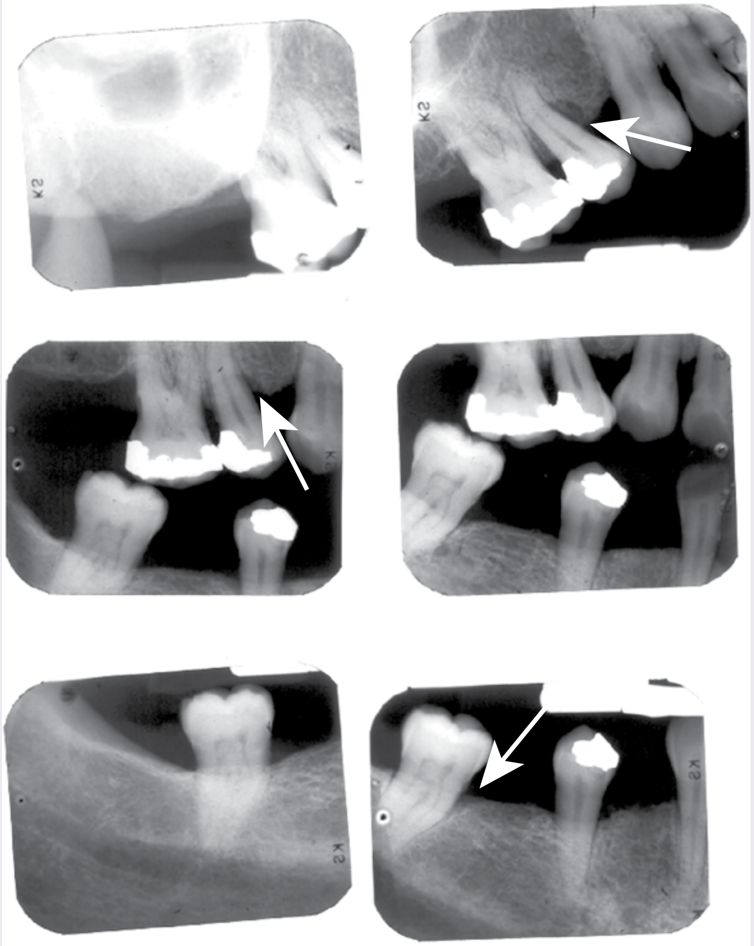

B

Figure 6-21B Radiographic Characteristics. The patient's radiographs reveal vertical bone loss (indicated by white arrows).

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Box 6-11 Case Example: Stage III, Grade C Periodontitis

Clinical Findings

- 40 years of age
- Interdental CAL of 2 to 13 mm
- No tooth loss due to periodontitis
- Probing depths of 2 to 13 mm
- Mostly horizontal bone loss; vertical bone loss extending to apical thirds of #31
- Evidence of 2 mm or more attachment loss over a 5-year period
- Percent of bone loss/patient's Age (based on most severely affected tooth #31) > 1.0.
- Nonsmoker, nondiabetic
- Preserved masticatory function, no mobility

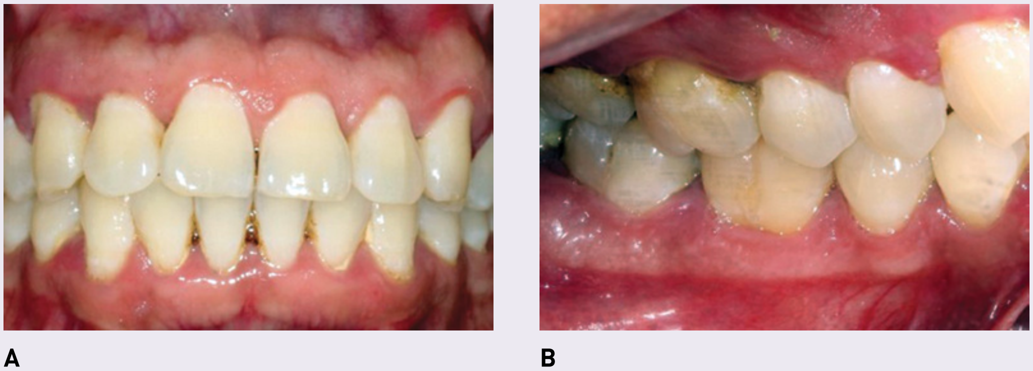

Figure 6-22A,B Clinical Findings. A and B show the front and right lateral view of a periodontitis patient with inflamed gingiva.

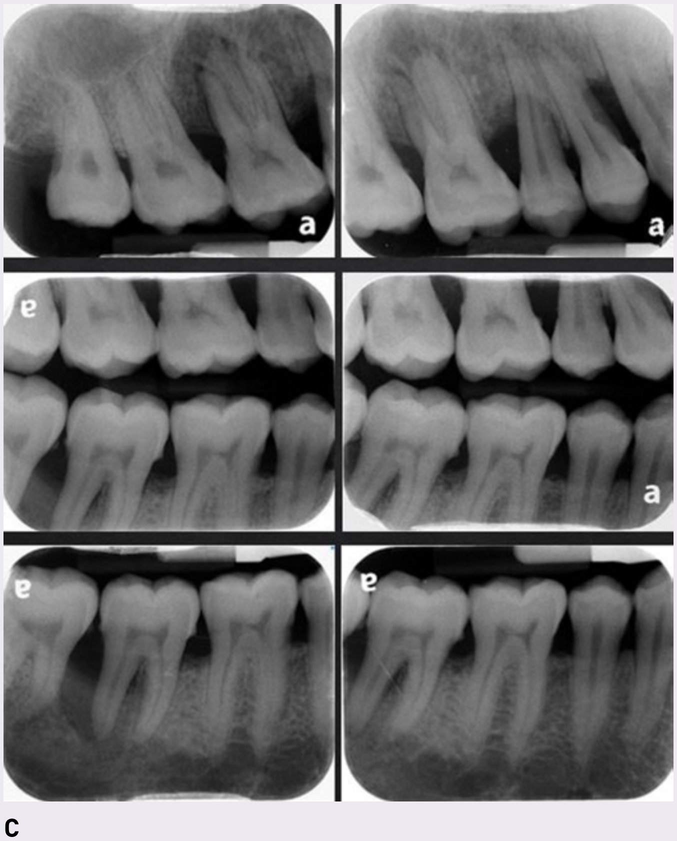

Figure 6-22C Radiographic Characteristics. Accompanying radiographs reveal severe horizontal bone loss around most teeth and vertical bone loss associated with tooth #31 extending to the apical one-third of the root.

Box 6-12 Case Example: Stage IV, Grade C Periodontitis

Clinical Findings

- 43 years of age
- Interdental CAL of 4 to 12 mm
- No tooth loss due to periodontitis
- Probing depths of 4 to 10 mm
- Mostly horizontal bone loss, extending to apical thirds of most teeth
- Evidence of 2 mm or more attachment loss over a 5-year period
- Percent of bone loss/patient age (based on most severely affected teeth, #23–#26) > 1.0
- Nonsmoker, nondiabetic
- Masticatory dysfunction (open bite), flaring, drifting, tooth mobility ≥ 2 on many teeth

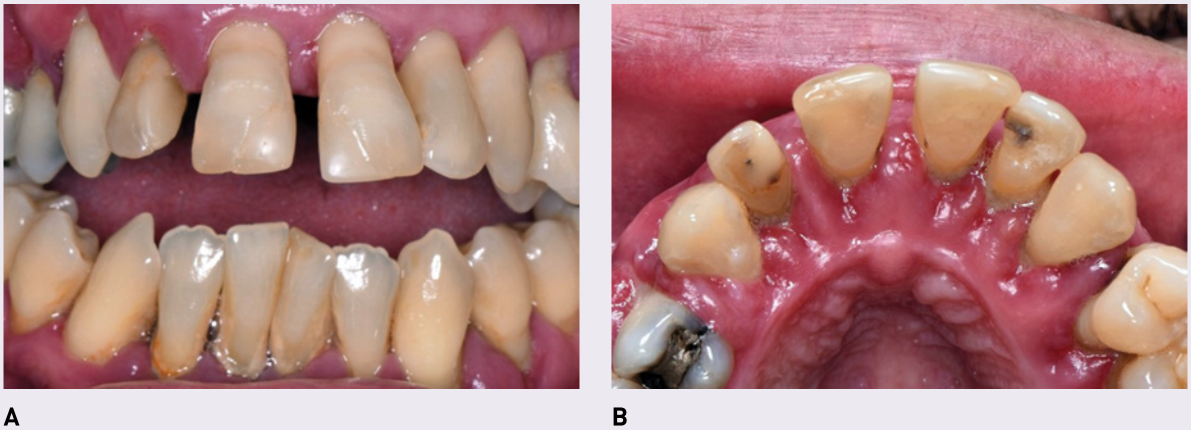

Figure 6-23A,B **Clinical Findings.** **A** and **B** show the front and occlusal views of a periodontitis patient depicting inflamed gingiva and flaring of the teeth.

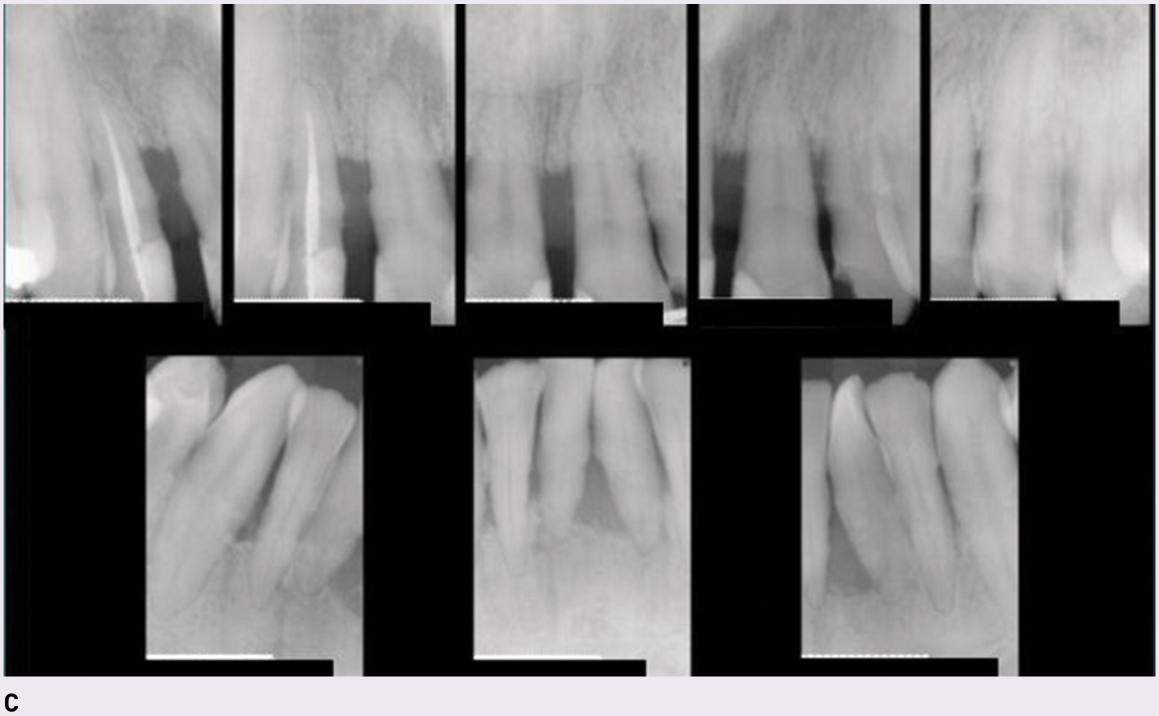

Figure 6-23C **Radiographic Characteristics.** Accompanying radiographs reveal severe horizontal bone loss around most teeth extending to the apical one-third of the roots.

Chapter Summary Statement

Periodontitis is a bacterial infection resulting in inflammation within the supporting tissues of the teeth, progressive destruction of the periodontal ligament, and loss of supporting alveolar bone.

- Periodontitis leads to irreversible loss of attachment and alveolar bone.
- Periodontitis may affect a single site of a tooth, several teeth, or the entire dentition. A patient can simultaneously have areas of health and areas with periodontitis.
- Untreated periodontitis usually is characterized by slow to moderate rates of disease progression and a favorable response to periodontal therapy. A limited number of periodontitis cases are characterized by rapid destruction of the attachment and rapid loss of supporting bone; these types of cases are difficult to manage with conventional nonsurgical periodontal therapy alone, and may warrant close collaboration between the dental hygienist, periodontist, and patient's physician to account for any undiagnosed medical condition(s) which may exacerbate the patient's periodontal condition.
- The desired outcomes of periodontal therapy are to stop the progression of the disease, to prevent further attachment loss, minimize the risk of tooth loss, and curtail any detrimental impact periodontitis may have on overall systemic health.

Section 3

Focus on Patients

Clinical Patient Care

CASE 1

A new patient has a diagnosis of Stage II, Grade B periodontitis. The patient tells you that it is hard for him to believe he has serious periodontal problems since he has never had any discomfort and has never even noticed any dental problems. How could you respond to the patient?

CASE 2

A patient who has recently moved to your city has an appointment with you regarding self-care instructions. The periodontal case diagnosis is Stage III, Grade B. In your discussion with the patient, you learn that the patient is upset because she has been treated for periodontitis twice during the past decade in other dental offices. She is upset because now she needs periodontal treatment again, and she states she is confused about how this might be possible. How could you respond to this patient's concerns?

CASE 3

Mr. B. is a 60-year-old male who presents to your office for a periodontal evaluation. From your clinical and radiographic assessment, you note that there is generalized clinical attachment loss of 3 to 4 mm, probing depths of 5 mm or less, profuse bleeding upon probing, and radiographic bone loss in the coronal third. The patient's records indicate evidence of less than 2 mm of additional attachment loss over a 5-year period. What would be your periodontal diagnosis? How would you explain to the patient the significance of the stage and grade of his periodontitis?

CASE 4

While reading a journal article you find a reference to a periodontal disease called chronic periodontitis. This is not a disease category in the currently accepted disease classification system. How does this terminology relate to the current 2017 disease classification system?

Ethical Dilemma 1

Mrs. W. is a new patient in your general dentistry practice—having changed from another dental practice in your city. Mrs. W. states that she "decided to change to your dental office because her friend Cynthia is so happy with the care she receives here."

After completing a comprehensive periodontal assessment and obtaining Mrs. W's records from the previous dental practice, the dentist's diagnosis is periodontitis Stage II, Grade B. Members of the dental team clearly communicate this finding and offer recommendations for a treatment care plan. After asking some questions, Mrs. W. indicates that she is grateful that everything has been so clearly explained and that she can clearly see the problems and how the recommended care plan will help her.

After accepting the recommended care plan, Mrs. W. has several additional questions for you. She states that she has seen a dental hygienist and dentist in the other practice every 6 months for years, never missing an appointment. Mrs. W. states that the dental team in the other practice never told her that she has any dental problems, and never suggested any treatment other than a 1-hour cleaning appointment once every 6 months. Mrs. W. states that she is upset that the other practice did not provide good care and wonders if she should sue them?

How can you respond to Mrs. W's concerns about the care she received in the other dental practice?

Ethical Dilemma 2

Your next patient, Josiah S., has recently relocated to your town, approximately 6 months ago, and is a new patient in your practice. He is a 45-year-old divorced male, who admits to smoking approximately 20 cigarettes per day and drinking a nightly alcoholic cocktail. He works in sales, and due to his hectic travel schedule, tells you that he has had to cancel his last few dental hygiene appointments.

Your intraoral exam reveals that Josiah presents with moderate calculus and plaque biofilm. His gingival tissues appear red and swollen, have blunted papillae, and bleed readily upon probing. You take a full-mouth series of radiographs that show generalized horizontal bone loss throughout his mouth. You begin to probe his mouth, to determine his pocket readings, when he sits up in the dental chair, and demands that you stop. He states that he has always refused periodontal probing, as he just "can't stand the pain" and the last dental office abided by his wishes. He refuses to let you continue "poking around his gums" and asks that you just proceed to cleaning his teeth, so he can be on time for his 11:00 a.m. business appointment.

1. What ethical principles are in conflict in this dilemma?
2. Do you have an ethical obligation to treat this patient?
3. What, if any, alternatives can you offer Josiah in terms of his treatment plan?

Ethical Dilemma 3

Your dental practice, which is within an urban city, has many long-standing patients. A number of the patients within the practice are on public assistance and struggle with money issues. Dr. Gordon is very generous and understanding about her patients' financial limitations, and often provides free dental services.

Your next patient of the morning is Jason S., a 14-year-old soccer player. His mother has made an emergency appointment, because he was hit in the mouth during a soccer practice today. His mother is concerned that Jason's front teeth may be lost as a result.

His mother reports that Jason had a thorough "cleaning" in another dental office 4 months ago. However, she brought Jason to your dental office today because it is closer to his school than the dental practice that they usually go to. Clinical examination reveals no trauma but some mobility, so you take radiographs. Clinically, Jason presents with no obvious signs or symptoms of disease, with lack of tissue inflammation and minimal amounts of plaque biofilm.

Jason's radiographs, however, show moderate vertical bone loss around the maxillary central incisors and mandibular central incisors. Bitewing radiographs also indicate moderate bone loss around the maxillary and mandibular first molars. Probe readings on his incisors and molars are in the 4 to 6 mm range. You are concerned that this amount of tissue destruction is unusual in a 14-year-old patient.

You present your findings to Dr. Gordon, who asks you to perform thorough periodontal instrumentation on Jason. You remind Dr. Gordon, that Jason had a dental hygiene appointment in another office 4 months ago and that his insurance only allows for periodontal instrumentation every 6 months. Dr. Gordon states that Jason is in dire need of this treatment and asks to you to instruct the office manager to delay submitting Jason's insurance claim until it is within the 6-month time frame.

1. Although you do not have all the information needed to classify Jason's periodontal condition, what do you suspect his case stage and grade might be? What are your biggest concerns about Jason's periodontium?
2. What ethical principles are in conflict in this dilemma?
3. What is the best way for you to handle this ethical dilemma?

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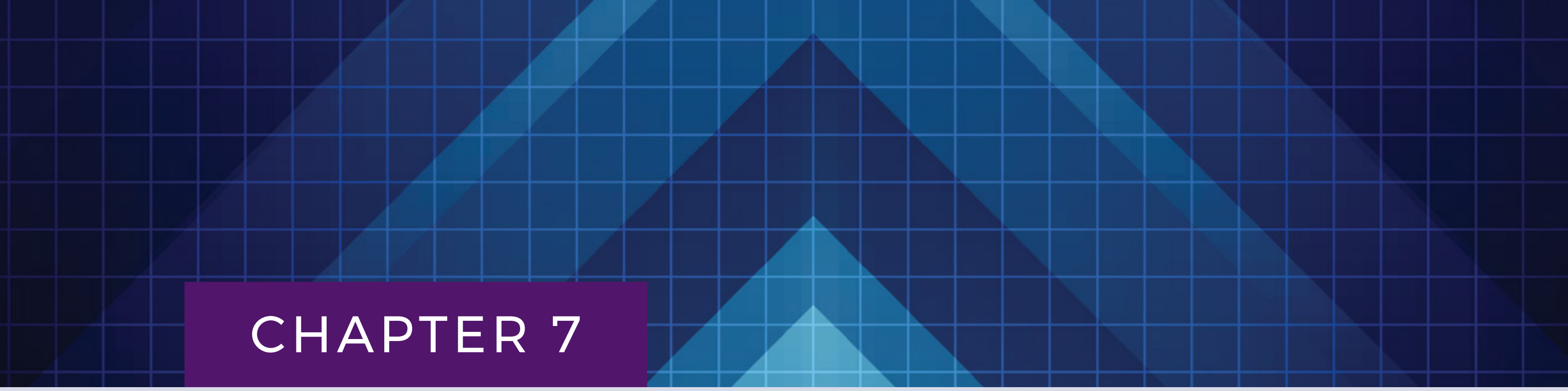

CHAPTER 7

Mucogingival Deformities and Conditions Around Teeth

- Section 1 Overview of Mucogingival Deformities
- Section 2 Normal Mucogingival Conditions and Periodontal Biotype
- Section 3 Recession of the Gingival Margin and Associated Risk Factors
- Section 4 Classification Systems for Recession of the Gingival Margin
- Section 5 Treatment Algorithm for Recession of the Gingival Margin
- Section 6 Focus on Patients
  - Clinical Patient Care

CLINICAL APPLICATION

With proper plaque control, an adequate amount of keratinized tissue at the tooth–soft tissue interface correlates to long-term success and stability. In contrast, keratinized tissue that is thin and friable is associated with a common periodontal condition known as a *mucogingival deformity*. A mucogingival deformity can occur in patients of any age group and may be present in individuals with or without periodontal disease. Among the mucogingival deformities, gingival recession is one of the most commonly encountered conditions in clinical practice and will be the focus of this chapter.

LEARNING OBJECTIVES

- Define the terms *mucogingival, normal mucogingival condition*, and *mucogingival deformity and condition*.
- Explain the significance of periodontal biotype and be able to describe the distinguishing features of thin-scalloped biotype, thick-scalloped biotype, and thick-flat biotype.
- Define the term *gingival recession* and explain what factors you need to take into consideration to determine if treatment is indicated.
- Explain the Miller and Cairo classification systems used to classify gingival recession.

KEY TERMS

Mucogingival
Mucogingival deformity and condition
Vestibular depth
Aberrant frenum
Normal mucogingival condition
Periodontal biotype
Recession of the gingival margin
Miller classification system
Cairo classification system

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Section 1

Overview of Mucogingival Deformities and Conditions

According to the American Academy of Periodontology's *Glossary of Periodontal Terms*, the term **mucogingival** is defined as "the portion of the oral mucosa that covers the alveolar process including the gingiva (keratinized tissue) and the adjacent alveolar mucosa."[1]

Thus, based on above definition, the term **mucogingival deformity and condition** encompasses any type of defect that is associated with a deviation from the normal anatomic/morphologic relationship between the gingiva and the alveolar mucosa.[2] All age groups are susceptible to mucogingival deformities and conditions. These conditions can also affect any tooth or implant and can occur in the presence or absence of periodontal disease. Conditions included in this vast category are (1) gingival/soft tissue recession, (2) lack of keratinized gingiva, (3) decreased vestibular depth, (4) aberrant frenum/muscle position, (5) gingival excess, and (6) abnormal color (**Box 7-1**).[2] When present, each of these conditions can potentially affect the form, function, and esthetics of the affected tooth and surrounding periodontium. Among the conditions listed, gingival/soft tissue recession is the most common type of mucogingival deformity/condition.

Box 7-1 Mucogingival Deformities and Conditions Around Teeth

1. Gingival/soft tissue recession
    a. Facial or lingual surfaces
    b. Interproximal (papillary)
    c. Severity of recession (Cairo RT1, 2, 3)
    d. Gingival thickness
    e. Gingival width
    f. Presence of carious and noncarious cervical lesions/cervical caries
    g. Patient esthetic concern
    h. Presence of hypersensitivity
2. Lack of keratinized gingiva
3. Decreased vestibular depth. **Vestibular depth** is the distance between the coronal border of the attached gingiva to the greatest concavity of the mucobuccal fold. An adequate vestibular depth is desired to enable placement of toothbrush and interdental cleaning aids, thus facilitating proper oral hygiene.
4. Aberrant frenum/muscle position. An **aberrant frenum** is an abnormal insertion of the frenal attachment too close to the marginal gingiva. In such a case, the aberrant frenum may limit movement of the cheek, lip, or tongue, or it may pull the gingival margin away from the tooth, thereby causing gingival recession.
5. Gingival excess
    a. Pseudopocket
    b. Inconsistent gingival margin
    c. Excessive gingival display
    d. Gingival enlargement
6. Abnormal color

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Section 2

Normal Mucogingival Condition and Periodontal Biotype

Not all individuals possess identical anatomy. Rather, there is some degree of anatomical variation that is acceptable and fits within the limits for what constitutes as normal. Likewise, variations in periodontal anatomy *which have no clinical significance and are considered within the spectrum of normal findings* are a common observation. Hence, a variation in mucogingival anatomy does not necessarily fall under the category of a periodontal abnormality.

1. **Normal Mucogingival Condition**
   a. **Normal mucogingival condition** is defined as the "absence of a disease state—for example the absence of *gingival recession, gingivitis, periodontitis*"[2] (**Figure 7-1**).
   b. Lack of keratinized tissue, decreased vestibular depth, and aberrant frenum fall under the category of normal mucogingival conditions because they are commonly observed in the absence of any underlying disease state. Instead, these types of mucogingival conditions typically occur because of congenital reasons or developmental reasons, not because of disease (**Figures 7-2** and **7-3**).
   c. If the patient consistently applies proper oral hygiene measures and adheres to a periodontal maintenance regimen, periodontal health can still be maintained even in the presence of a mucogingival deformity/condition.

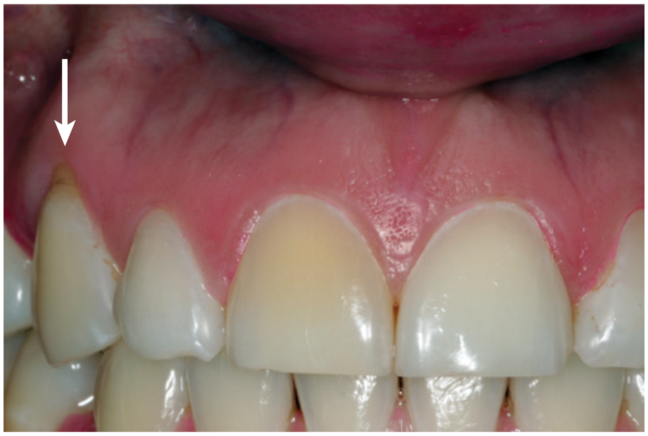

Figure 7-1 **Normal Mucogingival Condition.** Note the presence of a mucogingival deformity/condition (arrow) in healthy soft tissue. No signs of a disease state are visually apparent.

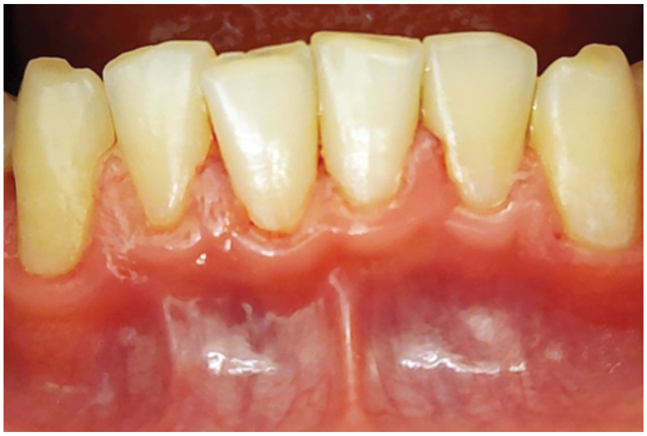

Figure 7-2 **Lack of Keratinized Tissue.** Note the minimal width of keratinized tissue (the band of soft tissue from the gingival margin to the mucogingival junction) in the absence of any appreciable disease state. In this case, the lack of keratinized tissue is not due to disease, but rather attributed to developmental reasons.

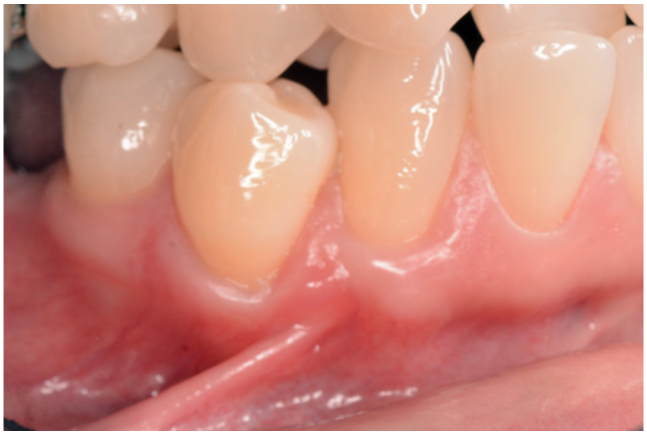

Figure 7-3 **Aberrant Frenum, Decreased Vestibular Depth, and Narrow Zone of Keratinized Tissue in Healthy Periodontium.** Note the proximity of the frenum to the gingival margin, the decreased vestibular depth, and the narrow zone of keratinized tissue in a healthy periodontium.

2. **Periodontal Biotype**
   a. The **periodontal biotype** (also referred to as periodontal phenotype) describes features of the periodontium, such as gingival thickness, gingival width, and the shape of the dental crown, that are influenced by both genetic and environmental factors. There is a strong association between certain types of periodontal biotypes and mucogingival deformities/conditions. Periodontal biotype refers to features of the soft tissues and the teeth they surround:
      1. Gingival biotype (gingival thickness and width of keratinized tissue)
      2. Bone morphology (thickness of the buccal bony plate)
      3. Tooth dimension of any given tooth.[2] There is a strong association between certain types of periodontal biotypes and mucogingival deformities/conditions.
      4. Three distinct biotype categories are outlined below (**Table 7-1**):
         a. Thin-scalloped gingival biotype corresponds to a tooth that exhibits the following features (**Figure 7-4**):
            1. A slender triangular-shaped crown.
            2. Thin, delicate, friable soft tissue with a relatively thin alveolar bone. The descriptive term "friable" refers to tissue that tears and bleeds more easily.
            3. Narrow zone of keratinized tissue.
            4. Accentuated scalloped (curved) gingival margin contour.
            5. A greater tendency for the onset and progression of a mucogingival deformity because the tissue is more fragile and friable.
         b. Thick-scalloped gingival biotype corresponds to a tooth that exhibits the following features (**Figure 7-5**):
            1. A slender triangular-shaped crown.
            2. Thick fibrotic gingiva.
            3. Narrow-to-moderate zone of keratinized tissue.
            4. Accentuated scalloped (curved) gingival margin contour.
            5. Despite having thick, fibrotic gingiva, the thick-scalloped biotype is prone to the development of a mucogingival deformity.
         c. Thick-flat gingival biotype corresponds to a tooth that exhibits the following features (**Figure 7-6**):
            1. A wide, square-shaped crown.
            2. Thick, dense, and fibrotic tissue.
            3. A wide band of keratinized tissue.
            4. Flat gingival margin contour.
            5. Compared to the thin-scalloped and thick-scalloped biotypes, the thick-flat biotype is more resilient and less susceptible to inflammation and trauma due to the above features. Therefore, the thick-flat biotype is more resistant to the onset and progression of a mucogingival deformity.

Table 7-1 Periodontal Biotypes (Phenotypes) and Their Characteristics

Tissue Biotype (Phenotype)	Crown Dimension	Periodontal Tissue Features	Potential Periodontal Ramifications
Thin-scalloped	Triangular-shaped crown (because of the high scalloped contour of the gingival margin)	Delicate, friable soft tissue Highly scalloped gingival margin contour Translucent appearance (tissue blanching upon probing) Narrow band of keratinized tissue Thin bony architecture	Low resilience to trauma and/or inflammation → greater susceptibility to the development and progression of mucogingival deformity
Thick-scalloped	Slender-shaped crown (because of the high scalloped contour of the gingival margin)	Thick, fibrotic soft tissue Highly scalloped gingival margin contour Narrow-to-moderate band of keratinized tissue Thin bony architecture	Low resilience to trauma and/or inflammation → greater susceptibility to the development and progression of mucogingival deformity

Tissue Biotype (Phenotype)	Crown Dimension	Periodontal Tissue Features	Potential Periodontal Ramifications
Thick-flat	Square-shaped crown (because of the flat contour of the gingival margin)	Thick, fibrotic tissue Low flat gingival margin contour Wide band of keratinized tissue Thick bony architecture	More resilience to trauma and/or inflammation → Lower susceptibility to the development and progression of mucogingival deformity More related to good periodontal health because tissue is dense and fibrotic with a large zone of keratinized tissue

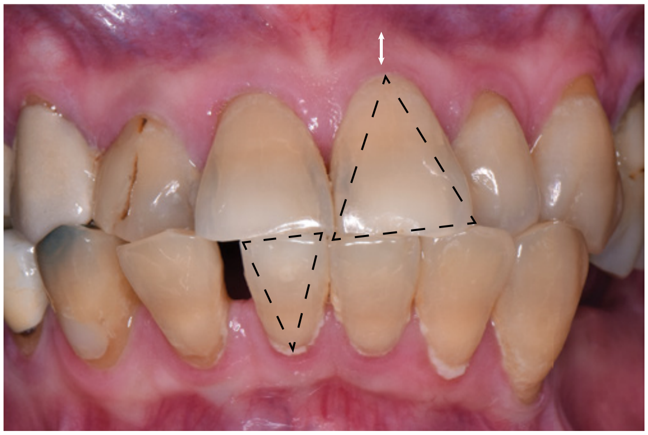

Figure 7-4 Thin-Scalloped Biotype. Note the accentuated scalloped margins and the narrow thin band of keratinized tissue in both the arches (double-ended arrows). Also note the triangular-shaped crowns.

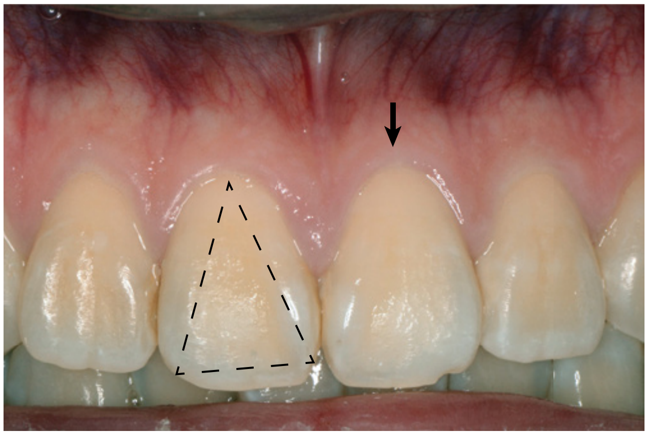

Figure 7-5 Thick-Scalloped Biotype. Note the slender triangular-shaped teeth, accentuated scalloped margins (arrow), and thick fibrotic gingiva.

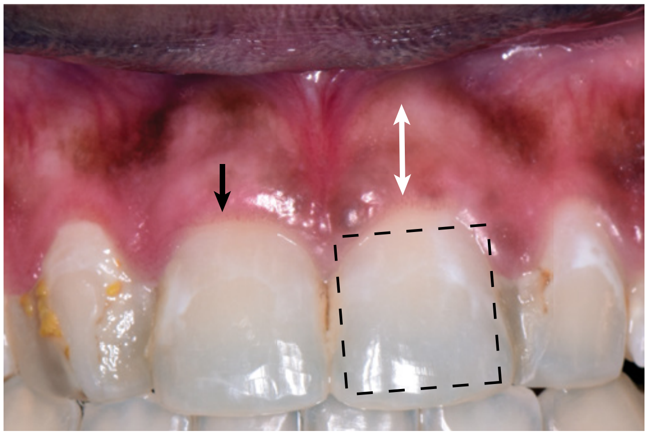

Figure 7-6 Thick-Flat Biotype. Note the thick gingival tissue and flatter contour of the gingival margin (black arrow). Also note the wide band of the keratinized tissue (white double-ended arrow) and the square-shape of the central incisor (black square)

b. In some cases, the periodontal biotype can vary even among different teeth within the *same* individual (**Figure 7-7**).

c. As part of a routine examination, the dental clinician should take the time to thoroughly assess and identify the periodontal biotype because certain biotypes confer a risk for the development and progression of mucogingival deformities.
   1. Assessing an individual patient's periodontal biotype is essential because it allows for timely identification of susceptible individuals and patient-specific risk factors that can be mitigated so that the development and progression of a mucogingival defect can be preempted.
   2. The dental clinician should collaborate with the patient to implement simple lifestyle interventions that can modify patient behavior away from self-injurious habits, such as aggressive toothbrushing, and consider various dental procedures that can minimize the risk of inflammation and/or mechanical injury to tissues that are susceptible to gingival recession.
   3. The role of all members of the dental team cannot be undervalued, as each member plays an essential role in preventing these types of conditions and promoting periodontal health.

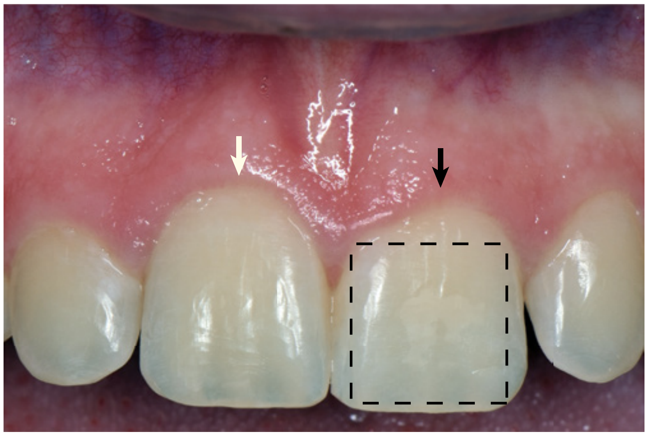

Figure 7-7 Different Periodontal Biotypes in the Same Individual. When comparing the biotype of the maxillary right central incisor to the maxillary left central incisor, one can see that the gingival thickness appears uniform. However, the contour of the gingival margin of the right central incisor appears more scalloped (white arrow), whereas the gingival margin contour of the left central incisor appears flatter (black arrow). Note the difference in the shape of the crowns: the left central incisor appears wider and more square-shaped, whereas the right central incisor appears slender.

Section 3

Recession of the Gingival Margin

1. **Recession of the gingival margin** (also known as gingival recession) is defined as the movement of the gingival margin to a point apical to the cementoenamel junction (CEJ).[3]
   a. Recession of the gingival margin is the most common mucogingival deformity. Approximately 88% of individuals older than 65 years and 50% of people 18–64 years have at least one or more sites with gingival recession.[4]
   b. Recession can affect any age group. However, its prevalence increases with age, and it can be observed in individuals with or without periodontal disease.
   c. It corresponds to attachment loss that exposes the root surface to the oral environment. Examples of recession of the gingiva are provided in **Figures 7-8** to **7-11**.
   d. In some cases, gingival recession may be esthetically unacceptable for many patients, may contribute to dentin hypersensitivity, or may be associated with carious or noncarious cervical lesions (loss of hard tissue near the CEJ due to mechanical trauma such as excessive toothbrushing).

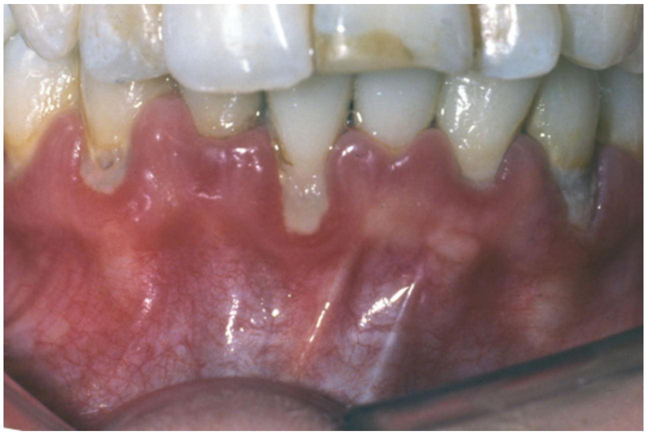

Figure 7-8 Recession of the Gingival Margin. Recession on the mandibular central incisors extending to the mucogingival junction.

Courtesy of Dr. Ralph Arnold, San Antonio, TX.

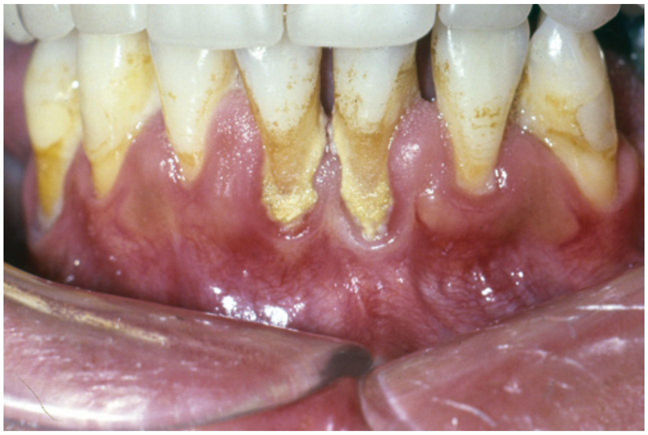

Figure 7-9 Recession of the Gingival Margin. Recession on the mandibular central incisors extending to the mucogingival junction.

Courtesy of Dr. Ralph Arnold, San Antonio, TX.

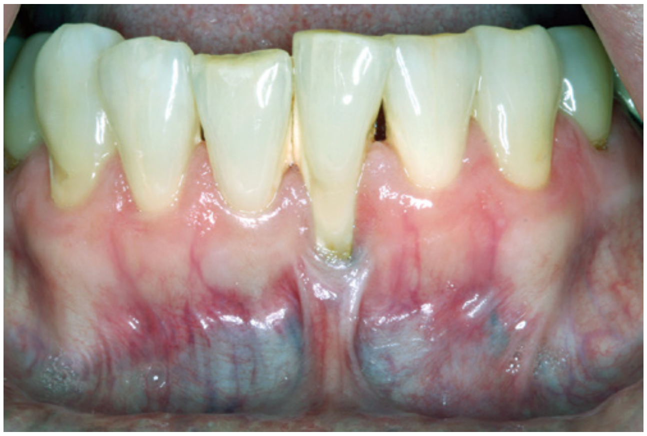

Figure 7-10 Frenum Attachment. Tension of the frenum may pull the gingiva away from the tooth and may be conducive to plaque biofilm accumulation and recession of the gingival margin.

Courtesy of Dr. Richard J. Foster, Educational Solutions and Design, Greensboro, NC.

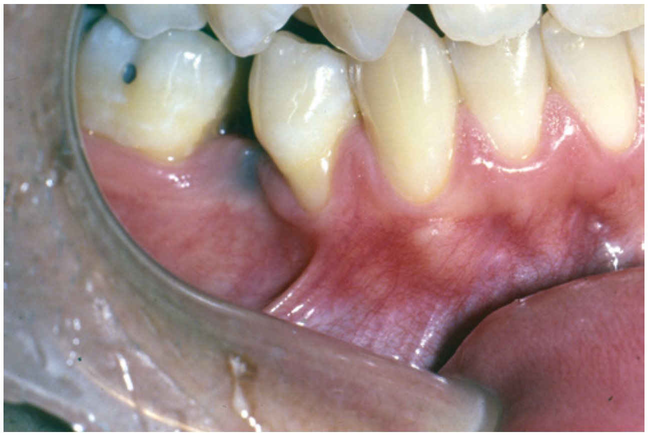

Figure 7-11 Frenum Attachment. Tension of a frenum may pull the gingiva away from the tooth and may be conducive to plaque biofilm accumulation and recession of the gingival margin.

Courtesy of Dr. Ralph Arnold, San Antonio, TX.

2. **Risk Factors for Development of Recession of the Gingival Margin**
   a. **Thin Tissue Biotype**
      1. Thin keratinized tissue, as described above, is one of the factors that can contribute to tissue recession in a patient.
      2. A commonly used method to clinically distinguish thick versus thin biotypes is by inserting a periodontal probe into the gingival sulcus, then evaluating for probe visibility. If the outline of the periodontal probe tip is visible through the overlying gingiva, then the gingival biotype is thin (**Figure 7-12**); if it cannot be seen through the gingival tissue, then it is considered to be thick (**Figure 7-13**).
      3. A gingival thickness less than or equal to 1 mm is considered to be thin; greater than 1 mm is considered to be thick.
   b. **Narrow Band (or Zone) of Keratinized Tissue**[3–5]
      1. The "band" of keratinized tissue is the zone of soft tissue that includes the free gingiva and the attached gingiva. It is measured from the gingival margin to the mucogingival junction.
      2. The presence of a broad (wide) band of keratinized tissue is favorable because it offers greater resistance to tissue recession. Thick-flat biotype corresponds to a broad zone of keratinized tissue. In contrast, a narrow band of keratinized tissue (seen in scalloped biotypes) is more likely to be susceptible to tissue recession.

3. The current consensus is that about 2 mm of keratinized tissue and about 1 mm of keratinized gingiva are desirable around teeth to maintain periodontal health, even though a minimum amount of keratinized tissue is not needed to prevent attachment loss when optimal biofilm control is present[6] (**Figure 7-14**).

c. **Reduced Thickness of the Alveolar Bone Due to Abnormal Tooth Position in the Dental Arch.** Figure 7-3 shows a malpositioned mandibular first premolar with a narrow band of keratinized tissue and a thinner than normal tissue biotype. All the above factors may contribute to gingival recession in the future.

d. **Mechanical trauma.** Mechanical trauma applied to the soft tissue through the actions of improper toothbrushing habits (e.g., aggressive brushing technique, use of a hard bristle toothbrush) may contribute to recession, especially in thin-scalloped or thick-scalloped biotypes.

e. **Orthodontics.** Orthodontic treatment that results in tooth movement in the buccal (or facial direction) may also contribute to gingival recession.

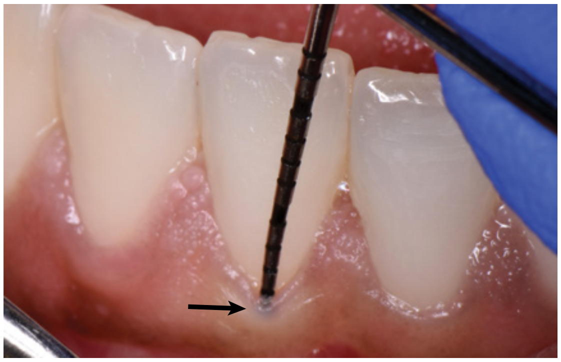

Figure 7-12 Thin Biotype. Observe the outline of the periodontal probe through the thin, transparent tissue biotype (black arrow).

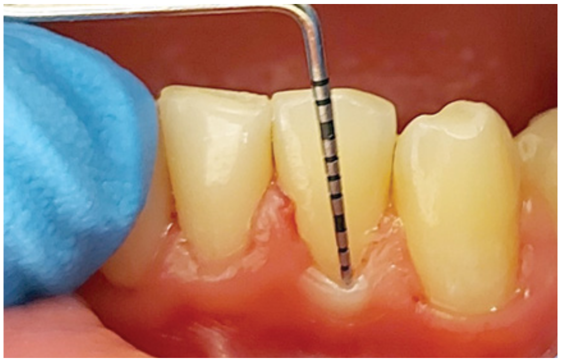

Figure 7-13 Thick Biotype. Observe that the outline of the periodontal probe is not visible through the thick tissue biotype.

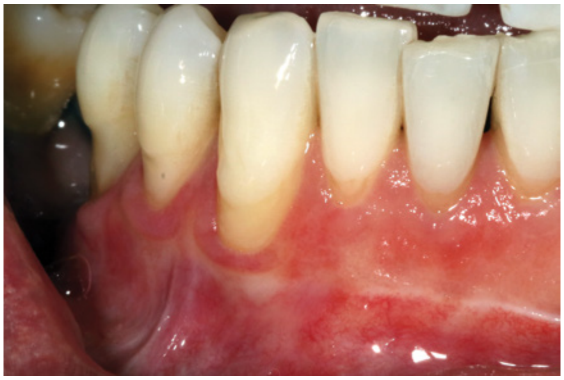

Figure 7-14 Narrow Band of Keratinized Tissue in a Healthy Periodontium. Note the narrow band of keratinized gingiva around the canine and the gingival recession. Also, note the healthy appearance of the soft tissue and the lack of plaque accumulation along the gingival margin. This patient displays evidence of proper plaque control measures.

Section 4

Classification Systems for Recession of the Gingival Margin

1. **The Miller Classification System for Recession of the Gingival Margin**[7]

a. For close to four decades, the **Miller classification system** was the most widely followed and most widely accepted gingival recession classification system. The Miller classification system is based on the level of gingival margin with respect to the mucogingival junction and the underlying alveolar bone.

  b. The Miller classification system consists of four recessions categories. Please refer to Chapter 19, Figure 19-3A–D for an illustration of the Miller classes.
    1. **Class I: Marginal tissue recession that does not extend to the mucogingival junction (MGJ).** There is no periodontal loss (bone or soft tissue) in the interdental area, and 100% root coverage can be anticipated.
    2. **Class II: Marginal tissue recession that extends to or beyond the MGJ with no periodontal loss (bone or soft tissue) in the interdental area**. 100% root coverage can be anticipated.
    3. **Class III: Marginal tissue recession that extends to or beyond the MGJ with interdental bone or soft tissue loss and/or malpositioning of the teeth**. Partial root coverage can be anticipated due to the pre-existing interndental tissue loss.
    4. **Class IV: Marginal tissue recession that extends beyond the MGJ with severe loss of interdental bone to the level corresponding to the most apical extent of the marginal tissue recession.** The bone or soft tissue loss in the interdental area and/or malpositioning of teeth is so severe that root coverage cannot be anticipated.

2. **The Cairo Classification System for Recession of the Gingival Margin**[8]
  a. Despite its widespread use in dentistry, the Miller classification system has recently been subject to criticism for the following limitations[9]:
    1. From a clinical standpoint, it is sometimes difficult to identify the exact location of the apical extent of the recession defect with respect to the mucogingival junction. This makes it difficult to clinically differentiate a Miller Class I from a Miller Class II.
    2. The Miller classification does not clearly define the amount of interproximal soft/hard tissue loss needed to differentiate a Miller class III from a Miller IV.
    3. The reliability of the Miller classification system is currently unavailable and has never been tested in a clinical setting.
  b. Recently, a new classification system—the **Cairo classification system**—for gingival recession has been proposed that is based on the clinical attachment level measurements at both buccal and interproximal sites.[8]
    1. Compared to the Miller classification system, the Cairo classification system uses an objective identifiable criterion—namely, clinical attachment level—to classify the extent and severity of the soft tissue recession.
    2. Furthermore, because the Cairo classification system uses an objective identifiable criterion that can be reliably measured, it is a more reliable gingival recession classification system that can be used in clinical practice.
  c. The Cairo classification system classifies gingival recession into three types. These types are illustrated in **Table 7-2**, **Figures 7-15** to **7-17**.
    1. **Recession Type 1 (RT1): Gingival recession with no loss of interproximal attachment.** Interproximal CEJ is clinically not detectable at both mesial and distal aspects of the tooth. RT1 recession defects represent defects that are most likely associated with traumatic toothbrushing in healthy periodontal tissues.
    2. **Recession Type 2 (RT2): Gingival recession associated with loss of interproximal attachment.** The amount of interproximal attachment loss (measured from the interproximal CEJ to the depth of the interproximal sulcus/pocket) is *less than or equal* to the buccal attachment loss (measured from the buccal CEJ to the apical end of the buccal sulcus/pocket). RT2 represents a soft tissue defect that is most likely associated with periodontitis-induced horizontal bone loss.
    3. **Recession Type 3 (RT3): Gingival recession associated with loss of interproximal attachment.** The amount of interproximal attachment loss (measured from the interproximal CEJ to the apical end of the sulcus/pocket) is greater than the buccal attachment loss (measured from the buccal CEJ to the apical end of the buccal sulcus/pocket). Like RT2 recession defects, RT3 represents soft tissue recession defect that is most likely associated with periodontitis, albeit with much more severe interproximal attachment loss. In most cases, RT3 recession defects are associated with an interproximal infrabony defect.

Table 7-2 The Cairo Classification of Recession of the Gingival Margin

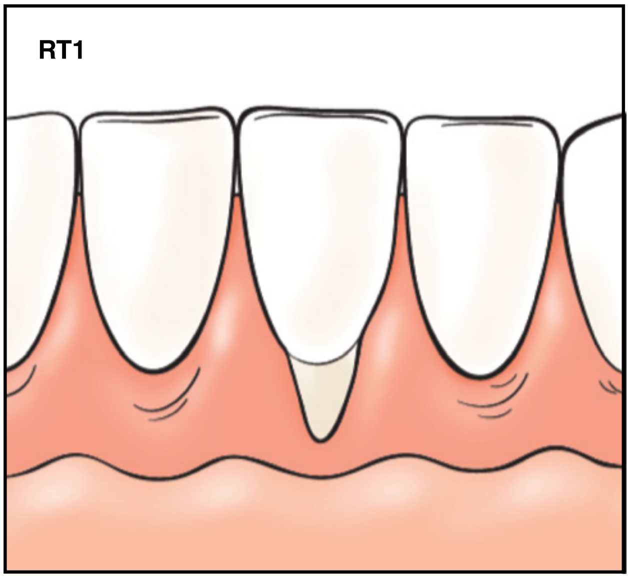

Figure 7-15 Recession Type 1 (RT1). Recession of the gingival margin on the facial aspect of the left central incisor:

- The level of clinical attachment loss on the facial aspect is 3 mm.
- There is no detectable loss of interproximal attachment.

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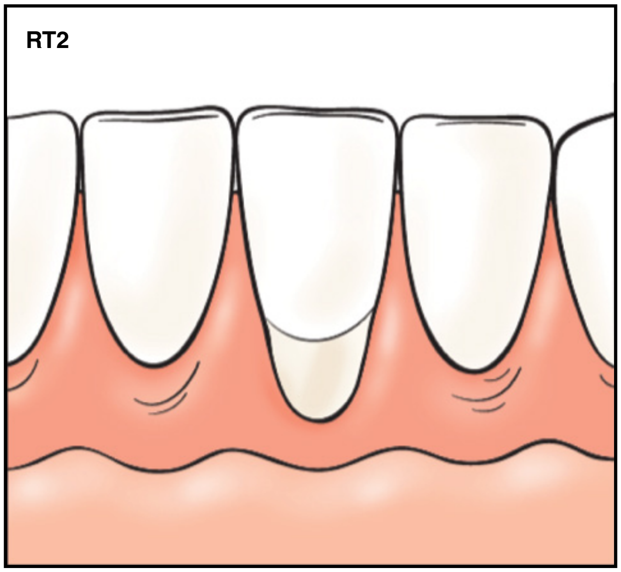

Figure 7-16 Recession Type 2 (RT2). Recession of the gingival margin on the facial aspect of the left central incisor:

- The level of clinical attachment loss on the facial aspect is 4 mm.
- Interproximal attachment loss of 3 mm.

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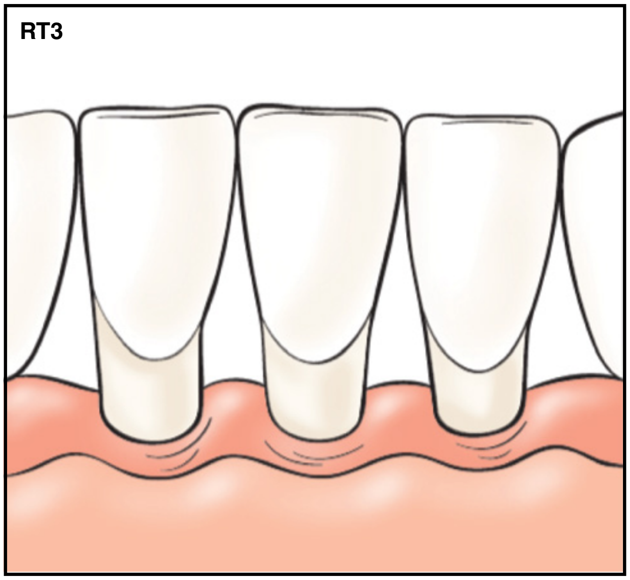

Figure 7-17 Recession Type 3 (RT3). Recession of the gingival margin on the facial aspect of the left central incisor:

- The level of clinical attachment loss on the facial aspect is 6 mm.
- Interproximal attachment loss of 8 mm.

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Section 5

Treatment Considerations for Gingival Recession Defects

1. When managing a gingival recession defect, the dental clinician must carefully assess the severity and extent of the symptoms, tissue biotype, and individualized patient-specific factors (e.g., caries risk status, periodontitis-risk status, and esthetic concerns), and then determine, on a case-by-case basis, whether monitoring with no treatment is sufficient or whether restorative/surgical periodontal intervention is necessary.
2. Because thin gingival biotype tends to remain less stable over time, some clinicians may intervene surgically (soft tissue augmentation procedures) to increase both the gingival thickness and the zone of keratinized tissue. Some clinicians may also elect to surgically intervene if the gingival recession is associated with esthetic impairment, dentin hypersensitivity, increased root caries activity, and/or severe plaque-induced gingival inflammation that jeopardizes the health of the tooth (**Figure 7-18**).
3. However, if the progression of gingival recession is arrested early on and the gingiva on the affected tooth appears stable and clinically healthy, then surgical intervention may not be required, *especially if the patient is able to exercise proper plaque control measures care and limit further mechanical trauma to the soft tissue* (**Figure 7-19**).
    a. It is therefore important to emphasize that surgical therapy is not warranted based solely on the presence of thin gingival tissue, gingival recession, probing depths extending beyond the mucogingival junction, and/or the absence or reduction of keratinized tissue.
    b. Furthermore, if the progression of recession is halted, the evidence suggests that gingival recession, in and of itself, does not significantly impact the long-term survival of the affected tooth provided that the patient adheres to proper plaque control measures.

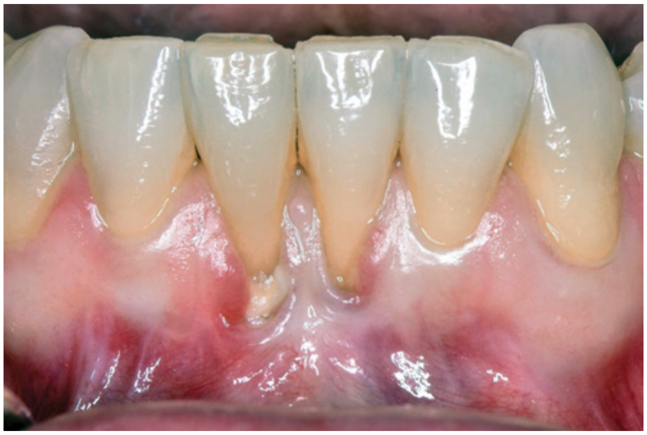

Figure 7-18 Recession of the Gingival Margin on a Diseased Periodontium. Recession of the gingival margin in the presence of persistent gingival inflammation. Because the gingival recession occurs in the presence of disease pathosis, surgical intervention is indicated.

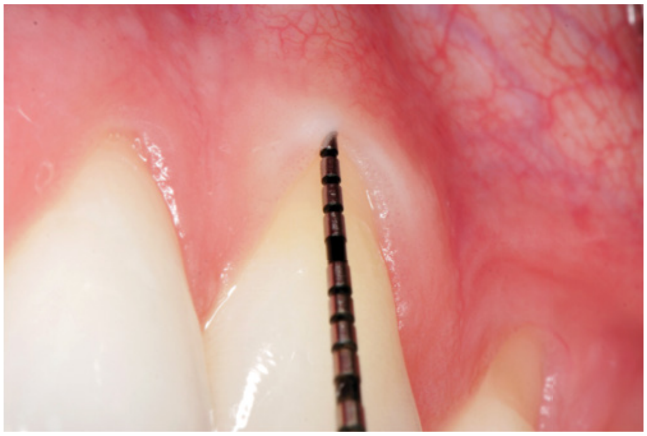

Figure 7-19 Gingival Recession on a Healthy Periodontium. Although the canine exhibits gingival recession and a narrow band of keratinized tissue at the mid-facial aspect, inflammation is not apparent (note the pocket depth is 1 mm with no bleeding upon probing). Because the patient presents with signs of periodontal health, good plaque control, and an absence of dentinal hypersensitivity, surgical intervention is not warranted. Rather, periodic monitoring of the gingival recession is recommended.

Chapter Summary Statement

This chapter focuses on a periodontal condition known as *mucogingival deformities and conditions* along with the accompanying clinical characteristics and associated risk factors. In and of itself, a mucogingival deformity/condition is not considered pathologic, but its presence may cause esthetic, psychological, and/or functional deficiencies. Early recognition of a patient's periodontal biotype is critical in preempting the onset and progression of a mucogingival deformity. It is also the first step for an effective management and prevention program. However, when present, the management of a mucogingival deformity can be complex and should be approached in an individualized manner that takes into consideration the specific features of a patient's periodontal tissue biotype, severity and extent of symptoms, and patient-specific factors. Treatment approaches should be tailored based on individual patient factors and on personalized therapy.

Section 6

Focus on Patients

Clinical Patient Care

CASE 1

A new patient presents for an examination. **Figure 7-20** shows the periodontal tissue that is being assessed. Describe the anatomical/morphologic features of the gingiva and the dimensions of the teeth. Assuming that the periodontal probe tip is not visible when placed in the sulcus, which type of periodontal biotype would this be classified as? Additionally, what is the likelihood of gingival recession occurring?

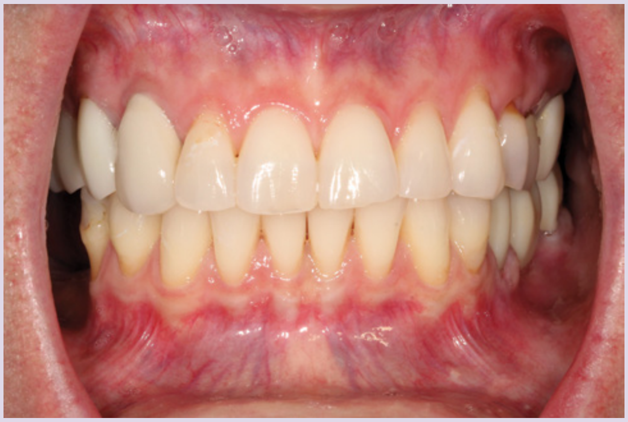

Figure 7-20 Patient Case 1

CASE 2

A new patient presents for an examination. **Figure 7-21** shows the periodontal tissue that is being assessed. Based on the presentation, how wide is the keratinized tissue? Assuming that the periodontal probe tip is not visible when placed in the sulcus, which type of periodontal biotype would this be classified as?

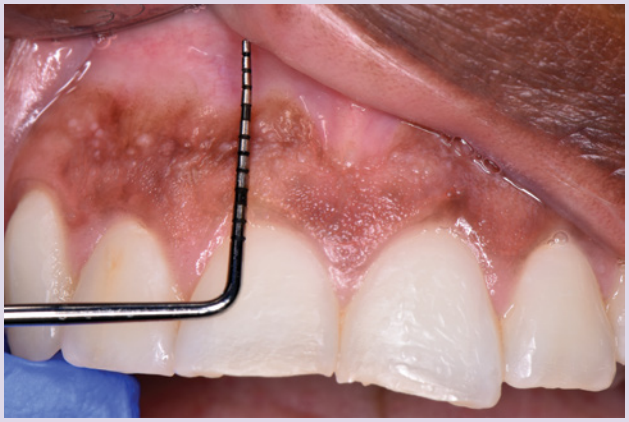

Figure 7-21 Patient Case 2

CASE 3

A patient presents for periodontal evaluation with the chief complaint "My gums hurt!" **Figure 7-22** shows the area related to the chief complaint. What steps would you take to perform a thorough periodontal evaluation? How would you classify this patient's periodontal biotype? With respect to treatment considerations, how would you approach this case?

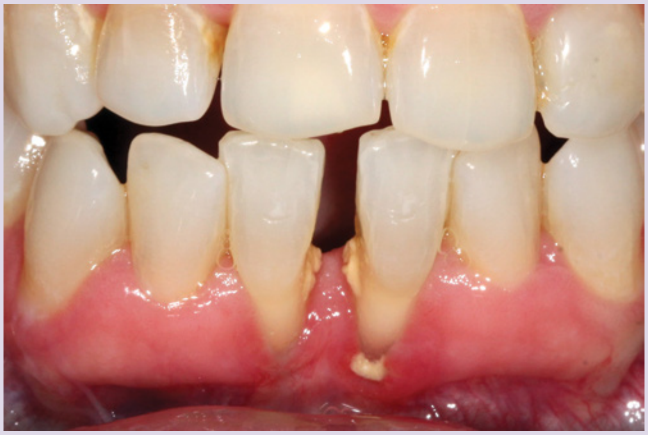

Figure 7-22 Patient Case 3

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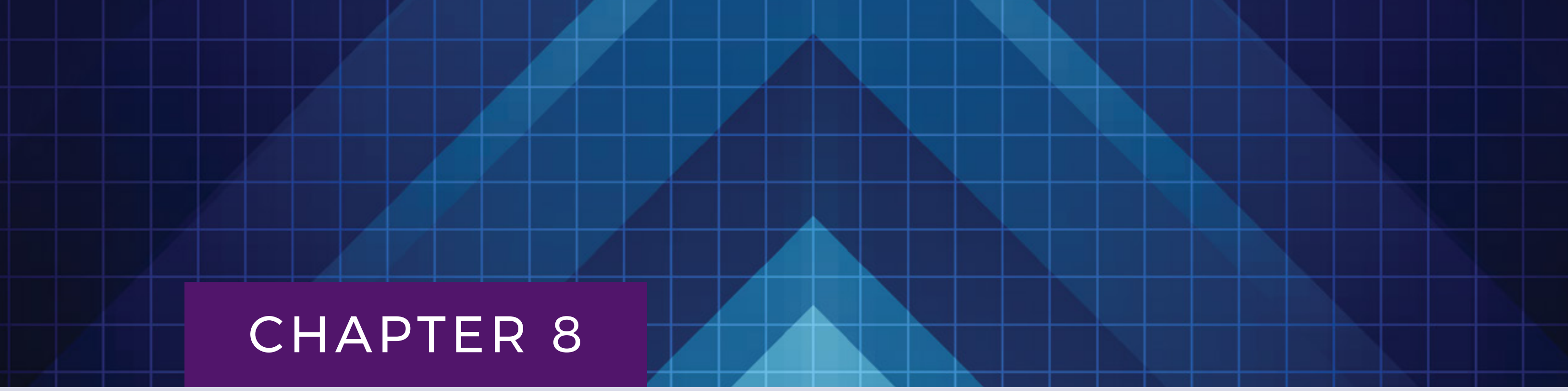

CHAPTER 8

Peri-Implant Health and Diseases

- Section 1 Anatomy of the Dental Implant and Surrounding Peri-Implant Tissues
- Section 2 Peri-Implant Health, Diseases, and Conditions
- Section 3 Recognition of Peri-Implant Diseases
- Section 4 Clinical and Radiographic Monitoring of Peri-Implant Health and Diseases
- Section 5 Guidelines for Maintenance of Patients with Dental Implants
- Section 6 Focus on Patients
  - Clinical Patient Care

CLINICAL APPLICATION

With a long-term survival rate of close to 95%, dental implants have earned the reputation of being the treatment option of first choice to replace missing teeth. In fact, according to the American Academy of Implant Dentistry, more than 3 million Americans have dental implants. The popularity of dental implants appears boundless, with estimates projecting the number of patients receiving implants to grow by 500,000 per year.[1] However, although dental implants are enjoying growing popularity, they are not without risks. At any time during the lifespan of the implant, biological and/or mechanical complications could arise that could jeopardize the survival of the dental implant. Consequently, dental health care providers, especially dental hygienists, will inevitably need to be able to distinguish peri-implant health from peri-implant diseases, such as peri-implant mucositis and peri-implantitis. Further, practitioners will need to know how to recognize and manage implant-related complications, such as peri-implantitis and biomechanical overloading of the implant. This chapter focuses on several fundamental aspects that encompass (1) the anatomical components of the dental implant; (2) the differences between the periodontium surrounding a natural tooth and the peri-implant tissues surrounding an implant; (3) the classification of peri-implant diseases and conditions developed by the 2017 AAP/EFP World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions; and (4) guidelines for the monitoring and maintenance of peri-implant tissues.

LEARNING OBJECTIVES

- Describe the components of a conventional dental implant and restoration.
- Compare and contrast the periodontium of a natural tooth versus the peri-implant tissues that surround a dental implant.
- Define the terms *peri-implant health*, *peri-implant mucositis*, and *peri-implantitis* and distinguish the key differences among them.
- Define the terms *osseointegration*, *biomechanical forces*, and *biomechanical overload* as they apply to dental implants.
- Describe an appropriate maintenance interval for a patient with dental implants.
- In the clinical setting, select appropriate self-care aids for a patient with dental implants.

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KEY TERMS

Dental implant
Implant body
Implant abutment
Biocompatible
Peri-implant tissues
Perimucosal seal
Osseointegration
Peri-implant health
Peri-implant mucositis
Peri-implantitis
Biomechanical forces
Biomechanical overload

Section 1

Anatomy of the Dental Implant and Surrounding Peri-Implant Tissues

It is important to review the individual components that make up the dental implant and the basic anatomy of the surrounding peri-implant hard and soft tissues. A **dental implant** is a nonbiologic (artificial) device surgically inserted into the jawbone to replace a missing tooth or provide support for a multiunit prosthesis, such as an implant-supported bridge or implant-supported dentures. Decades of *in vivo* research conducted on human subjects have validated the use of implants as a viable option to replace missing teeth in partially or fully edentulous patients.[2] Even today, research is ongoing to discover innovative ways to accelerate the rate of implant osseointegration and better preserve the ever-so-critical peri-implant bone throughout the lifetime of the implant.

In contemporary dental practice, the dental hygienist plays an important role in patient education and professional maintenance of the dental implant. Understanding the basic concepts of implantology and the anatomy of the peri-implant tissues are therefore prerequisites to distinguishing peri-implant health from peri-implant diseases and are critical to understanding the significance of effective maintenance for patients with dental implants.

1. **The Dental Implant System.** The dental implant system replaces individual teeth or multiple teeth by supporting a fixed bridge or removable denture (**Figures 8-1** to **8-3**).

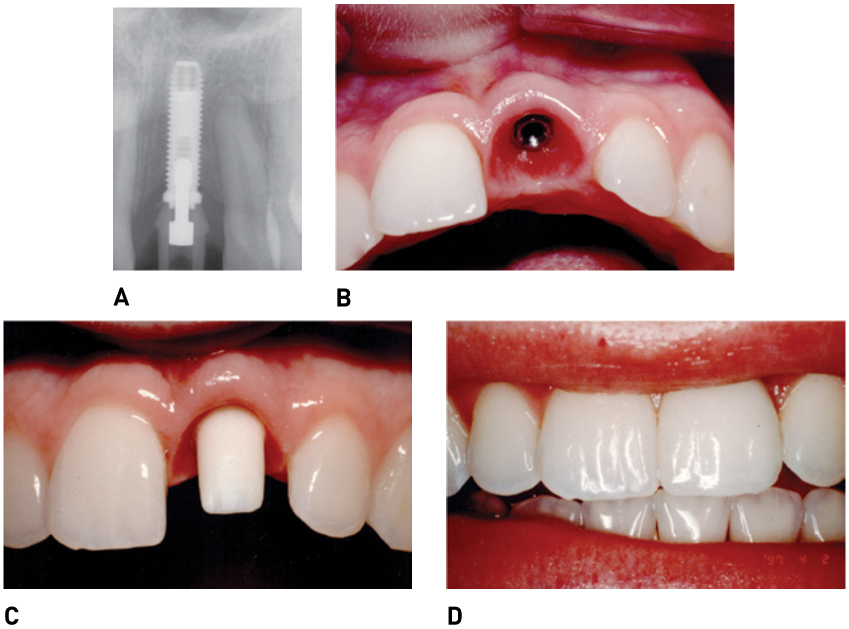

Figure 8-1 Dental Implant Replacing a Missing Maxillary Incisor. A. Dental radiograph showing the implant body surrounded by healthy bone. **B.** Clinical photograph showing the abutment post that is fastened to the underlying implant body. The abutment emerges from the peri-implant soft tissue. **C.** Clinical photograph of the abutment secured to the implant body. Note the healthy color and contour of the peri-implant soft tissue; its appearance is indistinguishable from the healthy periodontal tissue surrounding the neighboring natural teeth. **D.** Clinical photograph of the final implant-supported crown. Note the natural appearance of the implant-supported crown that appears very similar to the adjacent natural central incisor.

Courtesy of Rodger A. Lawton, DMD, FACP, Olympia, WA.

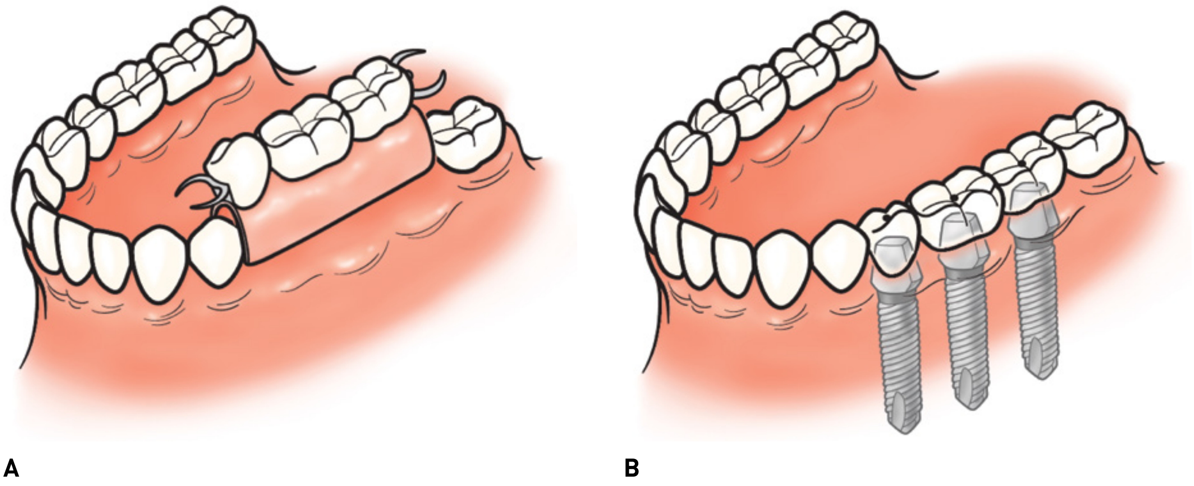

Figure 8-2 **Replacement of Missing Teeth. A.** Missing teeth replaced by a conventional removable partial denture. **B.** Missing teeth replaced by three individual dental implants.

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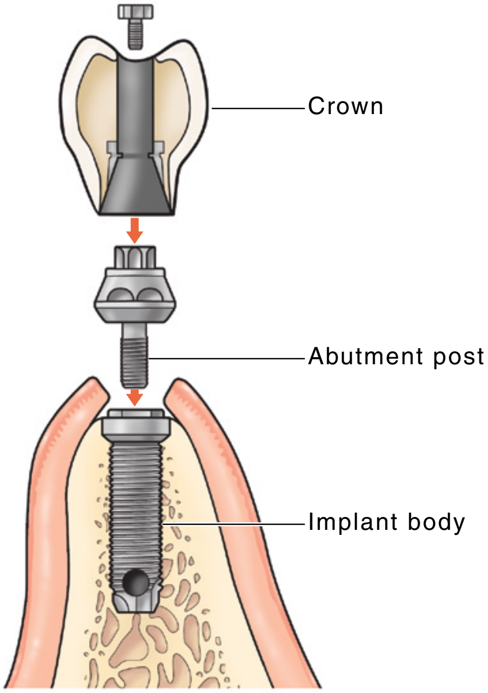

Figure 8-3 **The Dental Implant System.** The components of a dental implant system are the implant body, the abutment, and the crown (or other type of prosthesis, such as an implant-supported bridge or implant overdenture). The implant body is surgically placed in vital alveolar bone while the abutment post is fastened to the implant body and passes through the gingival tissues. The abutment serves as the connecting link between the implant body and the implant prosthesis. A crown or other type of prosthesis is connected to the abutment either by a screw or by dental cement.

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a. **Components of an Implant System.** The components of a dental implant system are (1) the implant body (also known as the implant fixture), (2) the abutment, and (3) the prosthetic crown or prosthesis.

1. **Implant Body**
   a. An **implant body** is the portion of the implant system that is surgically placed into the living, vital alveolar bone. **Figure 8-4** diagrams the steps in the surgical placement of a dental implant body. Please refer to Chapter 30, "Periodontal Surgical Concepts for the Dental Hygienist," for a more in-depth discussion on implant surgery.
   b. Acting as the "root" of the implant, the implant body is generally shaped like a threaded screw with varying degrees of surface roughness (**Figure 8-5**). These roughened-surface threads provide greater surface area to engage the adjacent peri-implant bone, thereby optimizing better stress distribution and lowering implant micromotion which favor osseointegration.

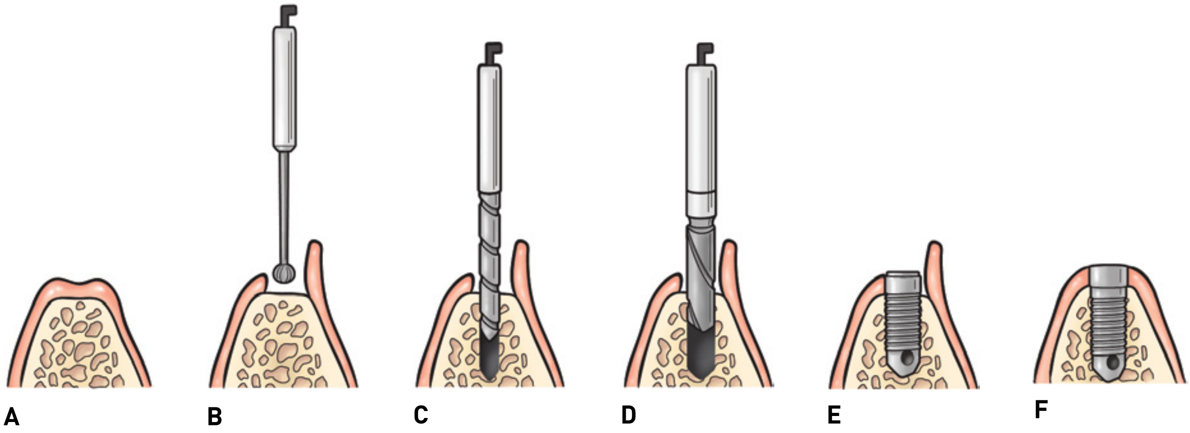

Figure 8-4 Surgical Placement of a Dental Implant. A. Edentulous alveolar ridge. **B.** Initial implant site preparation with a round bur. **C** and **D.** A series of sequential drills with increasing diameters are used to prepare the implant site to the size of the planned implant. **E.** Implant body seated in the implant site. The top of the implant body may be placed slightly above, level with, or slightly below the crest of the bone. **F.** Implant body seated in bone with cover screw attached. At the end of placement surgery, the implant can be covered with gingiva or left exposed to the oral cavity, as shown here. A healing time of several weeks to months is allowed so that osseointegration can occur.

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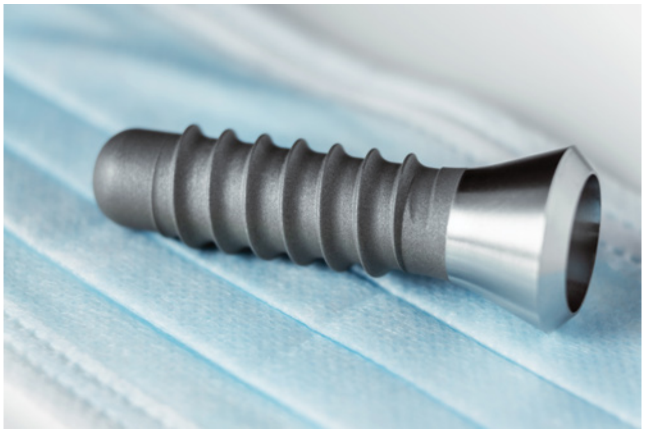

Figure 8-5 Titanium Implant Fixture. This implant is made of titanium and is almost always accepted by the body cells in the alveolar bone without any problems.

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1. The metal commonly used for conventional dental implants is titanium or titanium alloy for several reasons.
   a. Titanium is a lightweight metal with a high degree of strength and durability.
   b. Titanium is a **biocompatible** (not rejected by the body) metal when placed in the human body.

   c. Titanium is a passive metal that does not readily corrode (rust) when placed in a fluid-filled, aqueous environment, such as the oral cavity.
   d. Titanium is a poor conductor of heat and electricity. This is important because a metal with a high degree of electrical or thermal conductivity can readily transmit heat or electricity to the surrounding host tissues and damage it.
2. Titanium has several potential disadvantages, however.
   a. Titanium is softer than other dental restorative metals and is easily scratched by nontitanium metal instruments (such as a conventional stainless steel periodontal probe). This can lead to bacterial adherence to the scratched titanium surface.
   b. A few case reports have shed light that some individuals may be susceptible to developing unfavorable immunologic reactions to titanium particles released from titanium implants due to corrosion or wear of the implant. In turn, the deposition of these titanium particles in the surrounding peri-implant tissue can provoke an inflammatory reaction that may lead to implant failure and/or a hypersensitivity reaction (known as "titanium allergy").[3] However, our knowledge in this aspect is still rudimentary, and much more research is needed to determine the biologic mechanism behind titanium sensitivity. Nonetheless, at this time, titanium sensitivity cannot be excluded as a cause of implant failure and/or a cause of unwanted systemic inflammatory effects.
3. In light of some of these asserted disadvantages, implant manufacturers are looking into using other types of nonmetallic materials, such as ceramics, as a suitable alternative to titanium. One such ceramic material is zirconia (**Figure 8-6**). While the biocompatibility and the physical and mechanical characteristics of zirconia are similar, if not superior, to titanium, the ivory (whitish) color of a zirconia implant results in a better esthetic outcome, especially in patients with a thin gingival phenotype, compared to the grayish color of titanium that may be visible through thin gingiva (**Figures 8-7** and **8-29A**). As implant manufacturers investigate alternative nonmetallic materials to use, it is important for the dental hygienist to be aware of the many changes that are coming down the pipeline in the implant market.

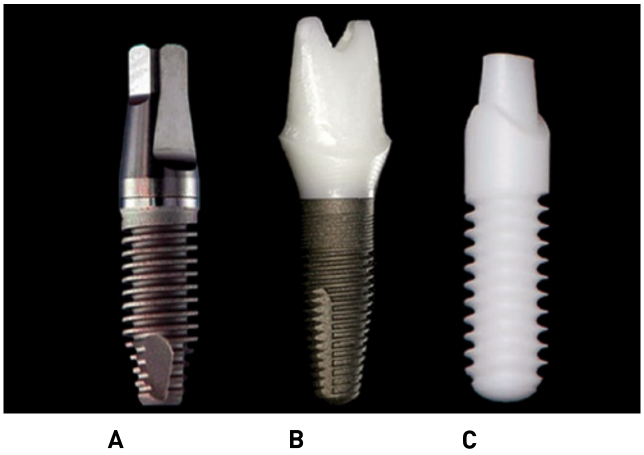
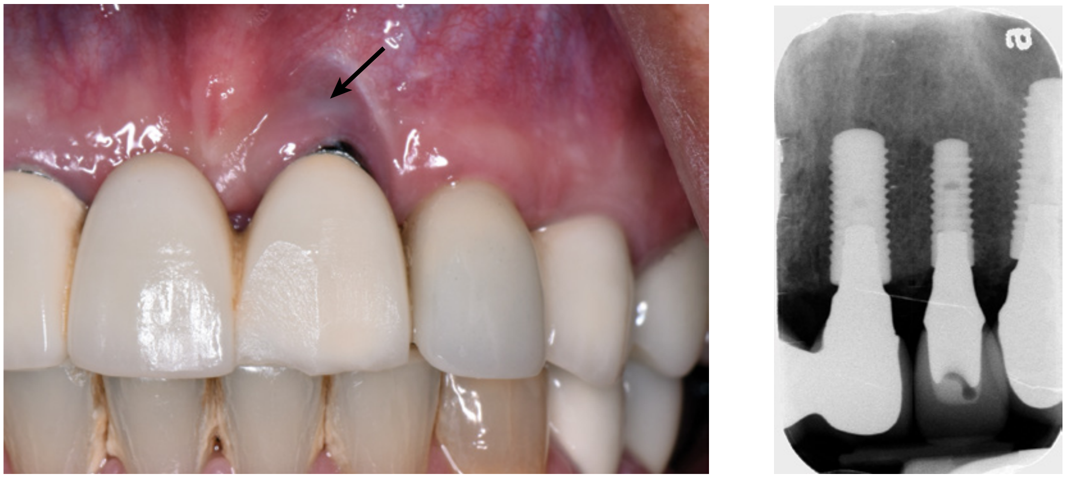


Figure 8-6 Differences in Esthetic Appearance of Titanium Versus Zirconia. Esthetic differences between titanium and zirconia are quite apparent from this figure. **A, B.** Titanium has a grayish dark color that could potentially shine through a thin gingiva and even detract from the most beautifully made implant crown. **C.** Zirconia, however, is more esthetically pleasing because of its ivory (white) color that does not shine easily through the gingiva and is therefore not visibly apparent through the thin gingiva. Combining a zirconia abutment with a zirconia implant improves the esthetics of the implant restoration because it eliminates the problem of trying to match the shade of adjacent teeth while masking the color of an underlying titanium abutment.

Courtesy of CeraRoot USA Inc.



A

B

Figure 8-7 Titanium Implant versus Zirconia Implants in a Patient with a Thin Gingival Phenotype. A. Three implants were placed in the maxillary left central incisor, maxillary left lateral incisor, and canine regions. All three implants have integrated well into the surrounding peri-implant tissues. However, observe the grayish, metallic shade of the underlying titanium implant in the central incisor region shining through the thin gingival phenotype (arrow). In contrast, the zirconia implants placed in the lateral incisor and canine regions are not evident because they do not shine through the thin gingiva. Unlike titanium, zirconia has a high degree of translucency, thereby providing a more high-end esthetic result. **B.** Accompanying radiograph confirms osseointegration of all three implants which supports the high degree of biocompatibility of zirconia and titanium.

2. **The Abutment**
    a. The **implant abutment** is a metal post that connects the implant prosthesis to the implant body and secures the implant prosthesis in place (**Figure 8-8**). The implant abutment either protrudes partially or completely through the gingival tissue into the oral cavity (**Figure 8-9**).
    b. Like the implant body, the abutment is made from either titanium or zirconia because of the extremely biocompatible property of these metals.

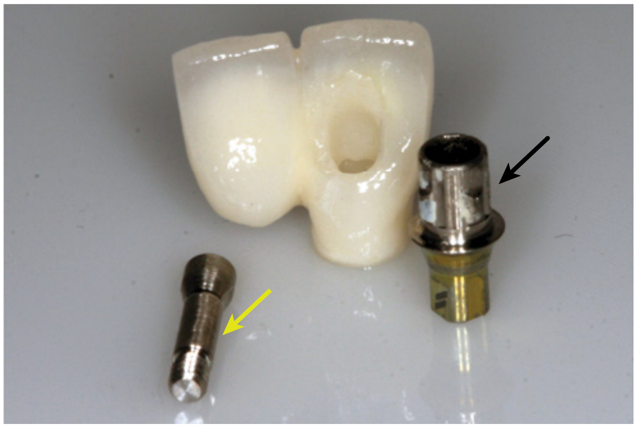


Figure 8-8 Implant Prosthetic Abutment and Abutment Screw. The prosthetic abutment (black arrow) is the connection between the implant prosthesis and the implant body. The abutment screw (yellow arow) fixates and secures the prosthetic abutment to the implant body.

Courtesy of Sopanis D. Cho, DDS, MSD, from a case developed by Sopanis D. Cho, DDS, MSD and Peerapat Kaweewongprasert DDS, MSD.

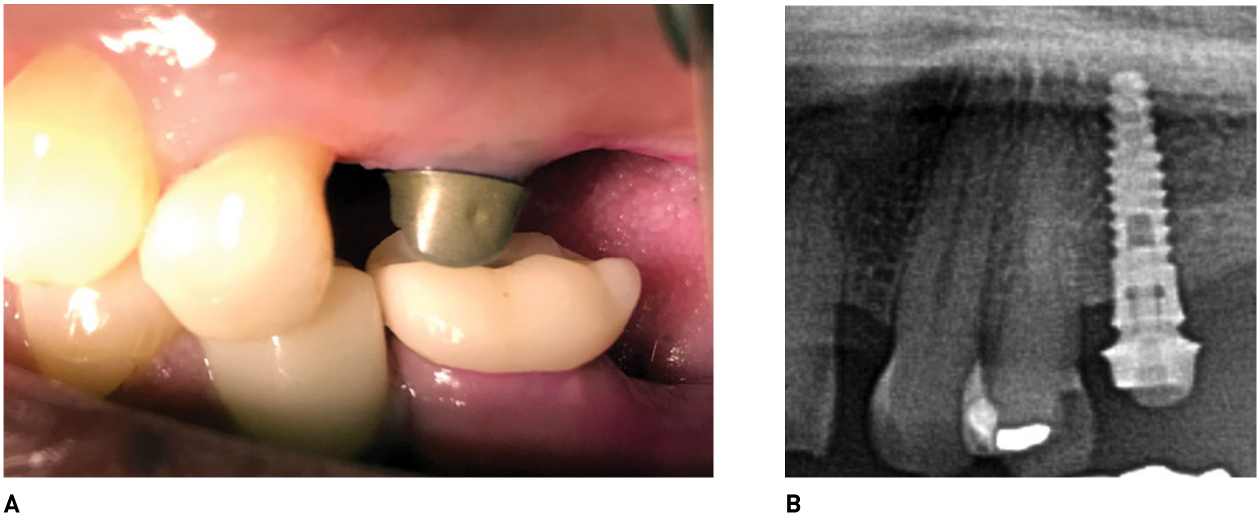


Figure 8-9 Implant Prosthetic Abutment. A. Prior to prosthetic (crown) delivery, a photograph of the prosthetic abutment was obtained. The prosthetic abutment is screwed into the implant body and will join the future prosthesis to the underlying implant body. Note the healthy appearance of the peri-implant soft tissue.
B. Radiograph of the same implant with the abutment prior to delivery of the implant prosthesis. Note the absence of peri-implant bone loss.

2. **The Peri-Implant Tissues.** The **peri-implant tissues** are the hard and soft tissues that surround the dental implant (**Figure 8-10**). The peri-implant tissues are similar in many ways to the periodontium of a natural tooth, but there are some important differences (**Table 8-1**).

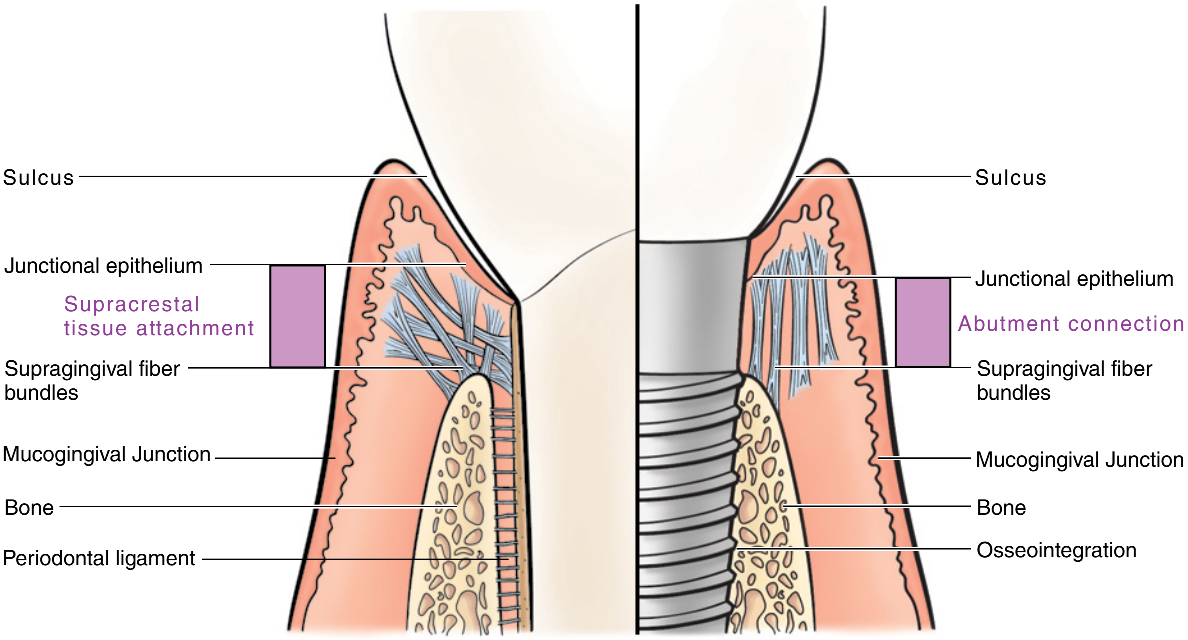

Sulcus
Junctional epithelium
Supracrestal tissue attachment
Supragingival fiber bundles
Mucogingival Junction
Bone
Periodontal ligament
Sulcus
Junctional epithelium
Abutment connection
Supragingival fiber bundles
Mucogingival Junction
Bone
Osseointegration

Figure 8-10 Comparison of the Periodontal Interface to a Natural Tooth Versus the Peri-Implant Interface to a Dental Implant. Unlike the periodontal interface to a natural tooth, the dental implant lacks the periodontal ligament connection to the alveolar bone. Further, the supragingival fiber bundles are oriented parallel or obliquely to the implant surface and do not insert into the implant.

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Table 8-1 Tissues Surrounding a Dental Implant

Tissue	Peri-Implant Tissue
Junctional epithelium	Attaches to the implant surface or abutment surface (biologic seal)
Connective tissue (supragingival fiber bundles)	Run parallel or obliquely to the implant and abutment surface or encircles the entire implant and abutment
Periodontal ligament	No periodontal ligament
Cementum	No cementum
Alveolar bone	In direct contact with the implant surface (osseointegration)

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a. **Implant-to-Epithelial Tissue Interface**
   1. The epithelium adapts to the abutment post or to the implant itself, creating a cuff-like **perimucosal seal** (biological seal). The union of the epithelial cells to the abutment or implant surface is very similar to that of the epithelial cells to the natural tooth surface.
   2. The perimucosal seal functions as a barrier between the implant and the oral cavity.
   3. As with a natural tooth, a sulcus lined by sulcular epithelium and junctional epithelium surrounds the implant abutment or, in some cases, the implant collar (the top-most part of the implant that is at the same level of the surrounding peri-implant bone).

b. **Implant-to-Connective Tissue Interface**
   1. The implant-to-connective tissue interface is significantly different from that of the connective tissue surrounding a natural tooth.
   2. The implant surface lacks cementum. Thus, the supragingival fiber bundles and the periodontal ligament do not insert into the implant surface as they do into the cementum of a natural tooth.
      a. On a natural tooth:
         1. The supragingival fibers brace the gingival margin against the tooth and strengthen the attachment of the junctional epithelium to the tooth. The supragingival fibers are oriented perpendicular to the long axis of the tooth and insert directly into the cementum.
         2. The periodontal ligament suspends and maintains the tooth in its socket.
         3. The periodontal ligament fibers also serve as a physical barrier to bacterial invasion.
      b. On an implant:
         1. The supragingival fiber bundles support the healthy gingiva against the abutment. The supragingival fiber bundles have been shown to be either (1) oriented parallel or obliquely in relation to the implant and abutment surface or (2) encircle the implant and abutment.[4] The fibers do not attach to the dental implant like they do on a natural tooth.
         2. There are no periodontal ligament fibers to provide protection for the dental implant. Therefore, periodontal pathogens can create inflammation and destroy bone much more rapidly along a dental implant than along a natural tooth with its protective barrier of periodontal ligament fibers.[5]
         3. Because there are no supragingival fibers or periodontal ligament fibers inserting into the titanium surfaces, a periodontal probe will pass more easily through the inflamed peri-implant soft tissues to the apical connective tissue or close to the peri-implant alveolar bone.

c. **Implant-to-Bone Interface**
   1. **Osseointegration** is the direct contact of the living bone with the surface of the implant body (with no intervening periodontal ligament). To put it another way, an osseointegrated implant is functionally ankylosed to the surrounding bone without the intervening periodontal ligament (PDL). Osseointegration is the major determinant for implant success.

2. The criteria for successful osseointegration are as follows:
    a. An absence of clinical mobility of the implant.
    b. No discomfort or pain when the implant is in function.
    c. No increased bone loss or radiolucency surrounding the dental implant on a radiograph.
    d. Less than 0.2 mm of bone loss annually after the first year in function.[6]

Section 2

Peri-Implant Health, Diseases, and Conditions

The 2017 AAP/EFP World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions proposed a new category titled “peri-implant diseases and conditions.” Four peri-implant conditions fall under this new category: (1) peri-implant health, (2) peri-implant mucositis, (3) peri-implantitis, and (4) peri-implant soft and hard tissue deficiencies.[7]

1. **Peri-Implant Health**
    a. **Peri-implant health** is characterized by an absence of erythema, bleeding on probing, swelling, and suppuration. Clinically, there are no visual differences between healthy peri-implant tissues and healthy periodontal tissues. **Figures 8-11** and **8-12** demonstrate cases of peri-implant health.
    b. Probing depths may be deeper at a healthy implant site compared to a healthy tooth site, but this is due to the vertical/oblique orientation of the connective tissue fibers, which offer less tissue resistance to probe penetration.

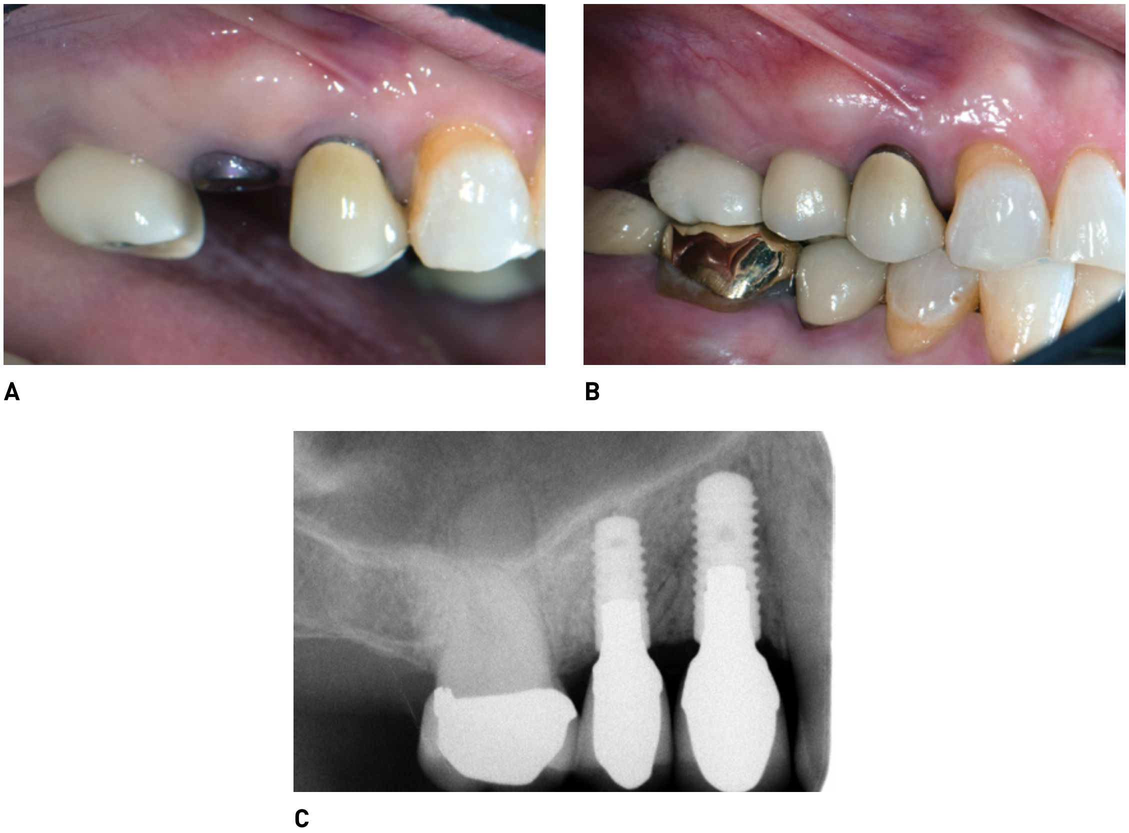

Figure 8-11 **Healthy Peri-Implant Tissues. A.** Note the healthy peri-implant tissue surrounding two implants placed in the premolar region. (Implant in the first premolar region was restored prior to obtaining the clinical image while the implant in the second premolar region has not been restored at this point in time.) **B.** One-year clinical photograph. Note there are no differences in appearance between the healthy peri-implant tissues and the healthy periodontal tissues surrounding the adjacent natural teeth. **C.** One-year radiographic follow-up. Note the absence of bone loss around both implants.

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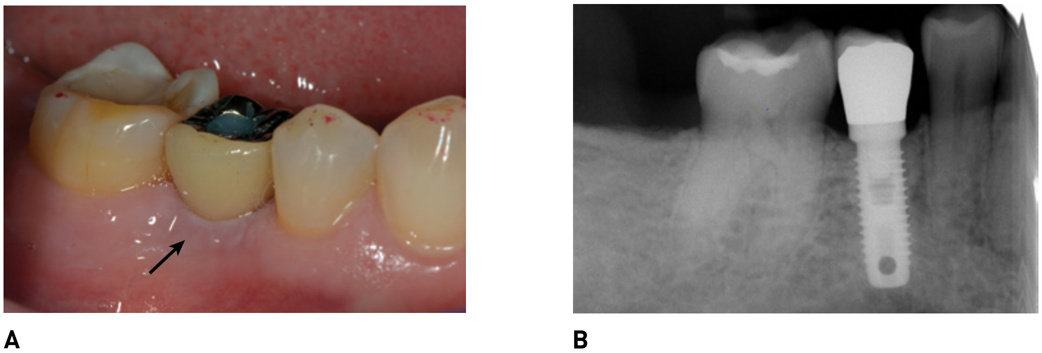

Figure 8-12 **Healthy Peri-Implant Tissues 15 Years Following Implant Loading. A.** Note the coral pink, knife-edge appearance of the tissue margin surrounding the dental implant (arrow). **B.** Radiograph of the same implant confirms no signs of peri-implant bone loss.

2. **Peri-implant mucositis** (also called peri-implant gingivitis) is a plaque biofilm-induced inflammation of the soft tissues—with no loss of supporting bone—that is localized in the mucosal tissues surrounding a dental implant.[8] **Figure 8-13** shows an example of peri-implant mucositis associated with the implant replacing the maxillary left lateral incisor. Compare this to the appearances of the healthy peri-implant tissues surrounding the implants replacing the maxillary right lateral incisor, maxillary right central incisor, and maxillary left central incisor.
   a. Peri-implant mucositis is reversible if the etiologic factors are removed.[9] However, if the etiologic factors are not removed, peri-implant mucositis will persist, or it may progress to peri-implantitis.
   b. Peri-implant mucositis has been reported to occur in about 80% of subjects and 50% of implant sites; peri-implantitis has been reported to occur in 28% to 56% of subjects and 12% to 43% of sites.[10] The reported prevalence, however, varies widely and changes as implant designs evolve.
   c. According to the 2017 AAP/EFP World Workshop, a diagnosis of peri-implant mucositis requires:
      1. Visual signs of soft tissue inflammation, red as opposed to pink (**Figure 8-14**) and/or swollen as opposed to no swelling (**Figure 8-15**).
      2. Presence of bleeding and/or suppuration upon probing. (Note that in the absence of inflammatory changes a local dot of bleeding resulting from traumatic probing should not be considered as a clinical sign of peri-implant disease.)
      3. Increased probing depths compared to previous measurements.
      4. Absence of bone loss.

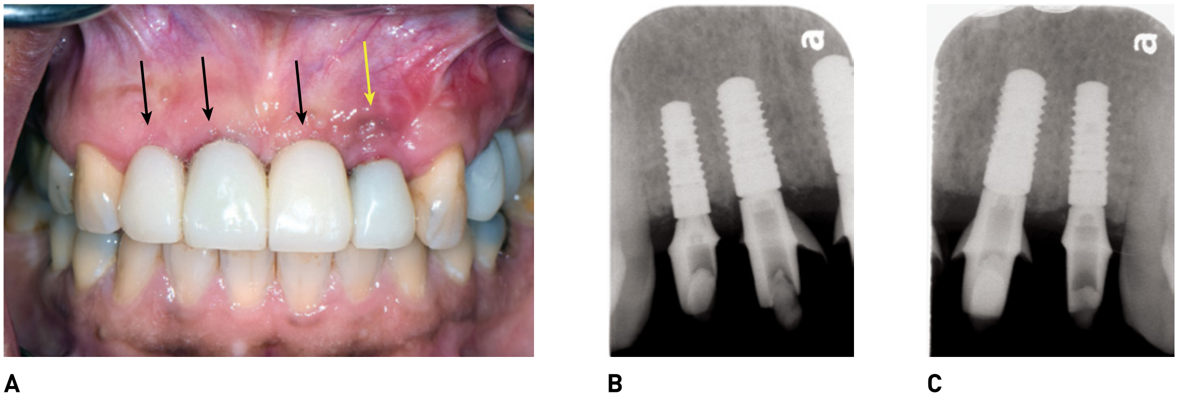

Figure 8-13 **Peri-Implant Health and Peri-Implant Mucositis.** Note the absence of any clinical signs of peri-implant inflammation of the implants in the maxillary right lateral, maxillary right central, and maxillary left central incisor positions (black arrows). However, note the clinical sign of peri-implant inflammation associated with the maxillary left lateral incisor implant (yellow arrow). The accompanying radiographs reveal no bone loss around the maxillary implants. Thus, based on the clinical and radiographic presentation, a diagnosis of peri-implant health was made for the maxillary right-sided implants and the maxillary left central incisor implant, whereas a diagnosis of peri-implant mucositis was made for the maxillary left lateral incisor implant. If the etiologic factors are completely removed, the peri-implant mucositis observed around the maxillary left lateral incisor implant may revert to peri-implant health.

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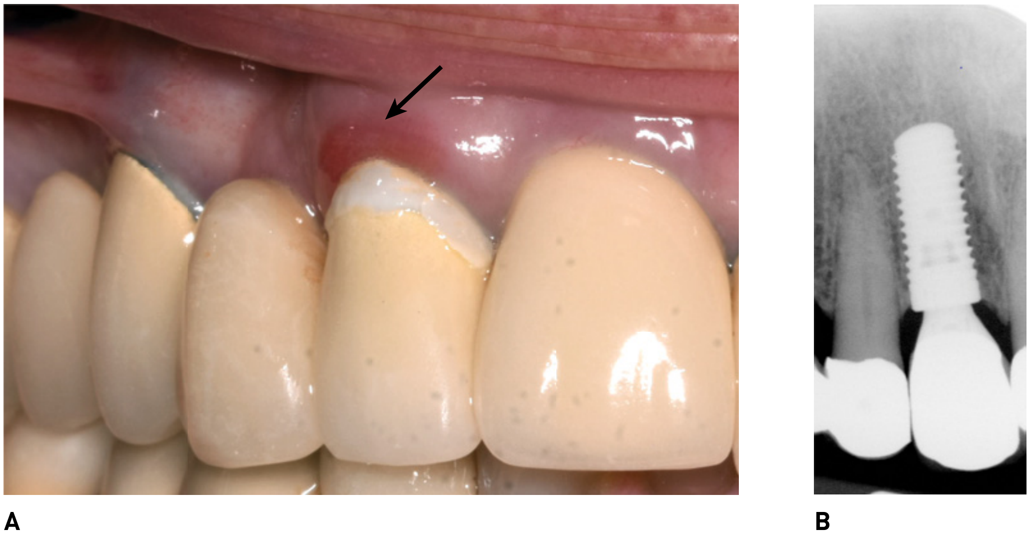

Figure 8-14 **Peri-Implant Mucositis. A.** Note the linear band of erythema along the margin of the peri-implant tissue (arrow). Also, note plaque buildup along the margin. The accumulation of plaque triggers the host inflammatory response, which leads to the erythematous appearance of the tissue margin. **B.** Accompanying radiograph of the same case. Note the implant collar coincides with the peri-implant crestal bone level. Additionally, note the absence of any radiographic signs of pathology around the implant.

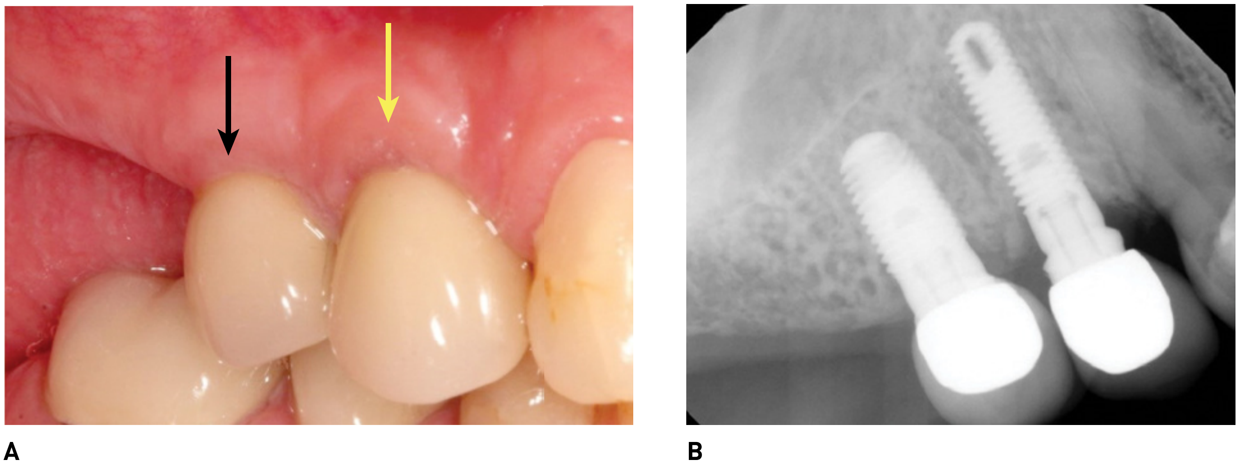

Figure 8-15 **Peri-Implant Health and Peri-Implant Mucositis. A.** Visually, note the knife-edged peri-implant tissue margin associated with the distal implant (replacing the maxillary second premolar), which is consistent with peri-implant health (black arrow). In contrast, note the rounded, thickened, swollen peri-implant tissue margin associated with the mesial implant (replacing the maxillary first premolar) (yellow arrow). **B.** Radiograph of the same case. Note that the collar of both implants coincides with the crestal bone. Also note the absence of any pathology around the implants.

3. **Peri-implantitis** is essentially periodontitis affecting the soft and hard tissues surrounding a functioning osseointegrated dental implant, characterized by a plaque biofilm-induced inflammation in the peri-implant mucosal tissues with progressive loss of supporting alveolar bone.[11]
   a. The onset of peri-implantitis may occur early during follow-up and may progress in a nonlinear and accelerating pattern.
   b. Peri-implantitis lesion can be diagnosed by the detection of radiographic bone loss around the implant (**Figures 8-16** and **8-17**).
      1. The implant does not become mobile until the final stages of peri-implantitis. In fact, in most cases, the patient is unaware that they have peri-implantitis until the implant becomes mobile (**Figure 8-18**).
      2. An implant that exhibits mobility is an indication that it has lost osseointegration. If this is observed, the implant should be explanted (removed).[12,13]

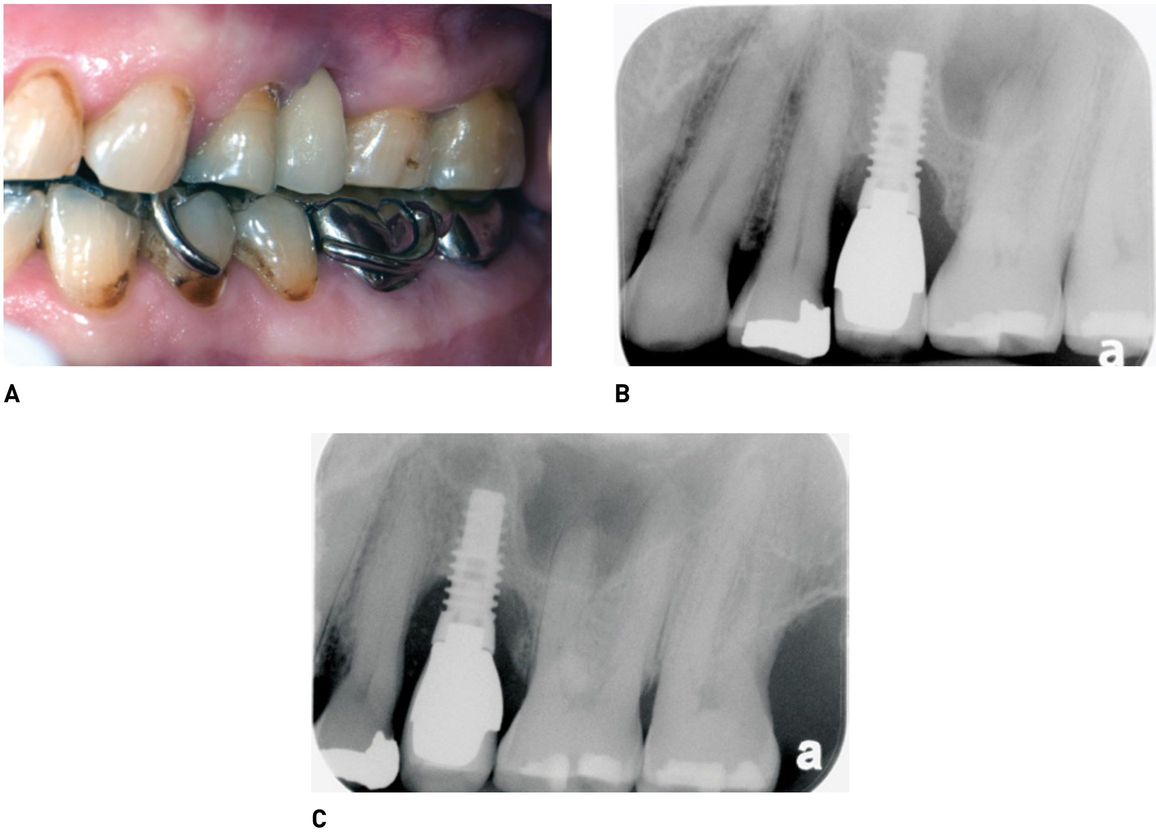

Figure 8-16 Peri-Implantitis (implant in the maxillary left second premolar position). A. Note the clinical signs of peri-implant soft tissue inflammation. Clinical evaluation reveals bleeding upon probing and peri-implant probing depths ranging from 7 to 10 mm. The clinical photograph was taken 18 months post-crown cementation and corresponds to the radiograph depicted in panel C. **B.** Previous periapical radiograph taken at the 12-month post-crown cementation appointment. Note that at 12 months the radiograph reveals loss of supporting bone. Nonsurgical therapeutic intervention was attempted at this stage but proved to be ineffective. **C.** 18-month post-crown cementation. By comparing this radiograph against the 12-month radiograph (B), one can appreciate the progressive nature of bone loss that has occurred between the two different time periods. If surgical therapeutic intervention is not performed, the implant will inevitably be lost.

Courtesy of Dr. Takaaki Kishimoto, Indianapolis, IN.

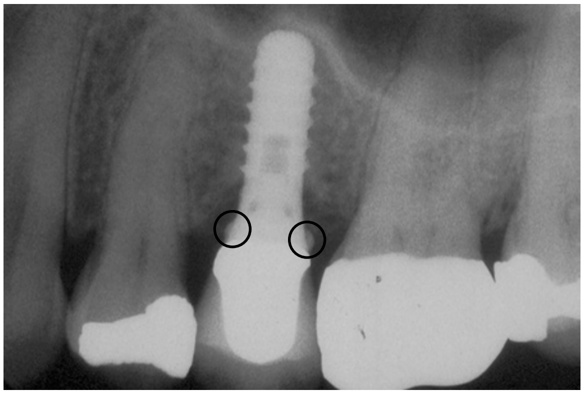

Figure 8-17 Peri-Implantitis. This radiograph shows bone loss around an implant supporting a single crown. Note the residual cement near the crown margin, which is a contributing factor to peri-implantitis.

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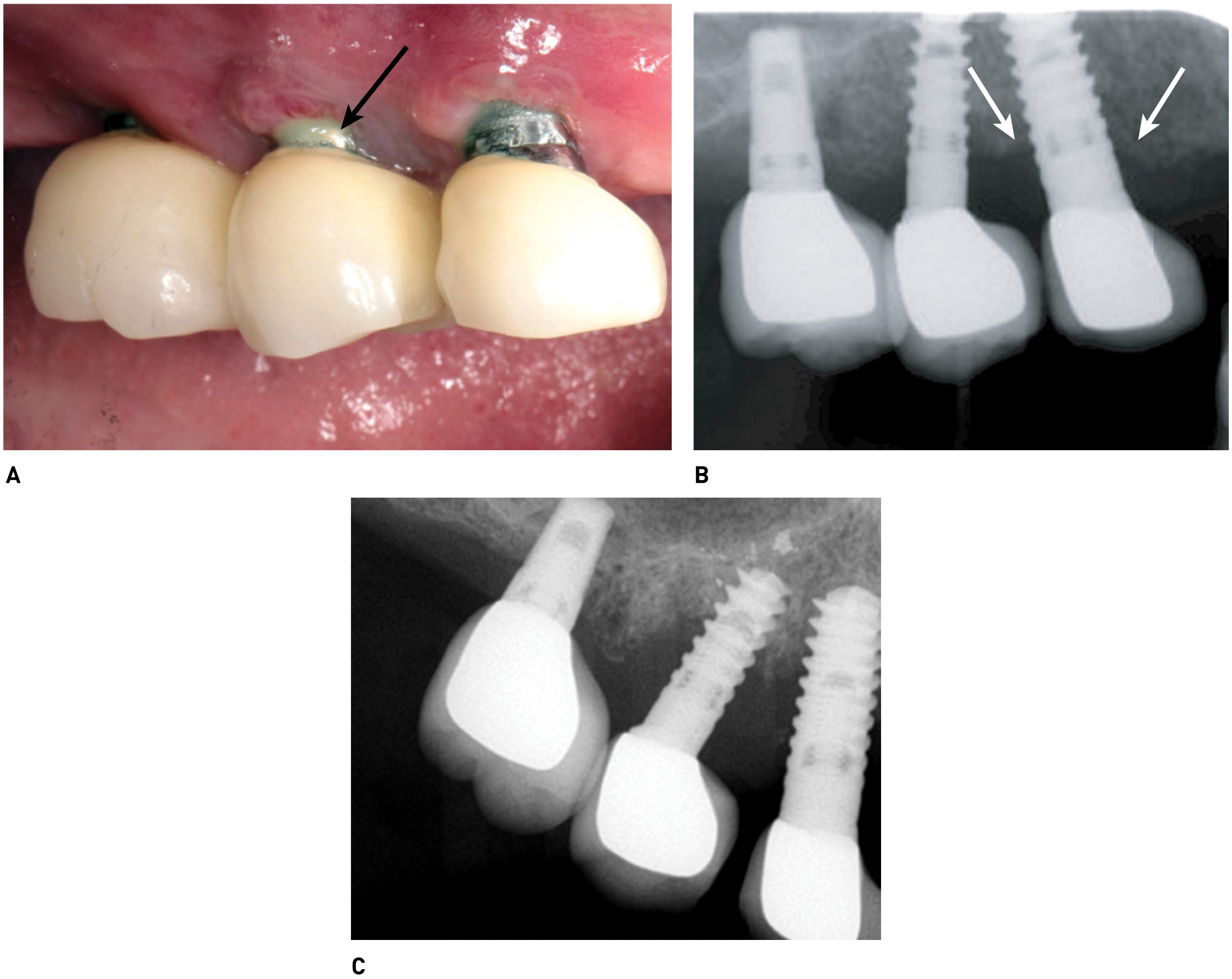

Figure 8-18 Peri-Implantitis. The 72-year-old patient presented in March 2021 with the chief complaint, "My implants feel loose." Implants were restored over 15 years ago. Patient has a history of noncompliance and irregular home care. **A.** Note the visual cues of inflammation associated with all three implants. Further, note the purulent discharge from the middle implant (arrow). **B.** Past peri-implant radiograph from April 2018. Note the early signs of peri-implant bone loss (blue arrows). **C.** Present peri-implant radiograph from March 2021. Note the progressive nature of the peri-implant bone loss associated with all three implants over the last 3 years. All three implants were subsequently explanted.

Courtesy of Dr. Takaaki Kishimoto, Indianapolis, IN.

c. Differences in the prevalence of peri-implantitis have been reported by a number of studies. The prevalence reported in these studies ranges from a prevalence of 6.61% to 47%.[14,15]

d. The 2017 AAP/EFP World Workshop has established the following criteria for a diagnosis of peri-implantitis:
   1. Visual signs of inflammation (red as opposed to pink, swollen tissue as opposed to no swelling, soft as opposed to firm tissue consistency).
   2. Presence of bleeding and/or suppuration upon probing. (Note that in the absence of inflammatory changes a local dot of bleeding resulting from traumatic probing should not be considered as a clinical sign of peri-implant disease.)
   3. Increased probing depths compared to previous measurements.
   4. Progressive bone loss seen by examining the bone levels between two radiographs taken at different time periods (i.e., comparison of radiograph taken at baseline vs. radiograph taken 12 months after cementation of the implant prosthesis).
   5. In the absence of initial radiographs and probing depths (lack of baseline recordings), radiographic evidence of bone level ≥ 3 mm and/or probing depths ≥ 6 mm in conjunction with profuse bleeding represents peri-implantitis.

4. **Hard and Soft Tissue Deficiencies**
    a. Following tooth loss, bone resorption naturally takes place. which leads to hard tissue deficiencies (**Figures 8-19** and **8-20**).
    b. The following factors may contribute to hard tissue deficiencies: loss of periodontal support caused by periodontitis; endodontic infections, which spread into the periodontal tissues (endodontic–periodontal lesions); thin buccal bone plates; extraction with additional trauma to the tissues; traumatic injury (i.e., sports injury); systemic diseases impairing normal bone remodeling (i.e., osteopenia or osteoporosis); and pneumatization of the maxillary sinus. To correct this type of deficiency, hard tissue augmentation (ridge augmentation with bone grafting) must be performed either before implant surgery or simultaneously with implant surgery.
    c. Another common type of deficiency seen with implants is soft tissue recession. This may be due to implant malpositioning; lack of buccal bone; thin, soft tissue; lack of keratinized tissue; status of attachment of the adjacent teeth; and surgical trauma. **Figure 8-21** shows a deficiency in which recession of the gingival margin resulted in the exposure of the implant collar.

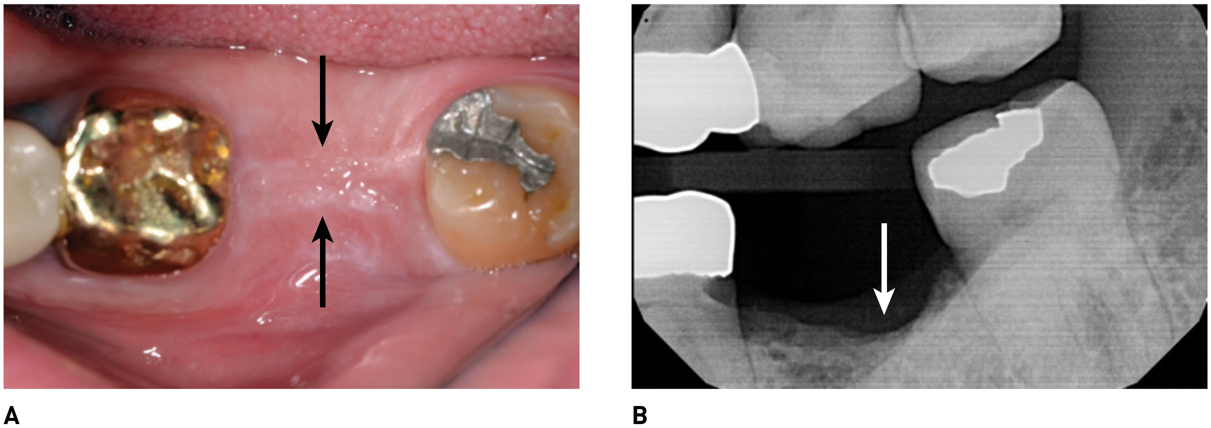

Figure 8-19 **Hard Tissue Deficiency. A.** Although it is evident that there is abundant keratinized gingiva, the edentulous ridge is narrow due to horizontal bone loss (arrows). **B.** Accompanying radiograph demonstrates that there is also a loss of apico-coronal bone height (vertical bone loss) in the region (arrow).

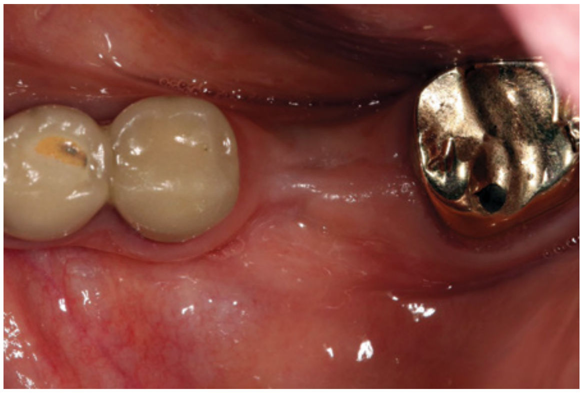

Figure 8-20 **Hard Tissue Deficiency.** Clinical photograph of an atrophic edentulous ridge demonstrating buccal bone loss.

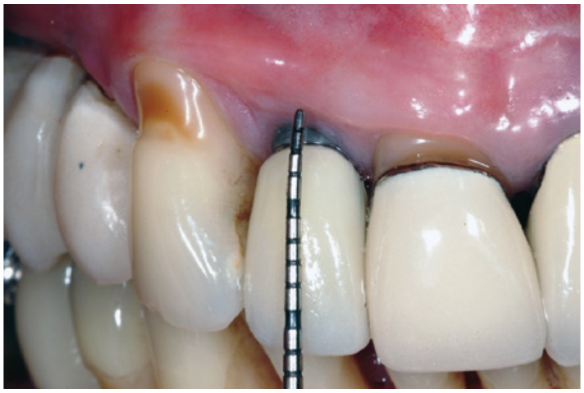

Figure 8-21 **Soft Tissue Deficiency.** This patient presented with the chief complaint, "I can see the top of my implant!" Clinical evaluation reveals an apical shift of the peri-implant gingival tissue and 2 mm of the implant collar exposed. To resolve the patient's chief complaint, soft tissue augmentation can be performed to cover the exposed implant surface.

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d. In some cases, both types of tissue deficiencies may be present at the same edentulous site, thus requiring both soft tissue augmentation and hard tissue augmentation either prior to or during implant surgery (**Figure 8-22**). The possible etiologies can be caused by a combination of factors that were listed above.

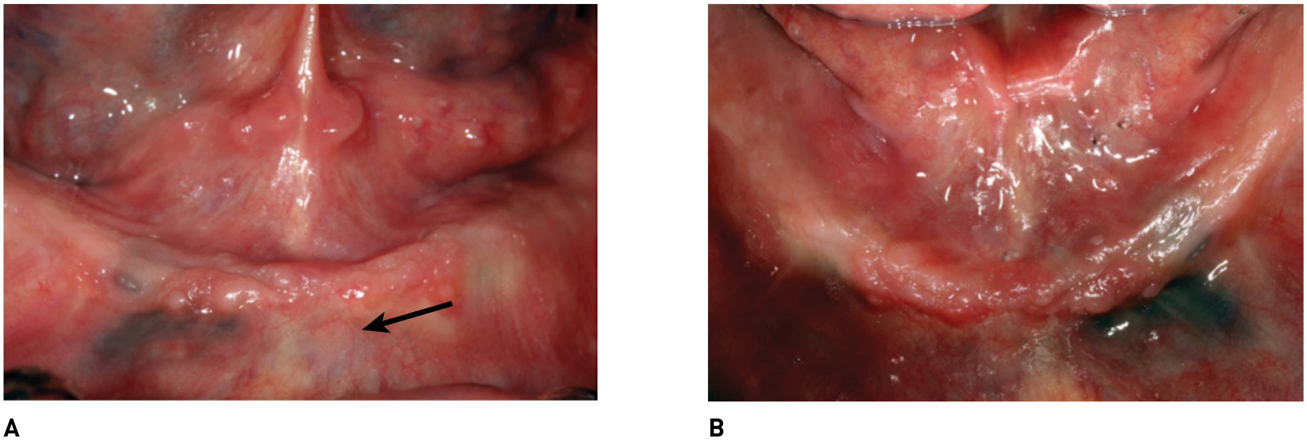


Figure 8-22 Hard Tissue/Soft Tissue Deficiencies in Edentulous Arch. Mandibular arch of a 72-year-old patient who has been fully edentulous for the past 15 years. From **A** and **B**, note the loss of vertical bone height (as evident by the level of the ridge coinciding with the floor of the mouth), lack of vestibular depth, and lack of keratinized tissue.

Courtesy of Dr. Chandni Batra, Indianapolis, IN.

Section 3

Recognition of Peri-Implant Diseases

The two forms of peri-implant diseases—peri-implant mucositis and peri-implantitis—share a common primary etiology—dental biofilm. Biomechanical factors, the patient's medical history control, and smoking status are additional factors that can contribute to peri-implant diseases.

1. **Etiology and Risk Factors**
   a. **Etiology: Dental Biofilm**
      1. Peri-implant mucositis—like dental biofilm-induced gingivitis—occurs primarily as a result of a biofilm-induced infection. It develops from healthy peri-implant mucosa following accumulation of bacterial biofilm around an osseointegrated implant.
      2. Peri-implantitis—like periodontitis—occurs primarily as a result of an overwhelming bacterial infection and the subsequent host immune response.[15] Peri-implant mucositis is considered to be a precursor of peri-implantitis. However, keep in mind that peri-implant mucositis does not always convert into peri-implantitis. Similar to our current understanding of the conversion of gingivitis to periodontitis, the conditions leading to the conversion from peri-implant mucositis to peri-implantitis are still not understood.
      3. Periodontitis and peri-implantitis progress in a similar fashion. The rate of tissue destruction, however, tends to be more rapid in peri-implant tissues than in periodontal tissues.
      4. No *single* microorganism has ever been implicated to be the causative agent of peri-implantitis. Rather, human cross-sectional studies have revealed that peri-implantitis is a polymicrobial infection (an infection that is related to several types of microorganisms) that is associated with several bacterial species associated with periodontitis.[17–21] A recent study identified a cluster of seven bacterial species that could be associated with peri-implantitis: *Tannerella forsythia*, *Porphyromonas gingivalis*, *Treponema socranskii*, *Staphylococcus aureus*, *Staphylococcus anaerobius*, *Streptococcus intermedius*, and *Streptococcus mitis*. The total bacterial load of these seven species was four times higher in implants demonstrating signs of peri-implantitis compared to healthy implants. Thus, it was concluded that the bacterial burden may be an important factor in peri-implantitis.[22]
   b. **Risk Factors.** The risk factors identified for each patient are essential in forecasting the prognosis of the implants, and these factors should be included in the determination of the appropriate maintenance

interval. As in the treatment of periodontal disease, each patient must be assessed for the presence of risk factors that may lead to peri-implant disease or its progression. Risk factors that should be considered include previous history of periodontal disease, ineffective self-care, residual cement, smoking, and biomechanical overload.[16]

1. **History of Previous Periodontal Disease.** Several systematic reviews indicated that peri-implantitis is a more frequent finding in patients with a history of periodontitis compared to patients without a history of periodontitis.[23–26]
2. **Poor Plaque Biofilm Control/Lack of Regular Maintenance Therapy.** Like natural teeth, there is evidence that poor plaque control and lack of regular maintenance therapy are risk factors for peri-implant diseases.
3. **Smoking.** Four systematic reviews concluded that there is an increased risk for peri-implantitis in smokers.[24,27–29] For a more thorough review on the impact of smoking on peri-implant diseases, the reader can refer to Section 6 of Ch. 17.
4. **Residual Cement.** Residual cement may have been inadvertently left behind because the positioning of the implant or the prosthetic contour complicates detection and removal.[30] When left behind, residual cement may induce inflammation due to its rough surface topography providing an environment for bacterial attachment.[31] Thus, at the time of cementation, the clinician should make every effort to ensure complete removal of excess cement.
5. **Biomechanical Overload**
    a. Collectively, the forces applied on an implant have been called **biomechanical forces** to underscore the complexities of both the “biological” and “mechanical” components of occlusion.
        1. Implants can withstand normal bite forces. However, in patients who have parafunctional habits (like bruxing or clenching), the implant may not be able to withstand the excessive magnitude of parafunctional loads.
        2. “Force duration” refers to the *time* that the teeth come together. In normal swallowing or eating, the teeth only come into contact briefly. But in patients with parafunctional habits (e.g. clenching or grinding), the teeth remain in contact more than the several hours each day. In such a case where a patient has an implant(s), the longer force duration would account for greater fatigue loads imposed on the implant(s).
    b. Biomechanical forces on implants are influenced by a variety of factors that must be assessed by the clinician. Factors that influence the biomechanical forces include how much occlusal force is placed on the implant(s), the position of the implant(s), the number of implant(s) supporting a prosthesis, and the distribution of the occlusal forces among the implant(s) and remaining teeth.
    c. At each visit, the dental clinician should assess the patient’s occlusion to verify that the applied biomechanical forces transmit appropriate loading forces, aid in patient comfort during mastication, and provide uniform stress distribution along the entire length of the implant.
    d. Implant-related complications can arise in the absence of plaque. An example of this occurs when biomechanical force exceeds the physiologic tolerance of the peri-implant bone to withstand loading forces. This is known as **biomechanical overload**.
        1. It is thereby important to reiterate that **even in the absence of plaque, implant-related complications such as biomechanical overload can lead to implant failure**.
        2. Factors that may lead to biomechanical overload include patient parafunctional habits (like bruxing), suboptimal implant design and size, an insufficient number of implants to support the restoration, improperly splinted abutments, and improperly positioned implants.
    e. Because dental implants do not have a periodontal ligament, forces placed on an implant are transmitted directly to the surrounding peri-implant bone.[32,33] It is therefore critical for the clinician to control the forces placed on an implant to avoid subjecting the implant to biomechanical overload that can damage the surrounding peri-implant bone.
        1. Around a natural tooth, the periodontal ligament functions to absorb some of the forces placed on the tooth and plays a key role in the normal bone remodeling process. The forces placed on natural teeth can arise from chewing food, supporting a dental appliance, or perhaps from parafunctional habits, such as bruxing.

2. Dental implants, on the other hand, lack the protective features of the periodontal ligament that are found on natural teeth. Instead, as mentioned above, an osseointegrated dental implant is in direct contact with the alveolar bone that completely supports it. If the osseointegrated implant is subjected to biomechanical overload, these excessive forces will be directed to the surrounding alveolar bone and irreversibly traumatize the bone support. If the overload is not controlled at its early stages, then loss of osseointegration may eventually occur.
3. To emphasize again, in the absence of plaque, an implant can still fail if the occlusal forces are excessive and not controlled. In such a case, the cause of implant failure is not due to bacterial invasion, but rather due to biomechanical overloading (**Figure 8-23**). This should not be confused with peri-implantitis, which requires the presence of plaque biofilm to trigger the host inflammatory response.

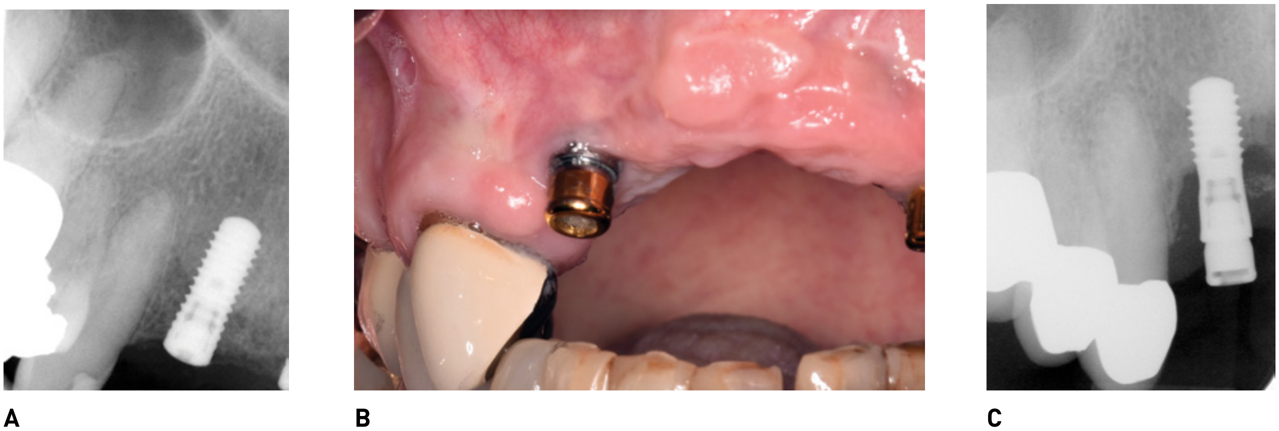


Figure 8-23 **Biomechanical Overload. A.** Baseline radiograph taken at the time of surgical implantation. **B.** Four-year postoperative clinical photograph of the implant with an implant locator attachment to support a multiunit implant prosthesis. Note the complete absence of plaque biofilm and the lack of any visual cues of inflammation. Peri-implant probing depths range from 1 to 3 mm with no bleeding upon probing. **C.** Four-year postoperative radiograph. Note the saucer-shaped osseous defect. Patient exhibited signs of good home care and was fully compliant and motivated. However, the implant-prosthesis was ill-fitting and contributed to significant occlusal interferences when the patient underwent protrusive movements (with the prosthesis in her mouth).

f. Both bacterial plaque-related causes and excess biomechanical forces can contribute to the development of peri-implant disease. Both should be assessed and controlled at each implant maintenance appointments.

6. **Spatial Placement of Implants.** Proper three-dimensional spatial positioning of a dental implant(s) within bone *at the time of surgery* is critical because it impacts the long-term function and esthetics of the implant restoration.
   a. A properly placed implant facilitates good plaque control and limits the biomechanical stress applied to the implant and the prosthesis.
   b. In contrast, strong evidence suggest that poor spatial positioning increases the risk of implant-related complications such peri-implant diseases and/or biomechanical overload. **Figure 8-24** shows the combined effect of plaque-biofilm accumulation and suboptimal placement of two adjacent implants resulting in peri-implant tissue loss.

2. **Detection of a Failing Implant**
   a. **Clinical Signs of a Failing Implant**
      1. **Soft Tissue Indicators.** Clinical signs of a failing implant include the presence of a peri-implant pocket, bleeding after gentle probing, and/or suppuration from the pocket. The surrounding gingival tissue may or may not be swollen. Pain is usually not present.

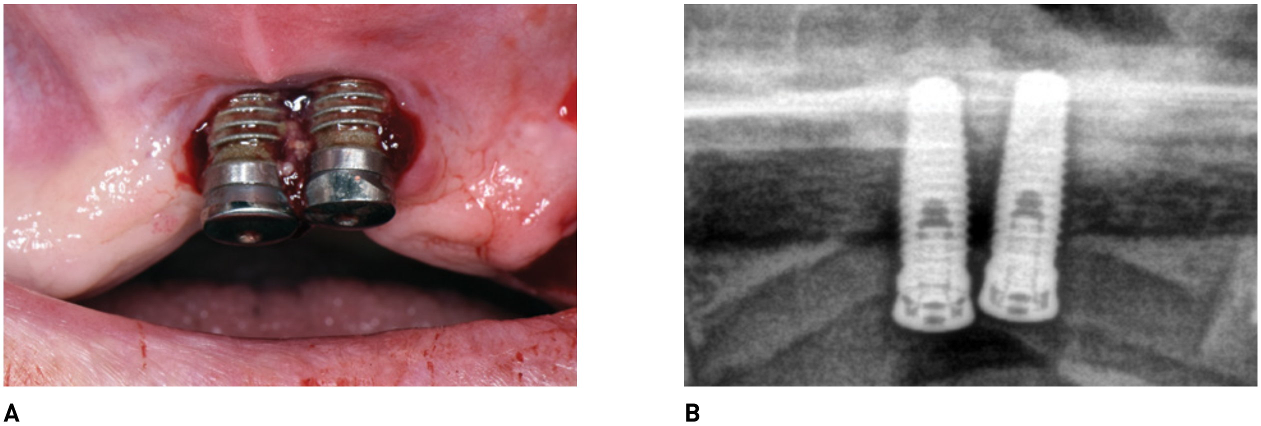


Figure 8-24 **Suboptimal Positioning of Dental Implants. A**. Clinical photograph showing two malpositioned implants spatially placed too close together and positioned too far facially. Note the thread exposure and the presence of plaque between the exposed threads. **B.** Accompanying radiograph. In this case, no single etiology could be identified as being the root cause for the peri-implant tissue loss. Rather, multiple factors—namely, plaque biofilm and suboptimal placement of the dental implants—have led up to the eventual outcome—implant failure.

Courtesy of Dr. Takahiro Fujii, Indianapolis, IN.

2. **Implant Mobility.** Absence of mobility is a very important clinical criterion for dental implants. The presence of mobility is the best indicator for diagnosis of implant failure.[5]
    a. Implants should not be mobile if osseointegrated and healthy.[34,35]
        1. Implant mobility may indicate a loss of osseointegration.
        2. In some instances, the implant restoration, not the implant body itself, may be mobile. This is usually because of a loose abutment or the rupture of the cement seal on cemented restorations rather than loss of implant osseointegration. Mobility also can result from a loosening of the internal screw that fastens the abutment to the implant or the restoration, and therefore is not the result of peri-implant disease.
        3. Severe mobility accompanied with discomfort also might indicate fracture of the implant itself.
        4. Long-term mobility or misfit between the prosthetic components (e.g., screws between the crown and implant) may lead to persistent inflammation, bone loss, and the eventual complete failure of the implant.
    b. The technique for assessing mobility of a dental implant is similar to that used to assess a natural tooth. Two instruments with plastic handles are used to grasp the implant restoration and apply force back and forth in the facial and lingual direction. Remember that the implant restoration can be mobile while the implant body remains osseointegrated.
    c. Radiographic evaluation is recommended when any mobility is noted. Loose internal screws or components will often be seen as a gap between the implant components on a radiograph.

b. **Radiographic Signs of a Failing Implant**
    1. Radiographic signs of peri-implantitis include vertical destruction of the crestal bone around the implant—which assumes the shape of a saucer—while the bottom portion of the implant remains osseointegrated.[36] **Figure 8-25** shows a radiographic series of a failing implant over a 3-year period.
    2. Another radiographic indicator of peri-implantitis is wedge-shaped defects along the implant[36] (**Figure 8-25D**).
    3. A peri-implant radiolucency usually indicates bone loss adjacent to the implant and is a key indicator of a failing implant.

3. **Treatment Modalities for Failing Implants**
    a. Various methods are available for the treatment of peri-implantitis, including nonsurgical periodontal instrumentation, the use of antiseptics, local and/or systemic antibiotics, and access flap surgery.[36]
        1. All these treatment modalities have been used with varying degrees of success, but at this time there is no standard protocol for the treatment of peri-implantitis.[36]

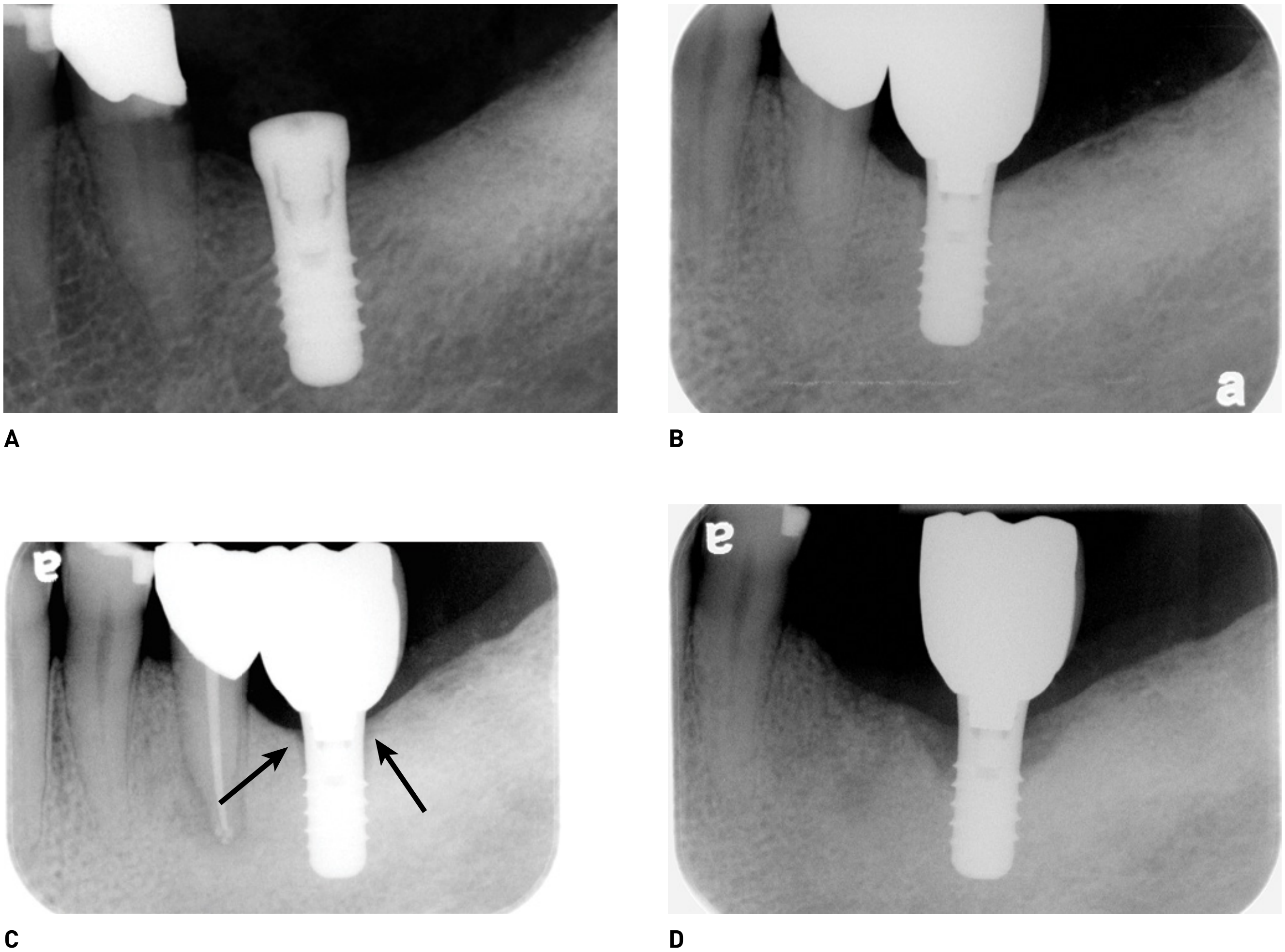


Figure 8-25 **Radiographs of Progressive Peri-Implant Bone Loss Over a 3-Year Period. A.** Baseline radiograph obtained at the time of surgical implant placement. **B.** Radiograph obtained at the time of implant crown delivery. **C.** One-year postoperative radiograph. Note a narrow wedge-shaped radiolucency defect developing at the coronal aspect of the implant (arrow) on both the mesial and distal sides. Also note the presence of a periapical radiolucency associated with the adjacent premolar, which was subsequently extracted. **D.** Three-year postoperative radiograph. Note a more widened saucer-shaped defect that extends down to the two-most coronal threads.

    2. Surgical treatment in which the lost bone is reestablished through bone grafting shows promising results.[36]

b. Nonsurgical periodontal instrumentation of peri-implantitis lesions with adjunctive local delivery of microencapsulated minocycline or chlorhexidine may be beneficial to patients with peri-implantitis.[18,37–40]

c. The available evidence suggests that subgingival glycine powder air polishing for biofilm removal may reduce clinical signs of peri-implant mucosal inflammation to a greater extent than periodontal instrumentation with plastic curettes combined with adjunctive irrigation with chlorhexidine.[41]

Section 4

Clinical and Radiographic Monitoring of Peri-Implant Health and Diseases

Routine monitoring of dental implants should be part of any comprehensive periodontal examination and maintenance. It is essential to the early detection and the effective management of peri-implant diseases.

1. **Probing**
   a. Initial probing of the implant should be done once the final restoration has been installed.[16,42]
      1. In the past, routine periodontal probing of dental implants was not recommended by some authors. More recent literature, however, indicates that routine probing does not harm the implant or the peri-implant tissues and should be done routinely to assess the status of the peri-implant tissues.[16,42] Probing around osseointegrated implants does not appear to have detrimental effects on the soft tissue seal and, hence, does not seem to jeopardize the longevity of oral implants.[43]
      2. Some implant surgeons recommend that probing should be avoided until postoperative healing is complete, approximately 3 months after abutment connection.
      3. A probing technique with light force (0.25 N, using a probe with a tip diameter of 0.45 mm) should be used because the perimucosal seal is weakly adherent to the titanium surface.[43] On the other hand, heavy probing force will be invasive because the probe can easily penetrate through the perimucosal seal and introduce bacteria into the peri-implant environment.[44–46] Similar to probing the periodontal tissues around a natural tooth, the depth of penetration of the probe tip in the peri-implant tissue is dependent on the health (or inflammatory stage of the peri-implant tissues) and the thickness of the tissue around the abutment.
      4. Commercially available plastic probes have long been used when investigating the depth of the peri-implant sulcus.[5] Recent literature recommends the use of conventional metal periodontal probes if probing pressures are kept light.[47] Light lateral forces on the probe will protect against damaging titanium surfaces. However, plastic probes are more flexible and sometimes advantageous in probing excessive contours and angles found in implant restorations (**Figure 8-26**).

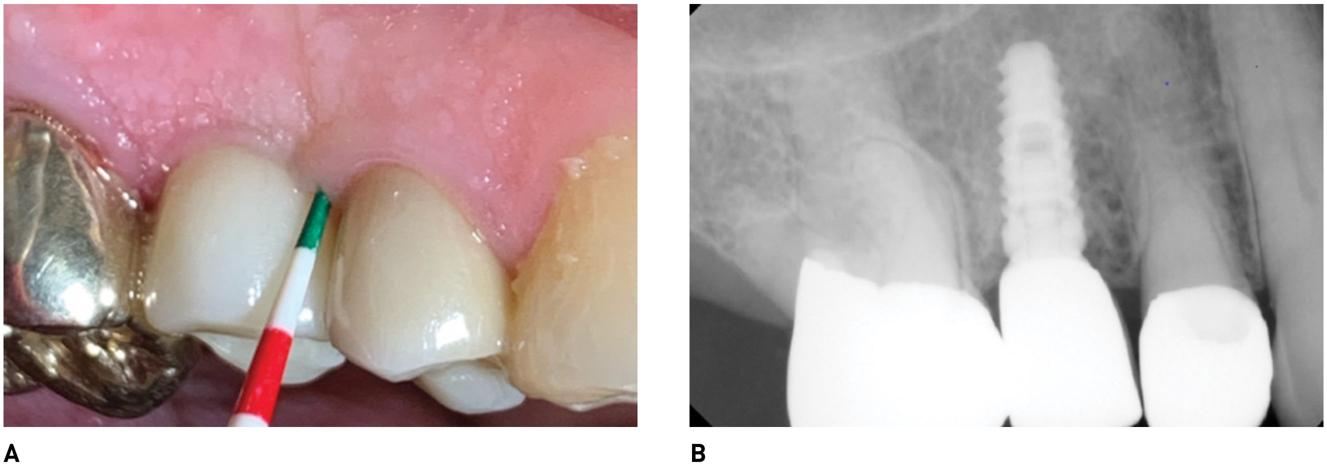


Figure 8-26 Plastic Periodontal Probe. A. Gentle probing of peri-implant tissues with plastic probe. Note the absence of bleeding upon probing and the shallow pocket depth. **B.** Accompanying radiograph taken during the same appointment. Note the absence of peri-implant bone loss.

      5. Unlike natural teeth, it is not possible to define a range of probing depths compatible with peri-implant health.
         a. Probing depth measurements taken at any single appointment—without previous measurements to compare against—provide very little valuable information regarding the health of the peri-implant tissues.
         b. However, *changes in probing depth measurements taken over time*—comparing measurements at today's appointment to those from previous appointments—is valuable for detecting changes in tissue/attachment health and alerting the clinician of the need for therapeutic intervention.

    Thus, peri-implant probing is still an essential component of each routine visit as one of the primary means of detecting the early signs of peri-implant diseases.
  c. Clinical attachment levels can be used to monitor peri-implant health.
6. To interpret probe readings, the clinician must have baseline data recording previous probing levels of attachment and a fixed reference point for repeatable probing comparisons.
  a. Initial probing of the implant should be done once the final restoration has been installed. Probing depth should be recorded and defined as the depth of probe penetration from the base of the implant sulcus to the crest of the mucosa. Similar to assessing natural teeth, the level of the crestal soft tissue attachment can be measured using a fixed reference point on the margin of the implant crown and should be noted as the clinical attachment level.
  b. Changes in probing attachment levels over time may be more important than the initial findings as implants may have deeper soft tissue probing depths. Due to variation in the depth of surgical placement, the tissue thickness at the site, as well as different lengths of the abutments and the connective tissue interface with the abutment, probing depths may be deeper than the 1- to 3-mm depths that are considered normal with natural teeth.
7. The depth of penetration of the probe tip also is dependent on the health (or inflammatory stage of the peri-implant tissues) and the thickness of the tissue around the abutment.

2. **Bleeding and Suppuration**
  a. Bleeding on probing and/or the presence of suppuration is an indicator of peri-implant tissue inflammation which should be further investigated.
    1. Lack of bleeding on gentle probing and a lack of suppuration indicates tissue health around implants.
    2. Elimination of the bleeding on probing in peri-implant mucositis is important and should be accomplished with improved biofilm removal by the clinician and the patient.
    3. Increasing probing depth and the presence of bleeding or suppuration warrant radiographic assessment of the implant.[10,48]
    4. If present, peri-implant bleeding and/or suppuration should be recorded at specific sites. Suppuration may spontaneously extrude from the sulcus or be expressed from the opening of the sulcus by gentle probing with an instrument or by gently compressing the tissue over the implant with a gloved finger or the blunt end of a dental mirror. The presence of pus is a foreboding sign of peri-implant disease and should be immediately addressed upon detection (**Figure 8-27**).

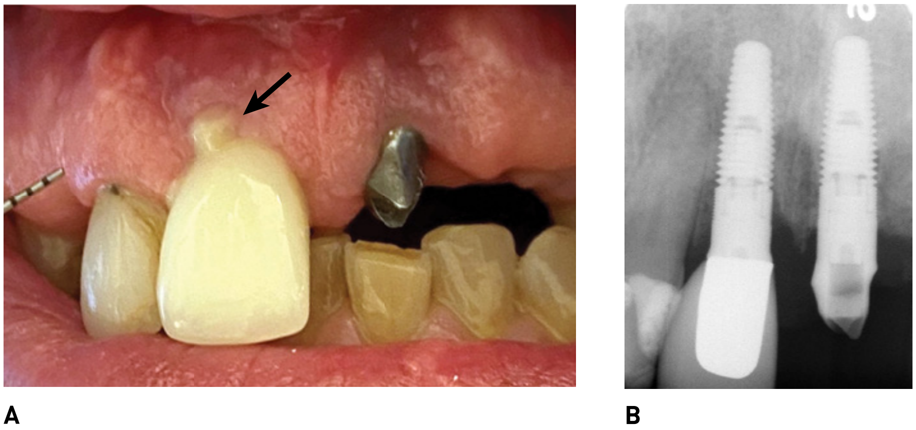


Figure 8-27 Peri-Implant Suppuration. A. Note the thick milky, yellowish, opaque discharge oozing out of the sulcus (arrow). Suppuration is a sign of infection in the host tissues. **B**. Accompanying radiograph reveals an angular defect along the mesial aspect of the implant and loss of trabeculation along the distal aspect of the implant. (Note that at the time that this clinical photograph and radiograph were taken the adjacent implant was not restored due to the presence of suppuration from the maxillary right implant.)

3. **Radiographs**
   a. *Prior* to cementation of the implant crown, the clinician should take a radiograph (1) to ensure that there is an absence of any pathology that may be indicative of an infection and/or (2) to verify the spatial positioning of the implant. If peri-implant pathology is noted (**Figure 8-28**) or if the spatial positioning of the implant looks suspect (**Figure 8-29**), then delivery of the final prosthesis should be delayed until the above issues are resolved to avoid any future implant-related complications.
   b. *On the day of* the cementing of the implant crown (or re-cementation of the crown), the clinician should take a post-prosthetic insertion x-ray to assess for the presence of remaining excess cement. Cements that are visible radiographically should be used for implant restorations, especially if the margins and probable location of excess cements are subgingival. Post-insertion radiographs also permit the clinician to assess the fit of the prosthesis and the integrity and adaptation of the different implant components (**Figure 8-30**).

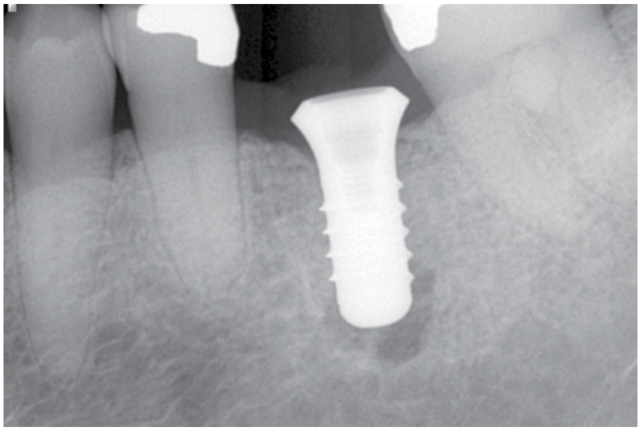
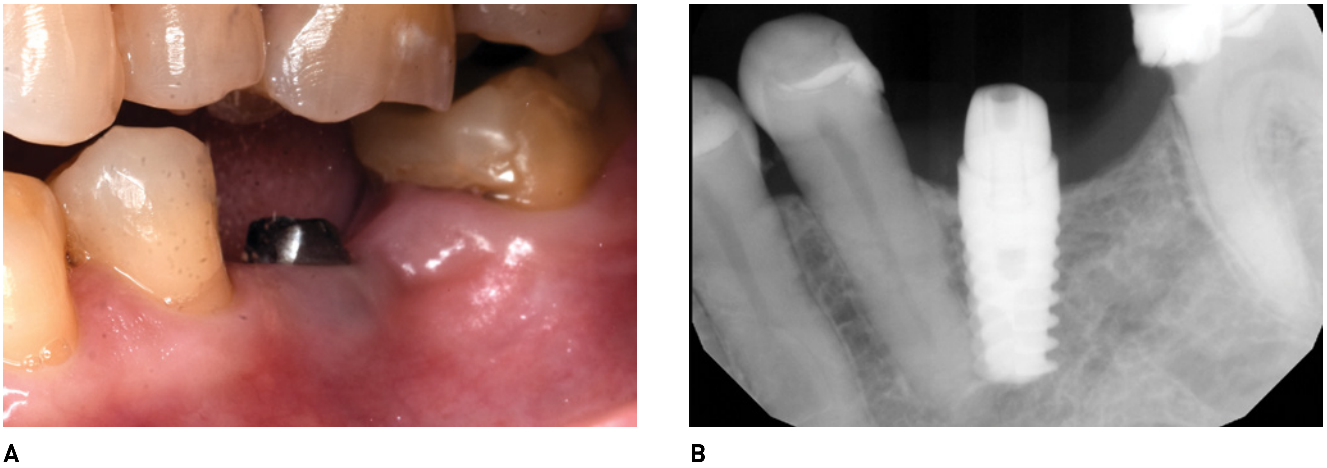


Figure 8-28 Residual Osseous Pathology Prior to Implant Loading. Note the presence of a radiolucency at the apex of the implant. Final prosthetic delivery was postponed until the residual peri-implant infection was successfully managed.



A

B

Figure 8-29 Poor Spatial Positioning of the Implant. A. Clinical photograph of the implant abutment on the day of implant crown delivery. Visually, the titanium implant appears to be in the correct spatial position. Yet, despite the peri-implant tissues appearing healthy, the grayish hue of the titanium implant shines through the thin gingiva which is apparent to the patient. **B.** Accompanying radiograph of the same implant, however, reveals something much more alarming. Note the orientation of the long axis of the implant in relation to the long axis of the adjacent teeth. If the implant was restored at this time, then the direction of the biomechanical force would be non-axially oriented (off-angled). This, in turn, would generate greater shear forces at the implant-to-bone interface and lead to implant-related complications. Hence, because of the off-axial orientation of the implant body, it was decided that the implant should not be restored.

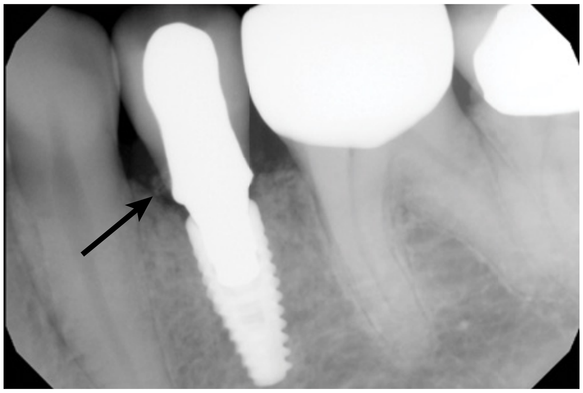


Figure 8-30 Post-prosthetic Insertion Radiograph Taken on the Day of Implant Crown Delivery. Note the close, intimate marginal fit between the various implant components. However, note the radiopaque cement on the mesial surface of the implant crown (arrow).

c. Following successful delivery of the definitive implant prosthesis, the implant should be followed-up radiographically at least once a year and should be checked more often in patients in whom periodontal breakdown around an implant was noted at a previous visit. Radiographic evaluation of bone height and topography is necessary for the longitudinal monitoring of peri-implant stability.[45,46,48]

1. Vertical bone loss of less than 0.2 mm annually following the implant's first year of function is a criterion utilized to determine treatment success.
2. Baseline radiographs should include the day of surgical implant placement, the day of final prosthesis insertion, and periodically during implant maintenance.[16] Bone remodeling during the first year after the final prosthesis insertion is expected, and then there should be less than 0.2 mm of bone loss annually thereafter (**Figure 8-31**).
3. The shape and amount of bone remodeling following the final prosthesis insertion is different for different implant systems and configurations.
4. If prior radiographs are not available, use 2.0 mm of bone loss from the expected bone level for the implant at that time point as the "decision point" for the diagnosis of peri-implantitis.[47]
5. Radiographs made on the day of the final prosthesis insertion (or re-cementation) should be reviewed for the presence of remaining excess cement. Cements that are visible radiographically should be used for implant restorations, especially if the margins and probable location of excess cements are subgingival.
6. Radiographs also allow for the evaluation of the fit of the prosthesis and the integrity and adaptation of the different implant components.
7. Dental implants should be evaluated radiographically at least once a year and should be checked more often in patients in whom periodontal breakdown around an implant was noted at a previous visit.

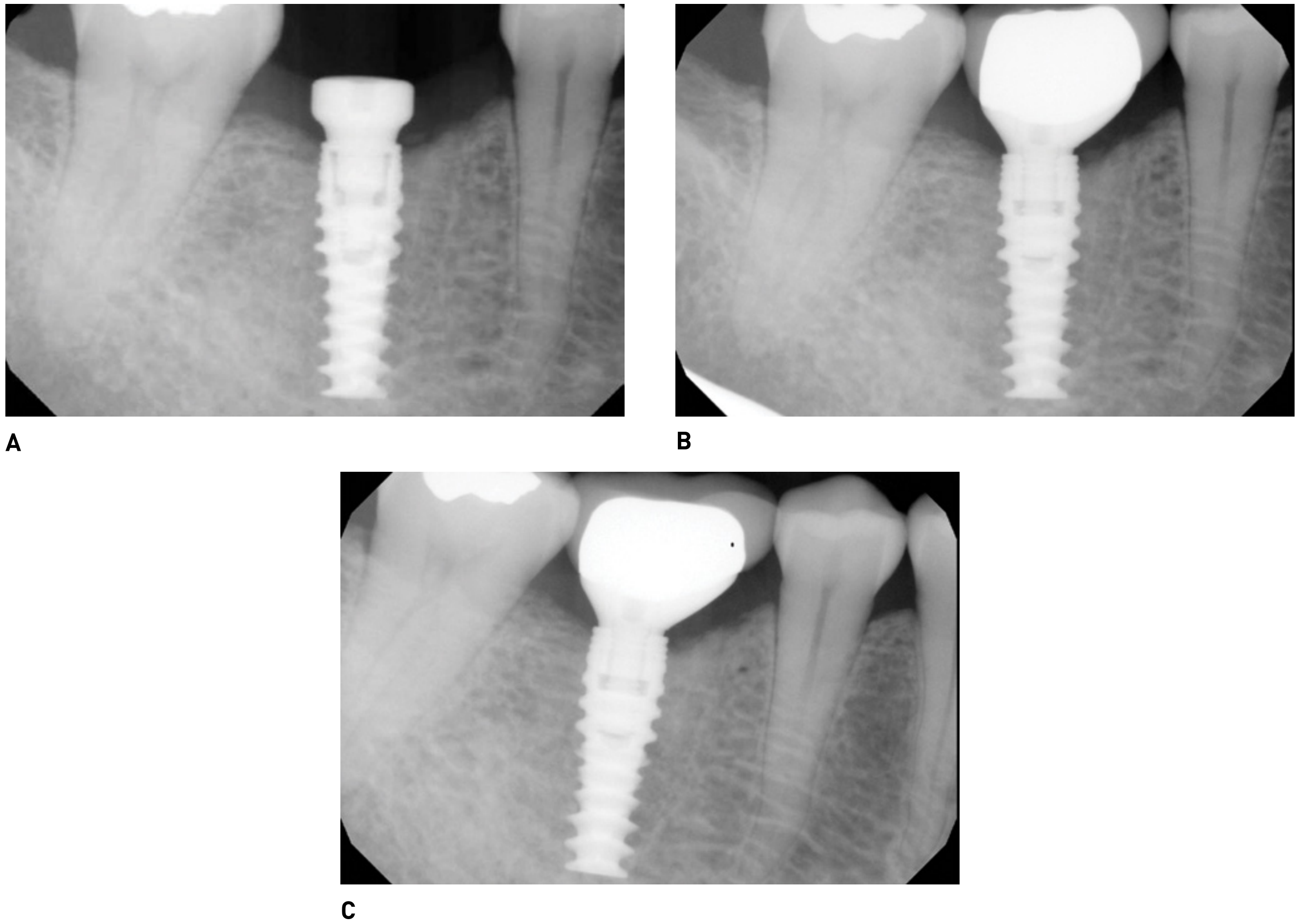


Figure 8-31 **Periodic Radiographic Assessment of a Dental Implant. A.** Baseline radiograph obtained immediately at the time of surgical placement of the implant body. **B.** Radiograph taken immediately following cementation of the final implant crown. **C.** One-year post-insertion radiograph. Note the preservation of peri-implant bone levels over the 1-year period.

Section 5

Guidelines for Maintenance of Patients with Dental Implants

When they occur, implant-related complications such as peri-implantitis and biomechanical overloading are extremely challenging to both manage and treat. If not addressed successfully or in a timely manner, implant-related complications can further deteriorate to a point that it jeopardizes implant survivability. Moreover, management of implant-related complications can be costly (as it will most likely require surgical intervention) and debilitating (if it requires removal of the implant and rehabilitation of the site).

In contrast, the saying "An ounce of prevention is worth a pound of a cure" is something that should be at the top of mind for both the dental clinician and the patient. Taking steps to stave off implant-related complications is easier and more efficient than trying to address an insidious and progressing implant-related complication that has the potential to compromise implant survival. Thus, *prevention* of implant-related complications should be a top priority in everyday clinical practice.

1. **Considerations for Implant Maintenance**
    a. **Goals of Maintenance Therapy for Dental Implants**
        1. **Maintenance of Alveolar Bone Support**
            a. Alveolar bone support is evaluated by use of good-quality radiographs taken with a long-cone paralleling technique at specific time intervals.

   b. The bone height and density around the implants is compared with previous radiographs of the site.
2. **Control of Inflammation**
   a. Patient and professional biofilm control are important to maintaining the health of the peri-implant tissues.
   b. Patient self-care must be reevaluated and, if necessary, reinforced each time the patient is seen for maintenance. The better the patient self-care, the better the possibilities of maintaining stable results.
3. **Maintenance of a Healthy and Functional Implant**
   a. Implant components should be checked for prosthesis integrity (such as mobility, loose screws, cement washout, material wear); implant, screw, or abutment fracture; unseating of attachments; and proper adaptation of all components.
   b. Any mobility of an implant or its restorative components requires immediate consultation with a dentist or specialist.
4. **Maintenance of a Healthy and Functional Periodontium Surrounding Neighboring Teeth**
   a. It is theorized that the natural teeth in a partially edentulous mouth act as a reservoir of periodontal pathogens that colonize the implants.
   b. This finding makes meticulous self-care of dental implants and natural teeth even more critical for the partially edentulous patient than for the fully edentulous patient.
   c. Before beginning an examination, it is very important to obtain information from the patient related to implant-supported restorations or prostheses. The patient can often identify problems for clinicians that are otherwise difficult to find. Implant patients should be encouraged to share their perceptions of any changes in the fit, tightness, or feel of the implant restoration, including the occlusion. Helpful questions to ask the patient include:
      1. **Questions About Daily Self-Care of Implant-Supported Restorations/Prosthesis**
         a. Are you able to clean around the neck portions of your implant(s) easily?
         b. Do you still have enough cleaning aids to perform daily oral self-care?
      2. **Questions Concerning Patient Satisfaction.** General questions regarding the patient's satisfaction with the implant-supported restorations/prosthesis are part of a quality management concept for maintenance.
         a. Are you satisfied with the way your implant(s) function(s)?
         b. Are you satisfied with the appearance of your implant(s)?
      3. **Questions Regarding Patient-Perceived Changes Since Last Appointment**
         a. Do you feel like any part of the implant(s) is/are loose?
         b. Do the gums around your implant(s) bleed?
         c. Do you notice a bad taste coming from your implant(s)?
         d. Have you noticed any changes in your implant(s)?

b. **The Dental Implant Maintenance Visit**
   1. Modern dental implants may be difficult to recognize intraorally because their restorations often have the same appearance as the crowns and fixed bridges used to restore natural teeth (**Figure 8-32**). For this reason, dental implants should be clearly noted in the chart so that all dental team members are alerted to the exact location of the dental implant in the patient's mouth. Also, the patient's radiographs should be updated annually and reviewed before the start of periodontal instrumentation.
   2. Each maintenance visit should be individualized based on previous examinations and history; evaluation of peri-implant tissue health; examination of prosthesis/abutment components; evaluation of implant stability; occlusal examination; assessment of patient's self-care; radiographic examination; and treatment (e.g., periodontal instrumentation).

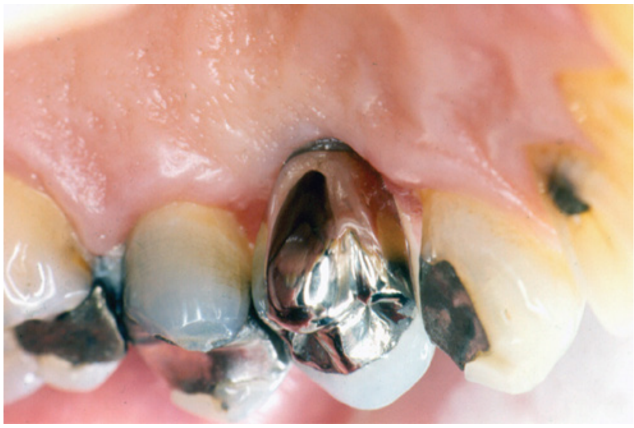


Figure 8-32 **Fixed Prosthetic Crown.** The first premolar in this photo is a prosthetic crown supported by a dental implant. During an intraoral examination, it would be difficult to distinguish between a crown that is supported by a natural tooth and a crown supported by a dental implant. For this reason, the location of a dental implant should be noted on the patient's chart.

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2. **Guidelines for Professional Recall and Maintenance**
   a. **Maintenance Frequency**
      1. Patients with implant-supported restorations—fixed or removable—should be advised to obtain a professional dental examination at least every 6 months as a lifelong regimen.[49] Maintenance intervals should be determined on an individual basis because there is a lack of data detailing precise intervals.
         a. A 3-month maintenance interval is usually appropriate for the first year following restoration of the implant. The clinician, however, should use their best clinical judgement to tailor the recall interval on a case-by-case basis that accounts for an individual patient's compliance level, periodontal health, and overall systemic health.
         b. After the initial 12-month period, a 3- to 6-month maintenance interval may be used.[50] Periodontal maintenance appointments should be scheduled as frequently as necessary to keep the periodontium and peri-implant tissues healthy.
            1. Patients categorized at higher risk based on age, ability to perform oral self-care, or biological or mechanical complications of implant-borne restorations should be advised to obtain a professional examination more often than every 6 months.[49]
            2. The risk factors identified for each patient are essential in the estimation of the prognosis for the implants, and these factors should be included in the determination of the appropriate maintenance interval. The following are indications for more frequent maintenance intervals.
               a. **Reduced Bone Support Around Implants.** Reduced bone support indicates that close monitoring of bone support is needed or the dental implant might be lost.
               b. **Inflammation.** A patient who has signs of inflammation around implants, even in the presence of good biofilm control, needs more frequent maintenance visits.
               c. **Host Response.** Systemic conditions or diseases, such as diabetes, may affect the host–bacterial interaction. A shorter maintenance interval is needed for these patients.
   b. **Guidelines for Professional Maintenance.** A panel composed of experts appointed by the American College of Prosthodontists (ACP), American Dental Association (ADA), Academy of General Dentistry (AGD), and the American Dental Hygienists Association (ADHA) met to establish new clinical practice guidelines for recall and maintenance of patients with implant-borne dental restorations.[49] These guidelines for professional maintenance are summarized in **Table 8-2**.

Table 8-2 Guidelines for Recall and Maintenance of Dental Implants

Patient recall	Patients with implant-supported restorations—fixed or removable—should be advised to obtain a professional dental examination at least every 6 months as a lifelong regimen.
Professional maintenance	Meticulous patient self-care is an important factor for long-term implant health; instruction in self-care techniques for natural teeth, implant-supported restorations, or implant abutments should be part of every maintenance visit. ■ Dental professionals should recommend appropriate oral topical agents and self-care aids suitable for the patient's at-home maintenance needs. ■ Chlorhexidine gluconate is the oral topical agent of choice when an antimicrobial agent is indicated. ■ Dental professionals should use periodontal instruments compatible with the type and material of the implants, abutments, and restorations and powered instruments such as glycine powder air polishing.

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1. Previous research indicated that scratching of the implant surface was a significant cause of implant failure. This belief led to a recommendation that only plastic and graphite instruments be used on titanium surfaces. However, exclusive use of polymer instruments is no longer recommended for several reasons:
    a. Investigation of failing implants shows that residue left on the implant body following the use of a polymer scaling instrument may play a role in eventual implant failure. Residues may include cement, calculus deposits, or debris left from forceful use of polymer (plastic, graphite, resin) instruments.
    b. Modern implant material, itself, is very rough and porous. The implant companies make rough, porous implant material for better osseointegration of the implant to the bone. Rough surfaces are very common on portions or all of the implant body. These surfaces are usually intended to be adjacent to bone and not exposed to the oral environment. If an intentionally rough surface becomes exposed to the oral cavity—due to pocket formation or recession of the gingival margin—it needs to be instrumented as part of implant maintenance.
2. Removal of subgingival cement or calculus deposits requires a slender instrument working-end made of a metal compatible with the implant—in other words, a titanium instrument (**Figure 8-33**).

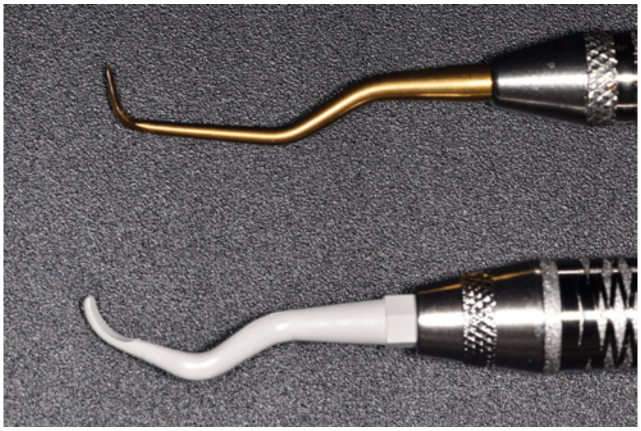

Figure 8-33 Implant Curettes. Titanium implant curet (top). Polymer implant curet (bottom).

3. The new guidelines from the American College of Prosthodontics state that implants should be instrumented with like metals—titanium-on-titanium—so as to not leave any residue behind.[49]
    a. Standard dental restorative materials, for example, gold and porcelain, can be cleaned with conventional periodontal instruments. Care must be taken, however, not to use these instruments apical to the margin of the prosthetic crown where titanium might be contacted.
    b. Titanium scaling instruments are recommended for removal of subgingival calculus deposits and residual dental cement. The working-ends of titanium instruments are slender and have cutting edges that can be sharpened. The working-ends of polymer instruments are bulky and not well suited for subgingival instrumentation.
    c. Residual dental cement is a frequent finding when restorations have been cemented to abutments. Cement deposits are typically located subgingivally. Some of the advanced cement formulations used today strongly adhere to the surface of the implant, which makes removal very difficult. If removal efforts are not successful, surgical access is highly recommended.

c. **Special Considerations for Polishing**
    1. Implants, abutments, and components do not require routine polishing.
        a. When indicated, polishing of the implant restoration can be accomplished with rubber cups and nonabrasive polishing paste or by supragingival air polishing.[51,52]
        b. Polishing has been shown to improve titanium surfaces that have previously been roughened or scratched. However, if no surface alterations are noted, the titanium surfaces should not be polished.
        c. A systematic review by Louropoulou et al.[52] indicates that for rough implant surfaces, air abrasives are the instruments of choice, if surface integrity needs to be maintained. Air polishing with glycine powder may be considered a better method to remove plaque biofilm from dental implants because glycine is less aggressive than sodium bicarbonate powder.[52]
    2. Air polishing with glycine powder for the removal of biofilm from titanium surfaces is a relatively new technology with clinical research supporting its safety and efficacy for use in the treatment of peri-implant disease. Glycine-based powders are biocompatible and gentle on the soft tissues of the oral cavity and subgingival epithelium.[53–55]

3. **Patient Self-Care of Dental Implants**
    a. **Considerations**
        1. Meticulous self-care is of the utmost importance in preventing peri-implant disease. An individualized self-care routine should be developed for each patient.
        2. Some patients undergoing implant therapy may have had a long history of dental neglect and/or poor plaque biofilm control.
        3. The dental hygienist can assist the patient in maintaining dental implants by providing self-care education appropriate for implants and home care devices that are effective and simple to use.
    b. **Care of Fixed Prosthetic Restorations**
        1. A fixed prosthetic crown is an artificial tooth that fits over the abutment. These are made from a variety of routinely encountered restorative materials.
        2. **Self-Care Challenges**
            a. The single tooth prosthesis (crown) can present a challenge for biofilm control in that the patient may quickly begin to regard it to be just the same as a natural tooth.
                1. In fact, these restorations often have different designs and unusual contours that require focused self-care attention. The better the patient understands the implant design and contours that must be cleaned on a daily basis, the more effective oral hygiene will be.
                2. Self-care practices must be modified to include aids that can effectively clean the altered morphology of the peri-implant region.
            b. The crown covering the implant abutment is larger in circumference than the abutment and will have contours added to it to make it look and function like a natural tooth.
                1. The "bulky" contours of the crown may contact the tissue and then "dip in" to meet the abutment at or below the gingival margin.
                2. Dental floss or tufted dental floss should be adapted along the margin of the crown and then pulled gently back and forth to direct it into the sulcus and around the abutment (**Figure 8-34**).

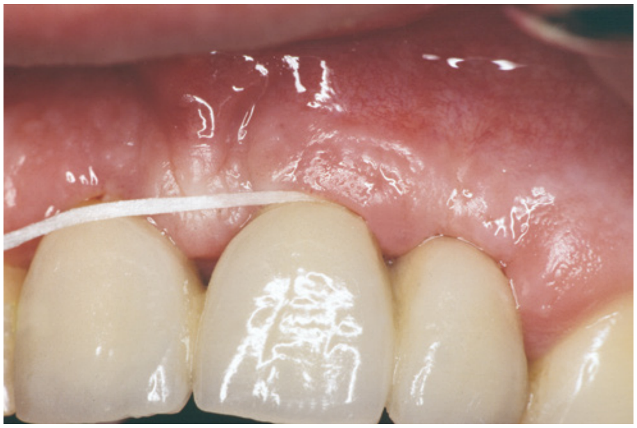

Figure 8-34 **Implant Self-Care.** Dental floss is used to clean a single implant with a prosthetic crown. The "bulky" contours of the crown may contact the tissue and then "dip in" to meet the abutment at or below the gingival margin.

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c. In some cases, the restoration may appear very similar to a fixed bridge (e.g., a pontic supported by two dental implants). Tufted dental floss, specially designed interdental brushes, and water flossers are effective cleaning devices for deplaquing the large embrasure spaces and getting into the sulci of these fixed bridge–type restorations.

d. Restoration of multiple tooth replacement situations can involve complex, denture-like prostheses that are attached to multiple implants and that are not removable by the patient.

   1. These prostheses can be removed by either the dentist or specialist, at which time cleaning access is optimal.
   2. Daily care by the patient requires an individualized plan and a combination of devices to overcome the limited access often encountered.

3. **Techniques and Devices**

   a. Standard multi-tufted, soft nylon bristle, manual toothbrushes can be used effectively by patients having sufficient dexterity and understanding of the task.

   b. Powered toothbrushes are safe for titanium surfaces; these devices are particularly effective for implant patients in general.

   c. Interdental brushes can be effective in biofilm removal and may effectively clean the peri-implant sulcus.[56]

      1. Note that interdental brushes used to clean implants should have a soft protective coating on the twisted wire that secures the bristles.
      2. The use of a standard interdental brush (that does not have a plastic or nylon coating on the twisted wire) should be avoided because the exposed metal wire could scratch the titanium surface.

   d. Dental floss can be used with dental implants. Patients must be instructed to gently use floss subgingivally. Flosses with expanded spongy or fluffy sections along with a stiffer, floss threader section (tufted floss) are particularly helpful.

   e. Oral irrigators may be safely used to remove plaque biofilm from dental implants.

      1. A study by Magnuson et al.[57] compared the efficacy of a manual toothbrush paired with either traditional dental floss or a water flosser. The study results demonstrate that the water flossing group had statistically significantly greater reduction in bleeding than the dental floss group. The authors conclude that water flossing may be a useful adjunct to implant maintenance.
      2. Patients should be instructed to use the lowest setting and direct the flow through the interdental contacts or perpendicular to the implants rather than directly into the implant sulcus.

   f. Daily use of an antimicrobial mouth rinse is beneficial for many patients.[49] The entire mouth can be rinsed twice daily after brushing. Or, if that is not acceptable to the patient, the mouth rinse can be applied topically to each implant area twice daily with a cotton-tipped applicator or with gauze.

c. **Care of a Removable Prosthesis**

1. An implant-supported removable prosthesis is similar to a traditional full denture except that, in the case of implants, it is attached to the abutments by devices such as O-rings, magnets, or clips (**Figure 8-35**).

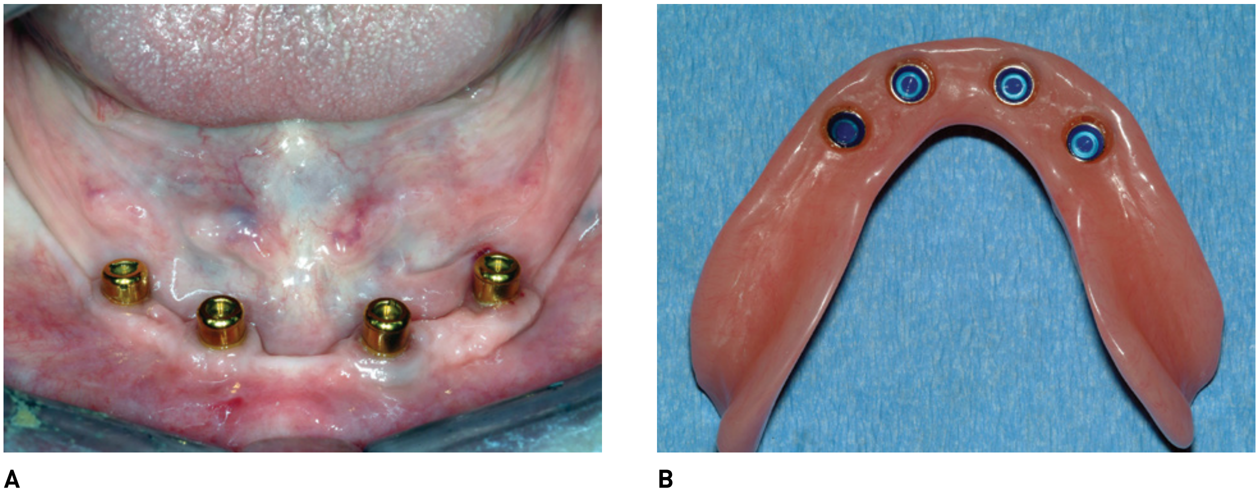

Figure 8-35 Implant-Supported Removable Prosthesis. Abutment posts on the mandibular arch. An implant supported removable prosthesis is attached to the abutment posts by O-rings, magnets, or clips.

Courtesy of Rodger A. Lawton, DMD, FACP, Olympia, WA.

   a. This type of prosthesis can also be designed as an implant- and tissue-supported prosthesis or as an overdenture.
   b. The patient can remove the prosthesis to clean it, the attachment devices, the abutments, and the remainder of the mouth.

2. **Techniques and Tools**
   a. Selection of cleaning devices for the abutments that support a removable prosthesis should be based on the knowledge that most implant components are made from titanium.
   b. Cleaning devices must be selected for ease of use by each individual patient, and the recommendation of fewer, rather than more, devices is best. The cleaning aids that are recommended should be demonstrated for the patient and then evaluated by the hygienist to verify their effective use by the patient.
   c. In some cases, a metal bar connects the abutments and is used to attach the prosthetic denture in the mouth. Tufted dental floss or an unfolded 2 × 2 gauze square can be useful in cleaning underneath the metal bar and around the abutments (**Figure 8-36**).

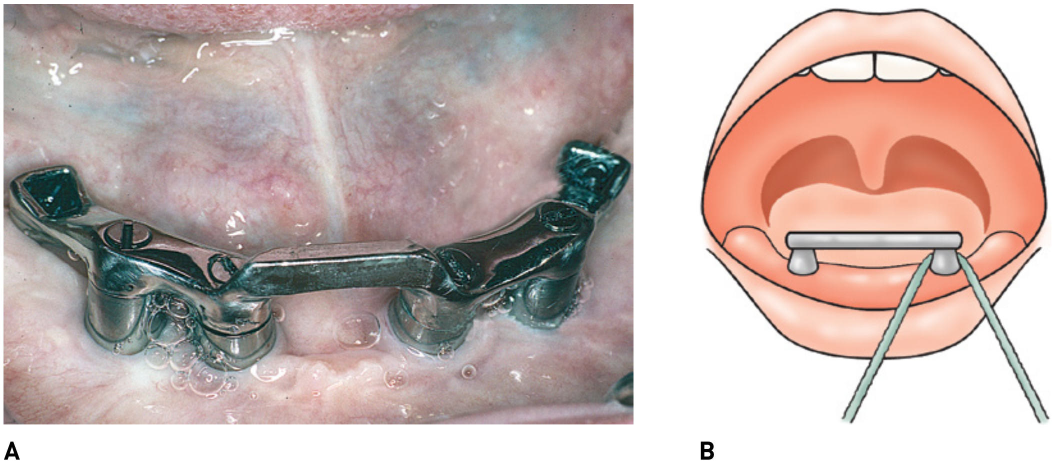

Figure 8-36 Abutments Joined by Metal Bar. Tufted dental floss is useful in cleaning the implant abutments and underneath the connecting metal bar.

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Chapter Summary Statement

As dental implant therapy becomes more common, dental hygienists will be providing care for increasing numbers of patients with implants. At the same time, it is inevitable that dental hygienists will encounter a significantly higher number of cases with implant-related complications, such as peri-implant diseases or biomechanical overloading. The primary goals of treatment of implant-related complications are (1) to arrest further pathologic breakdown of peri-implant tissues and (2) to maintain the implant in function with healthy peri-implant tissues. To be able to accomplish this, the dental hygienist must be able to distinguish peri-implant health from implant-related complications.

The evolution of peri-implant mucositis to peri-implantitis may be gradual and slow. It is therefore of utmost importance that the dental professional be able to recognize the early signs of disease, then take active countermeasures before it further jeopardizes implant survival. With individualized regular maintenance, cases of peri-implant diseases can be avoided with the close collaboration and effective communication between the patient and the dental practitioner. Professional maintenance is the most important step in not only detecting the early signs of peri-implant diseases, but, more important, maintaining oral health. Thus, along with determining effective treatment strategies and scheduling options on a case-by-case basis, dental hygienists play an crucial role in educating patients about the importance of meticulous self-care.

Controlling the biomechanical forces applied to the implant is crucial to preserving the peri-implant bone levels and maintaining osseointegration. Biomechanical overload imposes excessive occlusal forces upon the dental implant which exceed the tolerance of the peri-implant bone. Therefore, every effort should be made to control and monitor biomechanical forces throughout the lifetime of the implant.

Implant restorations necessitate customized self-care instructions and devices. Implant maintenance appointments should include monitoring of plaque biofilm levels, examination of soft tissues, assessment of the restorative integrity, reinforcement of patient self-care measures, periodontal instrumentation of implant abutments and prostheses, and radiographic examination.

Section 6

Focus on Patients

Clinical Patient Care

CASE 1

A patient returns to you for his regular 3-month periodic recall. He is completely edentulous and wears an implant-supported maxillary denture and an implant-supported mandibular denture. **Figures 8-37** and **8-38** are the clinical photograph and the radiograph of the implants.

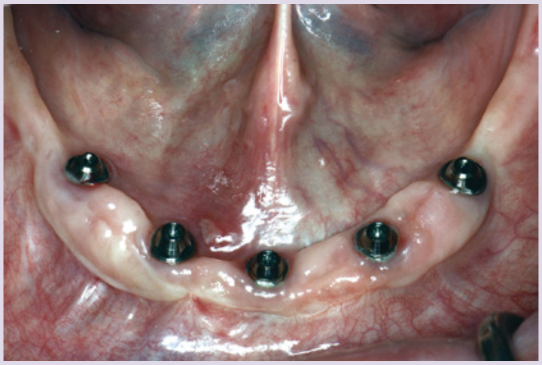

Figure 8-37 Clinical Photograph: Case 1.

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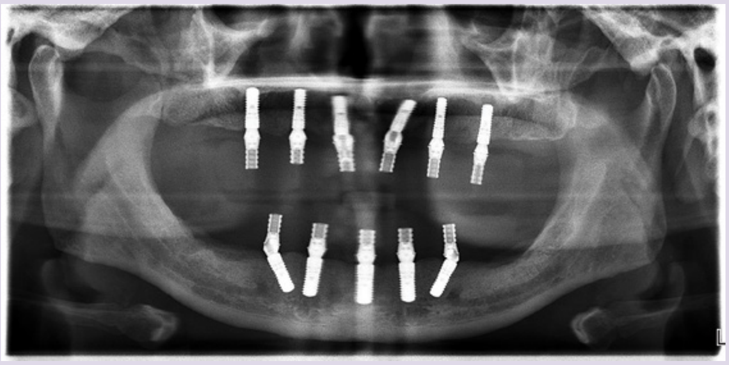

Figure 8-38 Radiograph: Case 1.

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Based on the patient's presentation, what would be the peri-implant diagnosis? What steps would you take to perform a thorough and complete peri-implant evaluation? What type of home care recommendations would you give to the patient? (Assume that the clinical and radiographic presentation of the peri-implant tissues in the mandibular arch is representative of the maxillary implants.)

CASE 2

While you are performing nonsurgical therapy on a periodontitis patient, the patient tells you that he is thinking about having all his teeth removed, because they are not healthy anyway, and just having some implants placed. He tells you that this would be easier, because he would not have to worry about the implants like he does his teeth. How should you respond?

CASE 3

You are scheduled to record the information needed to make a periodontal diagnosis for a patient new to your dental team. Radiographs have not yet been ordered for the patient. As you begin your probing, the patient informs you that she has two dental implants. Visual examination of the patient's dentition does not immediately reveal which teeth are replaced by the implants. How should you proceed?

CASE 4

At a maintenance visit for one of your team's patients you note obvious mobility of a crown supported by an implant. How should you (the dental hygienist) proceed?

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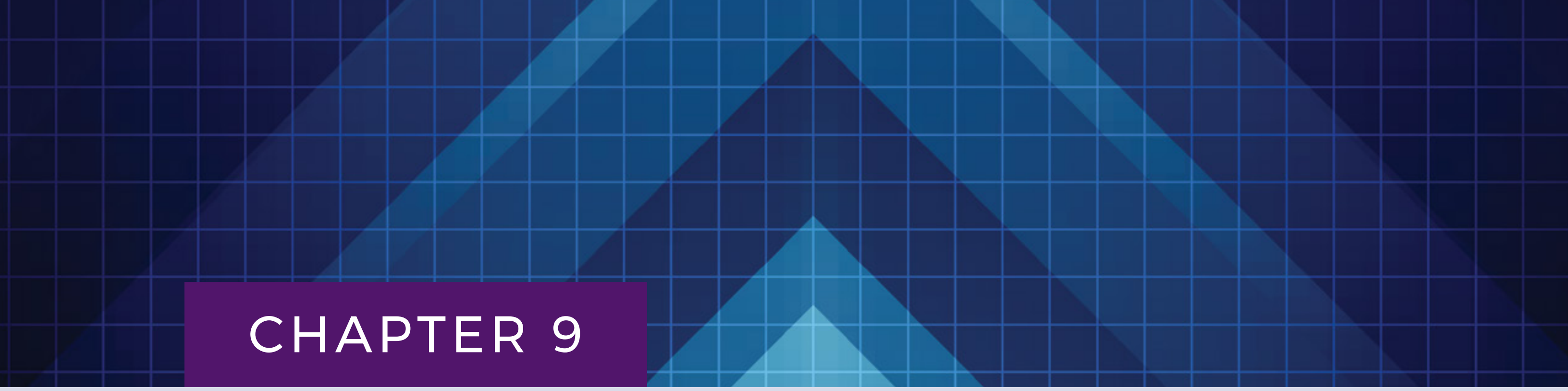

CHAPTER 9

Acute Periodontal Diseases

- Section 1 Introduction to Acute Periodontal Diseases
- Section 2 Abscesses of the Periodontium
- Section 3 Endodontic-Periodontal Lesions
- Section 4 Necrotizing Periodontal Disease
- Section 5 Primary Herpetic Gingivostomatitis
- Section 6 Focus on Patients
    - Clinical Patient Care
    - Evidence in Action
    - Ethical Dilemma

CLINICAL APPLICATION

Some periodontal conditions strike with such sudden onset and elicit such intense gingival pain that it compels a patient to seek immediate emergency care from a dental provider. These disorders fall under the umbrella of *acute periodontal diseases*. As one of the first members of the dental team to possibly confront acute periodontal diseases, dental hygienists will be at the forefront of promptly addressing the patient's immediate chief complaint. Dental hygienists must therefore be able to recognize the signs and symptoms pathognomonic of acute periodontal diseases and be armed with a clear understanding of how to manage and treat such conditions.

LEARNING OBJECTIVES

- Define the term *acute periodontal disease* and list the conditions that fall under this category.
- Describe the general characteristics of each of the acute periodontal diseases discussed in this chapter.
- Name and describe the three types of abscesses of the periodontium.
- List the possible causes of abscesses of the periodontium.
- Compare and contrast the periodontal and pulpal abscesses.
- Outline the typical treatment steps for a gingival abscess, a periodontal abscess, and a pericoronal abscess.
- Describe the possible outcomes of an untreated pericoronal abscess.
- Describe the pathogenesis of an endodontic-periodontal lesion.
- List the different categories of endodontic-periodontal lesions based on the signs and symptoms.
- Describe the characteristics of necrotizing gingivitis, necrotizing periodontitis, and necrotizing stomatitis.
- Outline the typical treatment steps for necrotizing gingivitis.
- Compare and contrast the tissue destruction that occurs in necrotizing gingivitis and necrotizing periodontitis.
- Compare and contrast the tissue destruction in periodontitis versus that seen in necrotizing periodontitis.
- Describe the symptoms of primary herpetic gingivostomatitis.
- List a step-by-step treatment plan to address a patient suffering from herpetic gingivostomatitis.

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KEY TERMS

Acute periodontal diseases
Abscess of the periodontium
Pus
Suppuration
Circumscribed
Pulpal abscess
Acute abscess
Chronic abscess
Gingival abscess
Periodontal abscess
Pericoronal abscess
Pericoronitis
Operculum
Trismus
Endodontic-periodontal lesion
Necrotizing periodontal disease (NPD)
Necrotizing gingivitis
Necrosis
Punched-out papillae
Pseudomembrane
Necrotizing periodontitis
Sequestrum
Necrotizing stomatitis
Primary herpetic gingivostomatitis

Section 1

Introduction to Acute Periodontal Diseases

1. Most periodontal diseases are chronic in nature and progress rather slowly. If left untreated, the disease can take years or decades to destroy the periodontium and lead to tooth loss. These diseases are rarely painful. In contrast, some periodontal conditions strike with such sudden intensity that it compels a patient to seek emergency care for immediate relief. The emergency conditions described in this chapter are known as *acute periodontal diseases*.
    a. The American Academy of Periodontology defines the term **acute periodontal diseases** as any periodontal condition that has a sudden onset and rapid progression accompanied by gingival pain and discomfort[1] (**Box 9-1**).
    b. All members of the dental team should be on alert for these conditions because their prompt recognition and early intervention can limit subsequent permanent damage to the periodontium, preserve functionality of the involved tooth (or teeth), and provide immediate pain relief to the patient. If an acute periodontal disease is not promptly diagnosed and managed, then the gingival pain and discomfort will continue unabated, and the prognosis of the affected tooth may worsen. Additionally, systemic dissemination (spread) of oral pathogens to distant sites in the body, as a result of transient bacteremia, is always a possibility.
    c. This chapter examines four periodontal disorders—abscesses of the periodontium, endodontic-periodontal lesions, necrotizing periodontal disease, and herpetic gingivostomatitis—that fall under the category of acute periodontal diseases and reviews their typical manifestations, along with possible treatment strategies to manage these conditions.

Box 9-1 Characteristics of Acute Periodontal Diseases

- Sudden onset of the condition.
- Rapid course of progression.
- Accompanied by pain and discomfort.
- May be unrelated to a preexisting gingivitis or periodontitis.
- Intraorally, the acute periodontal lesion may affect a localized site or it may be more widespread in the mouth.
- May present with systemic involvement.

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Section 2

Abscesses of the Periodontium

1. **Overview of Abscesses of the Periodontium**
    a. **Abscesses of the Periodontium**
        1. An **abscess of the periodontium** is a circumscribed, fluctuant collection of pus localized within the gingival wall of the periodontal pocket (**Figure 9-1**). It is one of the more common acute dental conditions that require immediate attention due to its rapid destruction of the periodontium.
        2. **Pus** (also known as purulence) is a whitish-yellowish milky-like exudate that consists primarily of dead and dying neutrophils, bacteria, cellular debris, and fluid leaked from blood vessels. Pus is produced by the body as the host defense mechanisms are activated in an attempt to combat an infection. The process whereby tissues and inflammatory exudates are liquified by the action of the offending microorganisms is known as **suppuration**. **Figure 9-2** shows suppuration expressed from an abscess of the periodontium.
        3. Abscesses of the periodontium are usually described as being circumscribed. The term **circumscribed** means that the abscess is localized or confined to a specific site (e.g. the facial surface of a single tooth or perhaps the gingival margin on a specific tooth). **Figure 9-3** shows a circumscribed abscess.

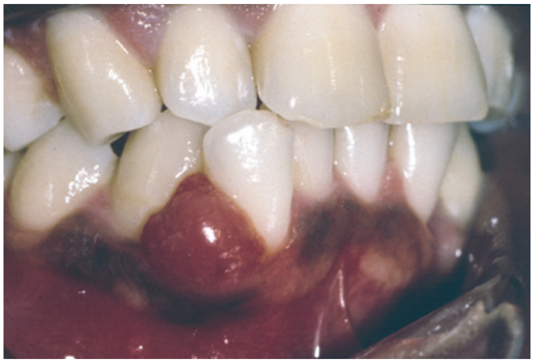

Figure 9-1 Abscess of the Periodontium. Note the localized swelling between the mandibular right canine and lateral incisor caused by the accumulation of pus beneath the epidermis. When palpated, an abscess feels soft and fluid filled.

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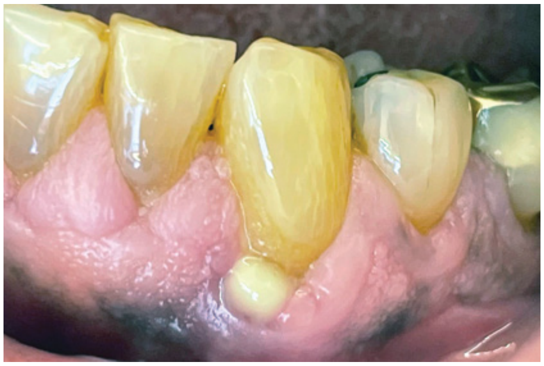

Figure 9-2 Suppuration. Note the thick, yellowish-whitish fluid oozing from the gingival margin. Pus can either spontaneously discharge or it can be drawn out by gently applying light digital pressure over the abscess to "milk" it. In some cases, the dental practitioner may need to make a small incision through the abscess to create an exit point for the pus to drain.

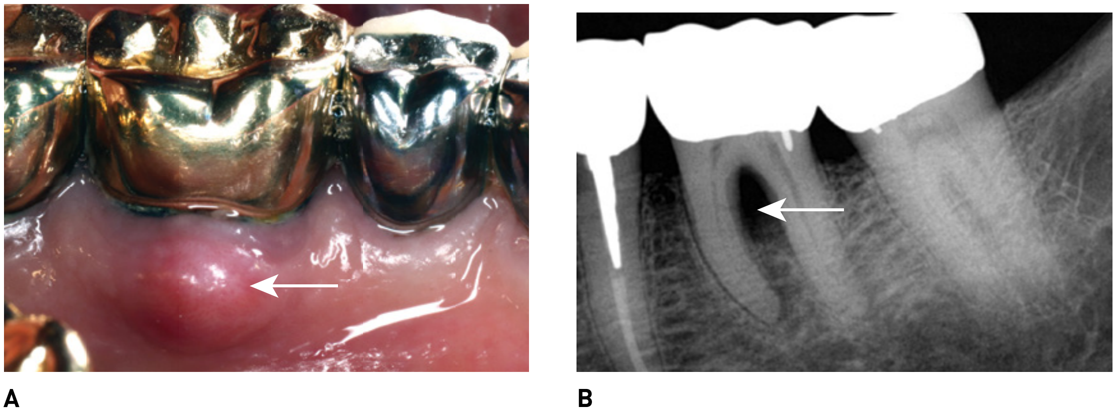

Figure 9-3 Abscess of the Periodontium. A. Abscess of the periodontium associated with the mandibular first molar (white arrow). Clinical exam reveals an isolated 9-mm pocket at the mid-lingual site; endodontic evaluation reveals a healthy, normal pulp with an absence of periapical pathology. **B**. Radiograph of the same tooth. Note the extensive furcal bone loss due to the pathologic destruction caused by the abscess.

Courtesy of Dr. Erik Rose, Indianapolis, IN.

4. The precise bacterial etiology of the abscess of the periodontium is not clear, but it is known that most of these lesions contain microflora that are predominantly Gram negative and anaerobic. Most studies indicate that the bacteria in these abscesses are similar to bacteria seen in periodontitis patients with deeper pockets.

b. **Implications of Untreated Abscesses**
   1. Periodontal abscesses represent a common dental emergency. In a North American study examining the number of various types of dental emergencies encountered in a dental private practice, approximately 14% of all emergencies were attributed to periodontal abscesses.[2] Likewise, in the United Kingdom, periodontal abscesses amounted to be the third most prevalent dental emergency treated in a private-practice setting.[3]
   2. If not addressed promptly, abscesses of the periodontium can initiate rapid periodontal destruction which can lead to premature tooth loss. Taking this into account, early detection and prompt management of the abscess may offer the tooth a fighting chance to survive.
   3. A number of case reports have shown a possible link between abscesses of the periodontium and systemic diseases. This is based on emerging evidence that incriminates oral microorganisms as possibly playing a role in the development of systemic conditions such as brain abscess, cervical necrotizing fasciitis, total knee arthroplasty infection, and respiratory distress. As such, oral pathogens can be suspected as possibly having an impact well beyond the oral cavity.[4–7] Thus, controlling acute conditions such as periodontal abscesses can possibly ameliorate systemic disorders.

c. **Characteristics of an Abscess of the Periodontium**
   1. Typical patient complaints related to an abscess of the periodontium are outlined in **Box 9-2**.
      a. Pain resulting from an abscess of the periodontium is usually described by the patient as a constant pain (as opposed to intermittent). Patients frequently report that the pain is easy for them to localize (i.e., the patient can point to the exact spot that hurts). Note, however, that in some cases a patient can report pain that is not at all localized to a specific location.
      b. In addition to pain and swelling, the patient may report difficulty in mastication and may report a foul taste in the mouth.
   2. Oral examination will usually reveal the presence of a circumscribed swelling of the soft tissue. This swelling may involve the gingiva only, or it may involve both the gingiva and the mucosa.
   3. In many cases, teeth with an abscess of the periodontium can exhibit a temporary increase in mobility.
   4. Dental radiographs of a tooth with an abscess of the periodontium frequently reveal alveolar bone loss in the area of the abscess, but the bone loss does not usually involve the tooth apex (unlike a pulpal abscess). **Figures** 9-3 and **9-4** are cases of an abscess where the accompanying bone loss does not extend to the root apex.
      a. Alveolar bone loss resulting from an abscess of the periodontium can occur extremely rapidly when compared with the rate of alveolar bone loss usually associated with periodontitis.
      b. Although a dental radiograph of an abscess of the periodontium may reveal alveolar bone loss, in a periodontitis patient it is not always possible to tell what part of the missing bone actually resulted from the acute infection and what part of the missing bone was caused by periodontitis that was already present before the abscess formed.

Box 9-2 Characteristics of an Abscess of the Periodontium

- An acute abscess is characterized by constant and localized pain.
- Typically, a chronic abscess is characterized by no pain. However, in rare cases, the chronic abscess may be characterized by dull pain.
- Circumscribed (localized) swelling in the periodontium.
- Possible increase in tooth mobility.
- Radiographic loss of alveolar bone not involving the tooth apex.
- Tooth usually has a vital pulp.

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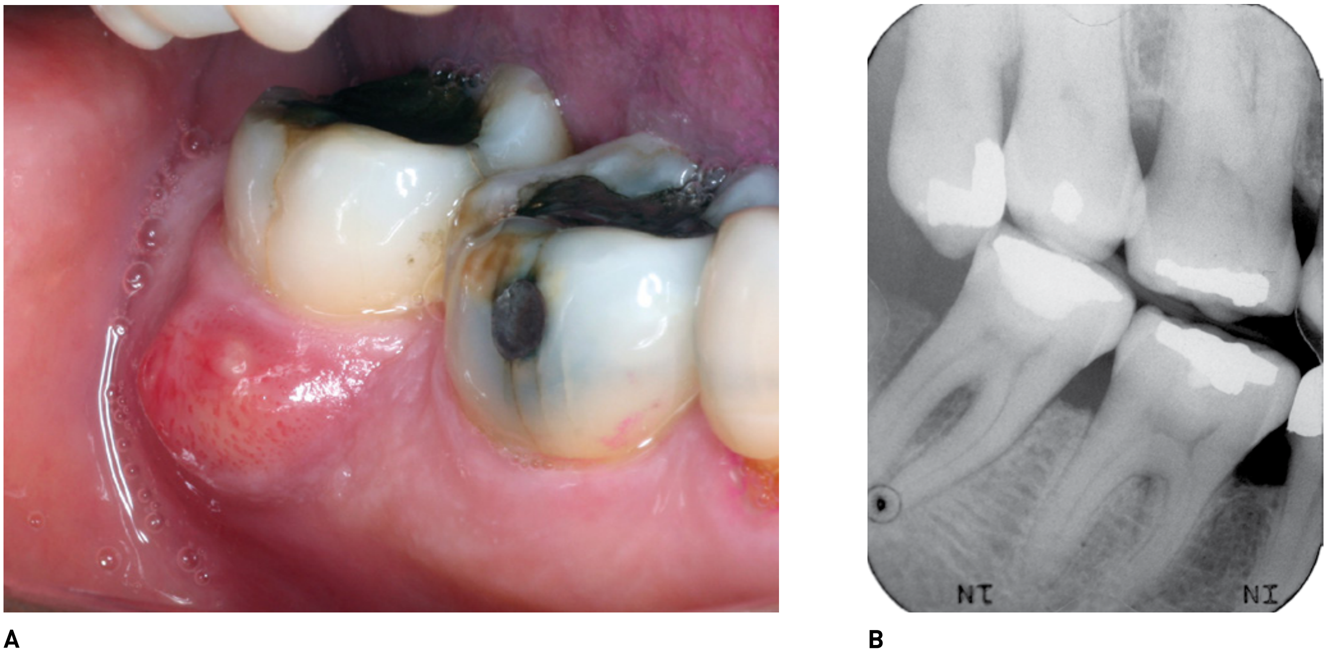

Figure 9-4 **Abscess of the Periodontium. A.** The clinical photograph shows circumscribed swelling and inflammation of the gingival tissue on the buccal aspect of a mandibular second molar, which is indicative of an abscess of the periodontium. **B.** The accompanying radiograph reveals furcal bone loss approximating the location of the abscess.

Courtesy of Dr. Richard J. Foster, Educational Solutions and Design, Greensboro, NC.

5. Teeth affected by an abscess of the periodontium are usually vital (have healthy pulp tissue) and respond positively if pulpal sensibility testing (like electronic pulp testing and thermal sensitivity testing) is performed.
6. Another possible clinical sign of an abscess of the periodontium can be an elevated body temperature (fever). An elevated body temperature would not normally be present unless the infection (from the abscess of the periodontium) spreads throughout the body. This would represent a serious sign, if present. Because many abscesses of the periodontium are circumscribed (localized), they frequently are not associated with an elevated body temperature.
7. When there is delay in treating an abscess of the periodontium, additional oral changes may occur.[8] The collection of pus can break through the surface tissues by draining through a sinus tract. This establishes a path of drainage for the pus on its own. **Figure 9-5** shows an abscess of the periodontium that has drained spontaneously by breaking through the surface tissues.

d. **Causes of Abscesses of the Periodontium.** Theories about the etiology of the abscess of the periodontium vary, but most investigators attribute formation of this type of abscess to one of the following scenarios.[9]

   1. **Blockage of the Orifice of a Pocket.** Blockage of the orifice (or opening) of a preexisting periodontal pocket has been suggested as a cause of some abscesses of the periodontium. Most periodontal pockets have readily accessible openings that give easy access to a periodontal probe. Some authors have theorized that in certain instances the opening of a periodontal pocket can become restricted in size or may become completely occluded because of temporary improvement of the surface tissue tone.[10,11] This improvement of tissue firmness could result in trapping bacteria and fluids in a preexisting periodontal pocket, leading to an abscess that begins within this existing periodontal pocket.

    2. **Accidentally Forcing a Foreign Object into the Tissues.** It has also been suggested that an abscess of periodontium can be caused by accidentally forcing and lodging a foreign object into the supporting tissues of a tooth.
        a. A variety of foreign objects have been implicated in the formation of some abscesses of the periodontium. For example, an abscess could result from a patient accidentally puncturing the gingiva with a toothpick, forcing bacteria into the tissue.
        b. Another common event that can result in an abscess of the periodontium is accidentally forcing some food product like a husk from a kernel of popcorn or a peanut skin into the tissues associated with the tissue inflammation as part of a periodontal pocket. As a result, the foreign object would act as a physical barrier that blocks the orifice of the pocket.
    3. **Incomplete Calculus Removal in a Periodontal Pocket.** Incomplete calculus removal in a periodontal pocket has also been suggested as a cause of an abscess of the periodontium.
        a. When this occurs, it is usually thought to be in a site with a very deep pocket where the calculus deposits are removed only in the most coronal aspects of the pocket near the gingival margin, but the calculus deposits deeper in the pocket are not completely removed because of difficulty of access for instrumentation of the tooth surface.
        b. It is theorized that removal of the more coronal calculus deposits allows the gingival margin to heal somewhat and to tighten around the tooth, like a drawstring of a pouch, preventing drainage of bacterial toxins and other waste products from the pocket.[12] Bacteria remaining in the deeper aspects of the periodontal pocket could result in the formation of an abscess of the periodontium.

2. **Comparison of Periodontal Abscesses and Pulpal Abscesses.** The clinical signs and symptoms of a periodontal abscess may be perplexing because of the many overlapping signs and symptoms with a pulpal (endodontic) abscess (also known as an acute apical abscess).
    a. Abscesses affecting the tissues around a tooth can originate from two different sources: (1) the periodontium itself (periodontal abscess) or (2) the pulpal tissues that are within the pulp chamber of the tooth (pulpal abscess).
        1. It is helpful for the dental hygienist to be familiar with the characteristics of these two types of abscesses because the periodontal abscess and the pulpal abscess have many similar, overlapping signs and symptoms.
        2. The characteristics of each type of abscess are outlined in **Table 9-1**.
    b. As already discussed, a periodontal abscess is an abscess that results from an acute infection of the periodontium.
    c. In contrast, a **pulpal abscess** is an inflammatory reaction to pulpal infection (pulpitis) or pulpal necrosis. Clinically, a pulpal abscess is characterized by rapid onset, spontaneous tooth pain, percussion sensitivity, swelling, and pus formation (**Figure 9-6**). Because the infection from the abscess spreads apically to affect the periapical tissues, a periapical radiolucency at the apex of the involved tooth is apparent (**Figure 9-7**).

Table 9-1 Periodontal Versus Pulpal Abscesses

Characteristic	Periodontal Abscess	Pulpal Abscess
Vitality test results	Usually, vital pulp	Usually, nonvital pulp
Radiographic appearance	Bone loss present as an angular defect and/or furcation radiolucency	Bone loss at tooth root apex
Symptoms	Localized, constant pain	Difficult to localize, intermittent pain

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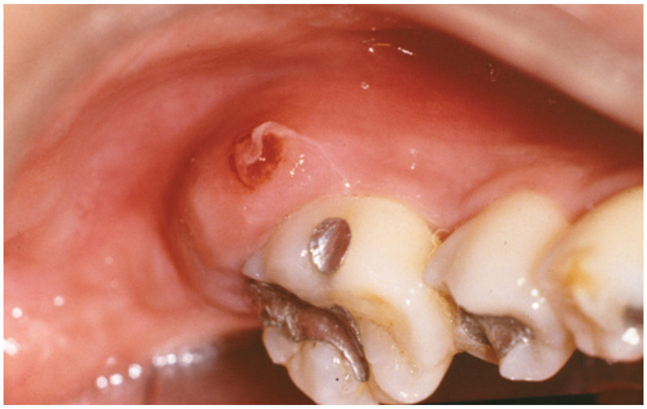

Figure 9-5 **Path of Drainage.** The abscess of the periodontium shown in this figure has broken through the surface tissues, establishing a path of drainage for the pus on its own.

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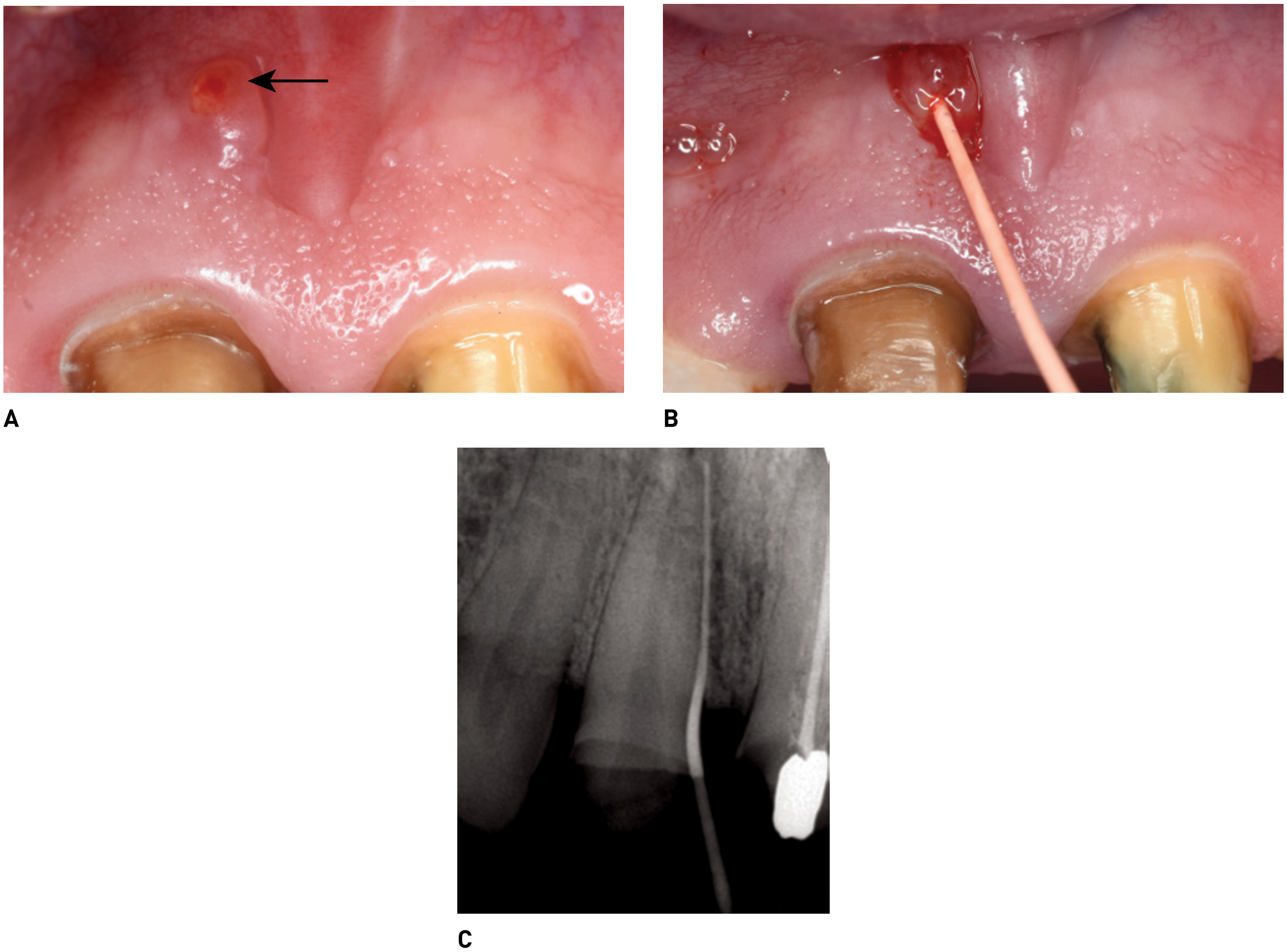

Figure 9-6 **Pulpal Abscess. A.** Patient presents with the chief complaint, "I have a tender bump on my gums." Visual examination reveals a 2 × 3 mm sessile circumscribed lesion at the mucogingival junction (arrow). Periodontal probing of the right central incisor ranges from 1 to 2 mm with an absence of bleeding upon probing; however, endodontic evaluation reveals atypical responses to pulp sensibility tests. (Note that provisional crowns on both central incisors were removed prior to taking the photographs to examine the status of the clinical crowns.) **B.** A radiopaque gutta percha cone is gently inserted into the abscess to trace the path of the sinus until resistance is felt (known as intraoral sinus tracing). **C.** Radiographic view of the gutta percha cone shows the termination of the sinus tract at the root apex, thus indicating that the source of the abscess has an endodontic origin, not a periodontal one.

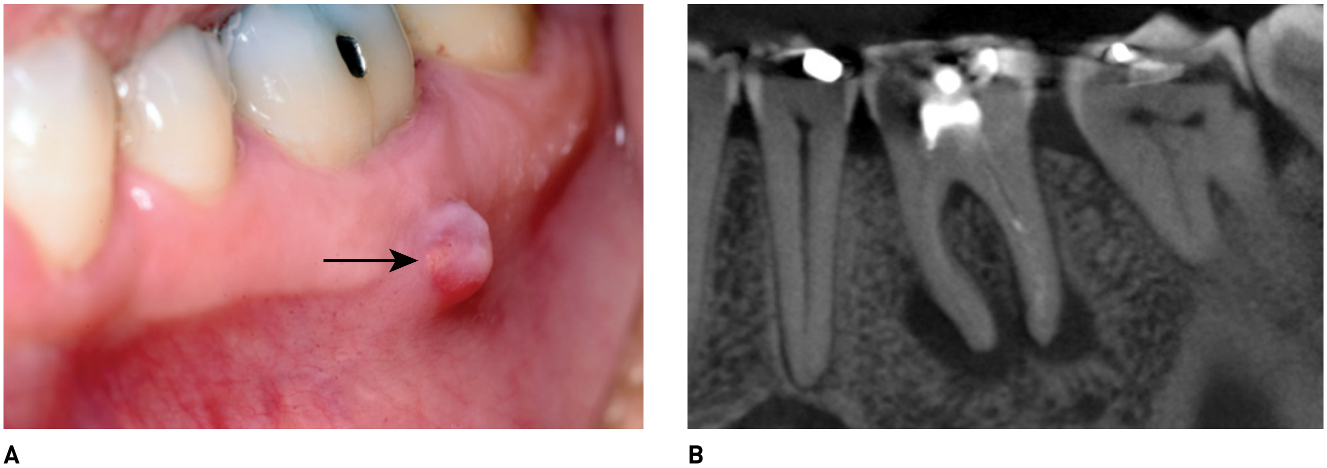

Figure 9-7 Pulpal Abscess. Patient presents with the chief complaint, "There's a painful gumboil right here!" (arrow). **A.** Examination reveals a 3 × 3 mm sessile, fluid-filled, circumscribed abscess at the mucogingival junction of the mandibular first molar. Periodontal assessments are consistent with a healthy periodontium. Endodontic evaluation reveals an abnormal, altered response to pulp sensibility tests; the dental history reveals that endodontic treatment was initiated over 5 years ago but never completed. **B.** Radiographic image shows deep radiographic decay extending into the pulp chamber, furcation invasion, and periapical radiolucencies affecting both the mesial and distal roots. Note normal crestal bone levels.

3. **Classification of Abscesses of the Periodontium**
    a. **Classification by Course of the Lesion.** One way to classify abscesses of the periodontium is according to the *course (i.e., progression) of the lesion.*
        1. An **acute abscess** has a rapid onset that is characterized by pain and discomfort. It is primarily caused by an exacerbation of a chronic inflammatory periodontal lesion.
            a. Contributing factors to the etiology of an acute abscess may be the lack of spontaneous drainage or inadequate host response.
            b. Unlike a chronic abscess (which will be discussed below), an acute abscess is symptomatic because, as it grows, the pressure rapidly builds up within the lesion and has nowhere to escape. As the pressure builds, the patient will experience mild-to-severe pain, which is a distinguishing clinical characteristic of an acute abscess.
        2. In contrast, a **chronic abscess** grows slowly and is not typically associated with pain. It forms after the spread of infection has been controlled by spontaneous drainage, host response, or therapy.
            a. In most cases, the pus from a chronic abscess is able to spontaneously drain through an abnormal "channel" (known as a sinus tract) in the periodontium and discharge out of the gingiva. Drainage of pus through a sinus tract will relieve the pain and discomfort associated with the pressure.
            b. As a result of the spontaneous drainage, some patients may not be aware that they have a chronic abscess. In rare cases, however, members of the dental team should keep in mind that the chronic abscess can also be associated with dull pain.
    b. **Classification Based on Location.** A second way to classify abscesses of the periodontium is based on the *location* of the abscess: (1) the gingival abscess, (2) the periodontal abscess, and (3) the pericoronal abscess. But there is considerable overlap in this very loose classification system.
        1. **Gingival Abscess.** The **gingival abscess** refers to an abscess of the periodontium that is primarily limited to the gingival margin or to the interdental papilla without involvement of the deeper structures of the periodontium.
            a. The gingival abscess can occur in a previously periodontally healthy mouth when some foreign object is forced into a healthy gingival sulcus. An abscess of the periodontium that is limited to the gingival margin area can follow this traumatic event.
            b. **Figure 9-8** illustrates a typical gingival abscess where the swelling is limited to the marginal gingiva of a single tooth.

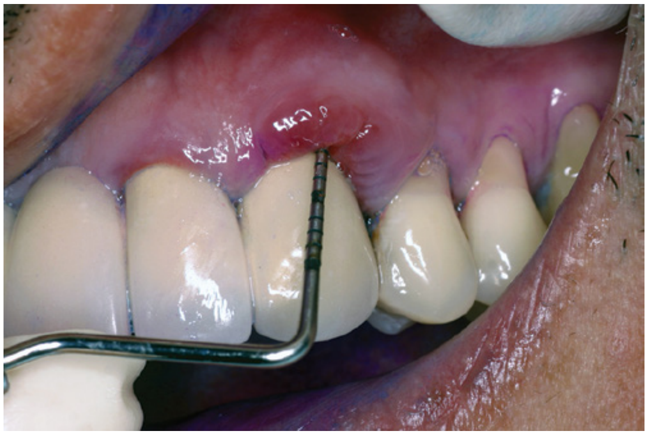

Figure 9-8 **Gingival Abscess.** Note that this abscess is limited to the gingival margin on the facial surface of this maxillary canine.

Courtesy of Dr. Richard J. Foster, Educational Solutions and Design, Greensboro, NC.

2. **Periodontal Abscess.** The true **periodontal abscess** refers to an abscess of the periodontium that affects the deeper structures of the periodontium as well as the gingival tissues. A periodontal abscess usually occurs in a site with preexisting periodontal disease including preexisting periodontal pockets and usually affects the deeper structures of the periodontium and is not limited to the gingiva only.
3. **Pericoronal Abscess.** The **pericoronal abscess** is an abscess of the periodontium that involves tissues around the crown of a partially erupted tooth (*peri* = "around"; *coronal* = "crown"). The associated soft tissue inflammation associated with the pericoronal abscess is referred to as **pericoronitis**.
    a. A pericoronal abscess is seen in teeth where some of the soft tissues surrounding the teeth actually cover part of the occlusal surface of the teeth. **Figure 9-9** shows a patient with a pericoronal abscess under a soft tissue flap partially covering a third molar tooth.
    b. The pericoronal abscess (or pericoronitis) is an inflammatory process most frequently seen around the crowns of partially erupted or fully erupted mandibular third molar teeth. Because many third molar teeth have inadequate space to erupt fully, a flap of gingival tissue covering part of the occlusal surface, known as an **operculum**, can form.
    c. Cleaning under an operculum can be a challenge for both the clinician and the patient. This makes the operculum a potential nidus of plaque accumulation that may trigger the host inflammatory response and eventually lead to an abscess (hence, the name of this condition—pericoronal abscess). Because of this, some authors refer to the pericoronal abscess as an operculitis.
    d. The operculum can be thought of as a "lid" of soft tissue that entraps plaque biofilm and food debris. Because of the difficulty of keeping the area clean, the operculum leads to soft tissue inflammation, which is a characteristic of pericoronitis.
    e. *Streptococci milleri* group bacteria, well known for their ability to cause suppurative infections, are most likely involved in the pathogenesis of acute severe pericoronitis of the lower third molar.[13]
    f. The signs and symptoms of the pericoronal abscess are discussed below and outlined in **Box 9-3**.
        1. Pain is common with the pericoronal abscess. The pain can arise from the tissue swelling itself, but pain can also arise when an opposing tooth occludes with the infected, swollen operculum.
        2. Soft tissue swelling (edema) and redness (erythema) also usually accompany the pericoronal abscess.
        3. As damage to tissue covering the partially erupted tooth progresses and the tissue swelling increases, the opposing tooth can frequently be seen to impinge (press) on the swollen tissue, creating additional tissue trauma and additional patient discomfort.
        4. Limited mouth opening, known as **trismus**, can be seen in some cases of advanced pericoronal abscess. Trismus is a painful condition that restricts normal mandibular movement and function as a result of a low-grade infection.
        5. Elevated body temperature (fever) and swollen lymph nodes (lymphadenopathy) also can be seen in advanced cases of pericoronitis.

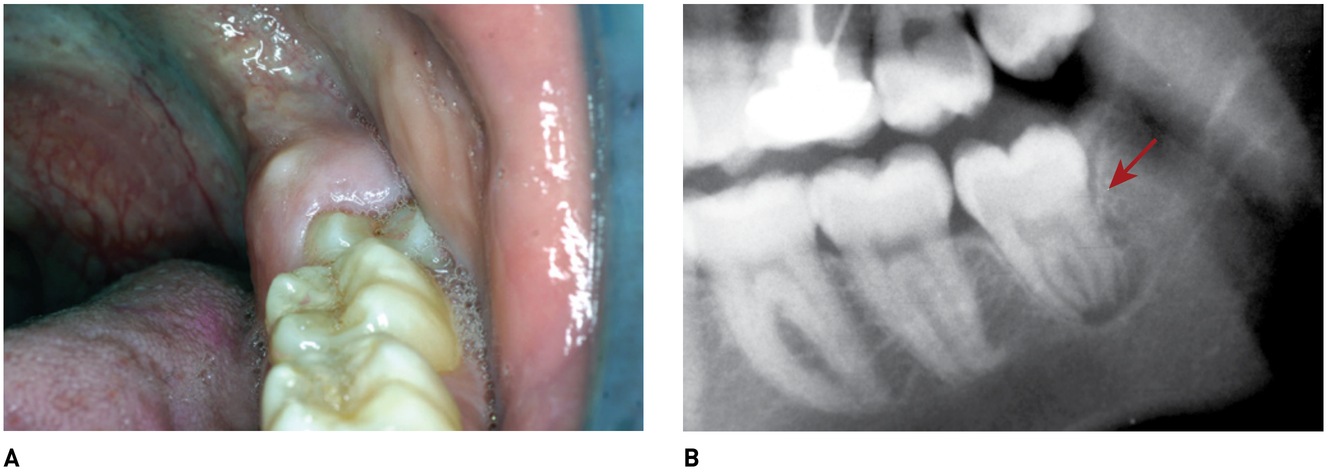

Figure 9-9 Pericoronal Abscess Involving a Mandibular Third Molar. A. The clinical photograph shows the typical appearance of a partially exposed mandibular third molar covered by an operculum. **B.** The accompanying radiograph shows the position of the third molar in close relationship to the mandibular ramus. Note the J-shaped radioluceny running down the the distal root of the molar which is a sign of the periodontal destruction caused by the pericoronal abscess (red arrow).

Courtesy of Dr. Richard J. Foster, Educational Solutions and Design, Greensboro, NC.

Box 9-3 Signs and Symptoms of a Pericoronal Abscess

- Pain at the site
- Swelling of operculum (operculitis)
- Possible trismus (limited mouth opening)
- Possible elevated body temperature
- Possible lymphadenopathy

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4. **Management of Patients with Abscesses of the Periodontium**
   a. The treatment of a patient with either a gingival or a periodontal abscess is similar. Fundamental treatment steps include (1) establishment of a path of drainage for the pus, (2) thorough periodontal instrumentation of the affected tooth surfaces in the area of the abscess, and (3) pain relief.
   b. Steps commonly followed in treatment of patients with a gingival or a periodontal abscess are discussed below and are outlined in **Box 9-4**.

Box 9-4 Steps in Treatment of a Gingival or Periodontal Abscess

- Administer local anesthesia.
- Drain pus.
- Perform thorough periodontal instrumentation.
- Adjust occlusion, if needed.
- Prescribe antibiotics, if needed.
- Recommend warm saline rinses.
- Prescribe pain medications, if needed.
- Establish follow-up appointments.

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1. It is normally necessary to anesthetize the site to be treated, because manipulation of the tissues involved by an abscess can be quite uncomfortable.
2. Drainage of the pus from the abscess is critical. The abscess can be drained either through the pocket itself or by performing periodontal surgery (as discussed in Chapter 30). When drainage is established through the pocket, the toe of a *sterile* curette is used to puncture the soft tissue wall of the pocket to allow the drainage. In some cases, drainage can be accomplished by externally incising through the fluctuant surface of the abscess with a surgical scalpel blade and draining the purulent discharge with light digital pressure. This type of procedure is known as an incise and drain procedure.
3. Since one of the possible etiologies of a gingival or periodontal abscess is incomplete calculus removal, thorough periodontal instrumentation of the affected tooth surfaces in the site of the abscess is important in bringing these types of abscesses under control.
4. Some adjustment of the tooth occlusion is usually also indicated since inflammation resulting from the abscess can force a tooth to extrude slightly from its socket, leading to trauma from occlusion and pain when masticating.
5. In more advanced cases of abscesses, antibiotics may also be needed, as with any other serious oral infection.
6. Some clinicians recommend using warm saline (saltwater) rinses several times each day to help keep the abscess draining until it has healed completely.
7. A prescription for pain medication should always be considered, but over-the-counter pain medications, like nonsteroidal anti-inflammatory drugs (NSAIDs), can be adequate in many patients once the abscess has been drained.
8. Following emergency treatment of a patient with any abscess of the periodontium, the dental team should appoint the patient for a thorough periodontal assessment because abscess of the periodontium frequently reoccurs in a patient with a preexisting untreated periodontal disease. Thus, routine follow-up periodontal assessment is warranted.
9. If recurrent abscesses occur, the patient should be referred to a medical practitioner to be screened for diabetes. Diabetic patients are more prone to recurrent bouts of periodontal abscesses due to compromised wound healing and impaired immune function, which facilitates the persistent growth of pathogenic bacteria in the periodontal tissues and reoccurrence of the abscess.

c. **Treatment of a Pericoronal Abscess.** Treatment of patients with pericoronal abscess differs slightly from treatment of patients with other types of abscesses of the periodontium because of the difference in the anatomic location of these abscesses.

   1. Fundamental treatment steps for a patient with pericoronitis include (1) establishment of a path of drainage for the pus, (2) irrigation of the undersurface of the operculum, (3) thorough periodontal instrumentation of the tooth surfaces in the area of the abscess, and (4) relief of pain.[14]
   2. Steps commonly followed in treatment of patients with a pericoronal abscess are discussed below and outlined in **Box 9-5**.

Box 9-5 Common Steps in Treatment of Patient with Pericoronal Abscess

- Administer local anesthesia.
- Drain pus.
- Perform thorough periodontal instrumentation.
- Irrigate under operculum.
- Prescribe antibiotics, if needed.
- Recommend warm saline rinses.
- Prescribe pain medications, if needed.
- Evaluate for need for third molar extractions.
- Establish follow-up appointments.
- If pericoronal abscess reoccurs, reassess if surgical excision of the operculum or extraction of the offending tooth is warranted.

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a. It is normally necessary to anesthetize the site to be treated, because manipulation of the tissues affected by an abscess can be quite uncomfortable.
b. Drainage of the pus from the abscess is critical. The abscess can be drained through the pocket itself. Nonsurgical steps to treat a pericoronal abscess are outlined below.
    1. The patient should be adequately anesthetized.
    2. The clinician should use a periodontal probe to gently lift the soft tissue operculum off the occlusal surface of the affected tooth.
    3. The underlying debris, such as plaque and food particles, can now be removed with hand instruments and gentle irrigation with sterile saline.
    4. The toe of a sterile curette can be used to puncture the soft tissue wall of the pocket to allow drainage. Abscesses around the crown of a partially erupted third molar tooth may be difficult to drain because of the anatomy of the region.
    5. If this attempt fails to achieve adequate draining of the abscess, then periodontal surgery is indicated (as discussed in Chapter 30).
c. In more advanced cases of abscesses, antibiotics may be needed, as with any other serious oral infection.
d. Some clinicians recommend using warm saline (saltwater) rinses several times each day to help keep the abscess draining until it has healed completely.
e. A prescription for pain medication should always be considered, but over-the-counter pain medications (NSAIDs) can be adequate in many patients once the abscess is drained.
f. Following emergency treatment of a patient with any abscess of the periodontium, the dental team should appoint the patient for a thorough periodontal assessment, because the abscess of the periodontium frequently occurs in a patient with existing untreated periodontal disease.
g. In some cases, following resolution of the abscess, it may be prudent to surgically excise to prevent the recurrence of the pericoronal abscess. In some cases, following resolution of the abscess, the dentist may recommend extraction of malposed third molar teeth if there is inadequate jaw space for the third molar teeth to fully erupt or if the pericoronal abscess continually reoccurs.

Section 3

Endodontic-Periodontal Lesions (EPL)

1. An **endodontic-periodontal lesion** (formerly known as a combined periodontal-endodontic lesion) is a localized area of bacterial infection characterized by infection of both the dental pulp and periodontal tissues in the same tooth.
    a. It is a complex disorder because endodontic-periodontal lesions can originate from either the dental pulp or periodontal tissues, or both, making the origin of the infection difficult to determine.
    b. The pathways for the spread of bacteria between the dental pulp and the surrounding periodontal tissues may be:
        1. **Periodontally derived**—infection in the periodontal pocket enters the tooth via the accessory canals and/or apical foramen of the root and initiates inflammatory changes in the pulp–root canal complex.
        2. **Pulpally derived**—infection in the pulp–root canal complex escapes out of the root of the tooth (via the accessory canals and/or the apical foramen) to trigger a secondary infection of the periodontal tissues.
    c. Consequently, diagnosing an EPL poses a challenge for even the most competent dental practitioner. **Figure 9-10** diagrams various pathophysiologic routes of an EPL through both the endodontic and periodontal compartments.
2. **Signs and Symptoms**
    a. The two most common signs and symptoms of an EPL are (1) deep pocketing that extends close to the apex and (2) a negative or altered response to pulp sensibility tests (electronic and thermal testing).[15]
    b. Other signs and symptoms that may occur, but that are not always present, are bone resorption in the apical or furcal regions of a tooth, spontaneous pain, pain upon palpation or percussion, purulent suppuration, tooth mobility, presence of a sinus tract, and gingival color alterations.

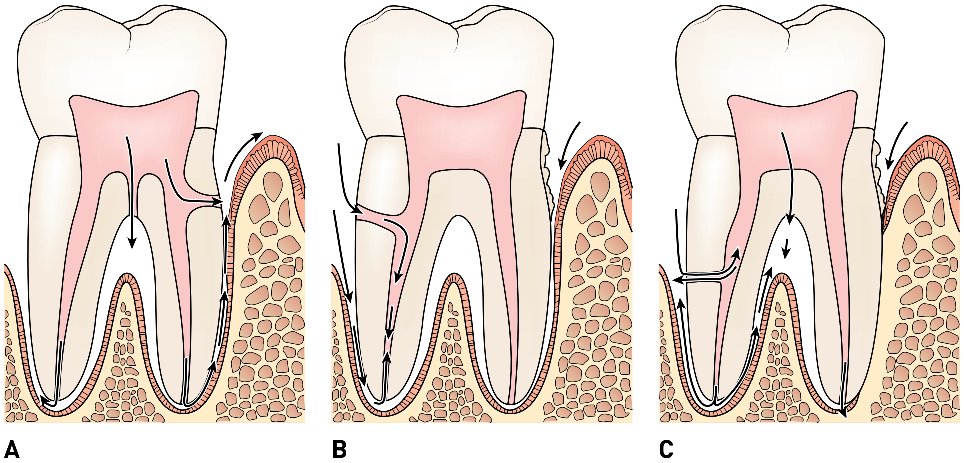

Figure 9-10 Various Routes of the Spread of an Endodontic-Periodontal Lesion. **A.** The endodontic-periodontal lesion originates from the pulp–root canal complex, then escapes through accessory canals in the furcation region or through the apical foramen to involve the periodontal tissues. **B.** The endodontic-periodontal lesion originates as periodontitis that progresses through the dentinal tubules and/or spreads to the apical region of the tooth to enter the tooth via the apical foramen, triggering an endodontic inflammatory response. **C.** Infection in the pulp–root canal complex joins an infected periodontal pocket progressing apically. These two infections occur simultaneously but form independently of each other until they join.

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Section 4

Necrotizing Periodontal Disease

Necrotizing periodontal disease (NPD) is a unique and unusual disease that does not share the typical manifestations of other periodontal diseases, such as gingivitis and periodontitis. Interdental tissue necrosis, intense gingival pain, and spontaneous gingival bleeding are distinct clinical characteristics of NPD. Other clinical characteristics, such as fetid breath, pseudomembrane formation, and systemic involvement (e.g. lymphadenopathy, fever, and malaise) are secondary clinical features of NPD, and may or may not be present in patients suffering from NPD.

Necrotizing gingivitis (NG), necrotizing periodontitis (NP), and necrotizing stomatitis (NS) are conditions that fall under the broad umbrella of NPD because evidence suggests that NG, NP, and NS represent *different stages of the same disease* with a similar etiology, clinical course, and treatment.[16,17]

1. **Necrotizing Gingivitis**
   a. **Overview of Necrotizing Gingivitis**
      1. **Necrotizing gingivitis** is a noncommunicable, destructive, inflammatory disease that is limited to the interdental and marginal gingiva.[17–19]
         a. Historically, this condition was called Vincent's infection, fusospirochetal gingivitis, trench mouth, ulceromembranous gingivitis, acute necrotizing ulcerative gingivitis (ANUG), or necrotizing ulcerative gingivitis (NUG). Many of these historical names are now considered misnomers and no longer valid for the following reasons:
            1. Ulceration is secondary to the distinguishing tissue.
            2. Because tissue necrosis is the hallmark of NPD,[20] the term *ulcerative* was stricken from the current disease nomenclature.
            3. A chronic form of this condition does not exist. Hence, the term *acute* was stricken from the name of this disease. Consequently, under the current nomenclature, this disorder is simply termed *necrotizing gingivitis*.

b. The distinctive characteristic feature of necrotizing gingivitis is tissue necrosis of the gingiva. The term *necrosis* refers to cell death. In the context of this disease, **necrosis** refers to the death of the gingival epithelial cells.

c. The progression of necrotizing gingivitis follows a predictable course. First, the interproximal gingiva necroses, which leads to the characteristic "punched-out" appearance. From there, the necrosis spreads to affect the marginal gingiva.

d. Necrotizing gingivitis is most often found in the mandibular anterior region.

b. **Etiology of Necrotizing Gingivitis**

1. In the late 19th century, Vincent used light microscopy to show the presence of spirochetes and fusiform deep within the tissues.[17] From this finding, Vincent concluded that the etiology of necrotizing gingivitis may be primarily due to an opportunistic infection of fusospirochetal microorganisms.
2. A subsequent electron microscopy study conducted by Listgarten in the mid-20th century confirmed the presence of spirochetal invasion deep within the tissues of necrotizing gingivitis lesions.[21]
3. Recently, more advanced microbial testing has revealed the presence of a diverse array of microorganisms deep within necrotizing gingivitis lesions that were not identified in previous investigations.
4. These microorganisms include *Treponema denticola*, *Fusobacterium nucleatum*, *Porphyromonas gingivalis*, *Tannerella forsythia*, and *Aggregatibacter actinomycetemcomitans*.[18]
5. Note that although these aforementioned microorganisms have been found in necrotizing gingivitis lesions, their presence within the tissues can be highly variable because they are not always present in all necrotizing gingivitis lesions. Thus, the exact microbial etiology of necrotizing gingivitis has never been established.
6. Further, there is still debate as to whether the invasion of these specific bacterial species occurs *before* or *after* the onset of necrotizing gingivitis. The bacterial invasion of certain species may either be the *cause* of the disease or the *consequence* of secondary bacterial colonization.
7. The inflammatory response appears to be a major contributing factor to the development of necrotizing gingivitis and can exacerbate the destructive effects of bacterial invasion.[21] This can be explained by the onset of necrotizing gingivitis which is histologically characterized by PMN infiltration of the affected tissue. This, in turn, leads to subsequent destruction of the soft tissue, probably due to the release of tissue destructive hydrolytic enzymes.[22] Conversely, the ensuing inflammatory response may also be impacted by a myriad of other factors that affect the immune system, such as stress, malnutrition, fatigue, psychosocial factors, chronic alcohol abuse consumption, poor oral hygiene, long-term drug abuse, and/or systemic disease which compromises the immune system, such as human immunodeficiency virus (HIV) or leukemia.
8. Compared to systemically healthy, non-HIV–infected individuals, HIV-positive individuals are more at risk of developing NPD. Thus, there is some evidence to suggest that NPD may be the first indication of HIV infection.[23]

c. **Prevalence of Necrotizing Gingivitis**

1. Necrotizing gingivitis can affect subjects of any age, but the highest prevalence is observed in individuals between 20 and 30 years of age.[24] Predisposing factors, such as stress and smoking, may account for why this age group is more susceptible to necrotizing gingivitis compared to other age cohorts.
2. In developed countries, the reported prevalence of necrotizing gingivitis is lower than that found in developing countries, especially among children and young people.[25,26] In a study assessing the periodontal status of 1,984 members of the U.S. military, 0.5% of the sample population were found to have necrotizing gingivitis. Similarly, in a sample population involving members of the British armed forces, the prevalence of necrotizing gingivitis was 0.11%.[27,28] In comparison, a Nigerian study showed a prevalence ranging from 1.7% to 15% in children of 2 to 6 years of age, whereas a Chilean study showed a prevalence of 6.7% among students.[29] Notably, *the higher prevalence found in the younger cohort in developing countries may be attributed more to malnutrition or an underlying systemic condition that may make them more susceptible to certain types of acute periodontal diseases.*

3. In North America, epidemiologic studies have shown that approximately 95% of all necrotizing gingivitis cases occur in Caucasian patients.[24,26,30]

d. **Characteristics of Necrotizing Gingivitis.** **Box 9-6** summarizes common characteristics associated with NG along with the prevalence of each of the signs and symptoms.

Box 9-6 Characteristics of Necrotizing Gingivitis

Primary Signs and Symptoms	Prevalence in Patients with Necrotizing Gingivitis
Necrosis and ulcers in the interdental papilla	94–100%
Gingival bleeding	95–100%
Pain	86–100%
Pseudomembrane formation	73–88%
Halitosis	84–97%
Lymphadenopathy	44–61%
Fever	20–39%

Data from Herrera D, Alonso B, De Arriba L. Acute periodontal lesions. *Periodontology. 2000;*65(1):149–177.

1. Patients with necrotizing gingivitis experience oral pain and frequently seek emergency care for that oral pain; the gingival ulceration associated with the infection results in exposure of connective tissue, which can be quite uncomfortable for the patients. Because of the exposure of connective tissue, spontaneous bleeding can occur.
2. The tissue necrosis that is characteristic of necrotizing gingivitis can lead to destruction of the interdental papillae, resulting in **punched-out papillae** (see **Figure 9-11**); the remaining gingival crater-like defects are normally covered by a collection of dead tissue cells and debris called a **pseudomembrane**.

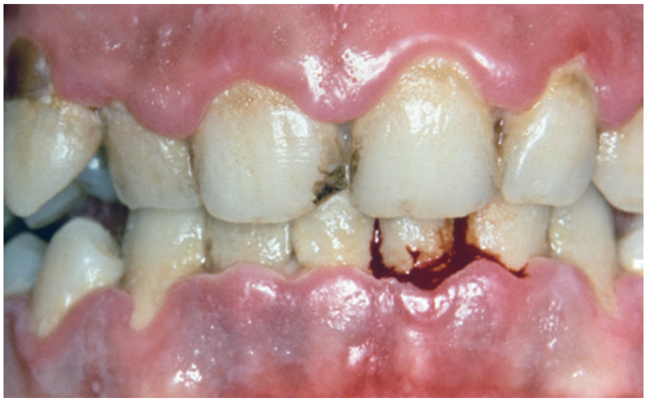

Figure 9-11 Punched-Out Papillae in Patient with Necrotizing Gingivitis. Note the punched-out appearance of the interdental papilla between the maxillary incisor teeth. Also note the spontaneous gingival bleeding from the mandibular incisor region.

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3. Grayish-white pseudomembrane overlying the necrotic punched-out interdental papilla and marginal gingiva is shown in **Figure 9-12**. In pathology, a pseudomembrane can be thought of as a false membrane that develops in the presence of inflammation and infection. The pseudomembrane can be easily peeled off with gentle traction, exposing the underlying necrotic, ulcerated surface (**Figure 9-13**).

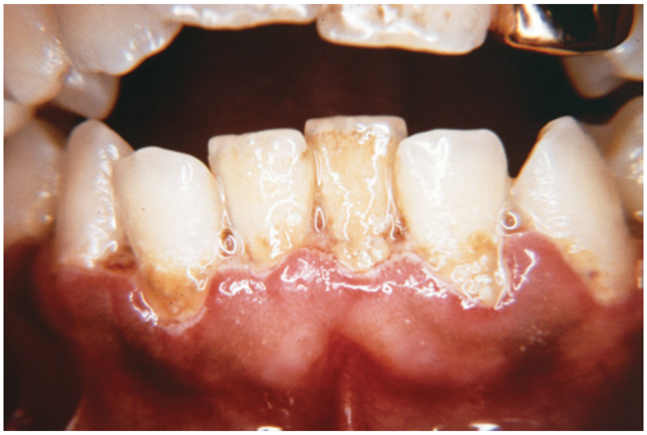

Figure 9-12 Pseudomembranous Tissue in Necrotizing Gingivitis. Note the grayish-whitish layer of pseudomembranous collar of tissue lining the marginal gingiva. In most cases, the punched-out papillae may be obscured by the pseudomembrane coating.

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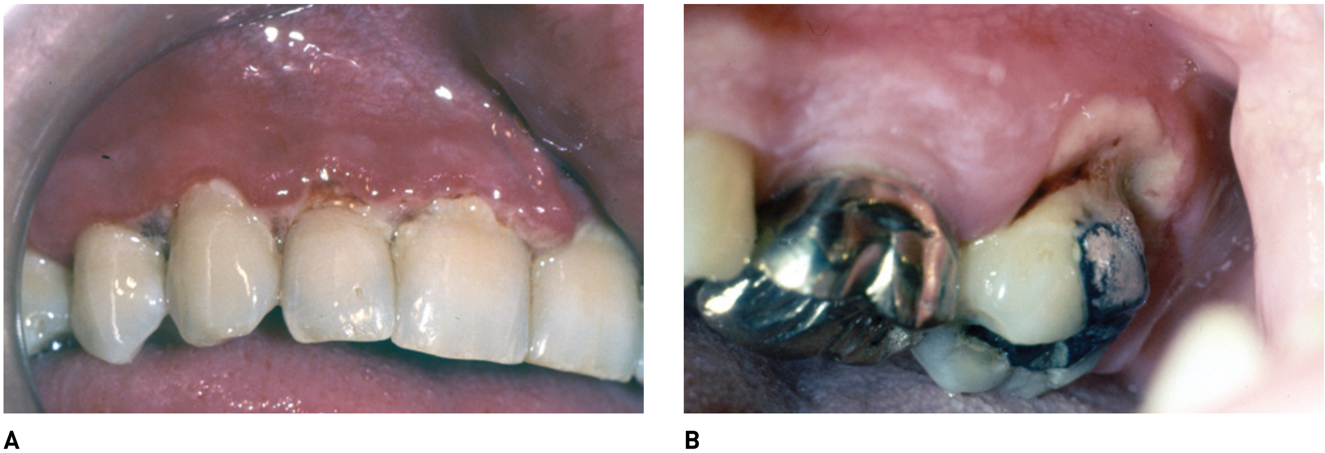

Figure 9-13 Post-Pseudomembranous Tissue Removal. Following removal of the pseudomembranous tissue, the ulcerated, necrotic papillae and gingival margins are more apparent from both photos.

Courtesy of Dr. Ralph Arnold, San Antonio, TX.

   a. Histologically, the pseudomembrane consists of dead cells, bacteria, and oral debris; right below the pseudomembrane is raw connective tissue that may be tender upon light palpation.
   b. Patients with necrotizing gingivitis may exhibit spontaneous gingival hemorrhage or pronounced bleeding that is elicited by the slightest provocation of the gingival tissues. This bleeding results from breakage of some of the tiny blood vessels in the underlying diseased connective tissues.
4. Patients with necrotizing gingivitis can present with swollen lymph nodes (lymphadenopathy), a vague feeling of discomfort (malaise), an elevated body temperature, sudden weight loss, fever, vomiting, and nausea. In some cases, the pain may be so debilitating that it may limit food and/or liquid intake.
5. Because of the necrosis (death) of cells, patients often have noticeable halitosis or malodor. Some authors have described this halitosis as a fetid odor.[17,31]

6. Certain associated behaviors and psychosocial conditions are frequently associated with patients who develop necrotizing gingivitis; these include a history of smoking, a history of poor nutrition, and a history of severe stress.
7. Because of its distinctive clinical features, a diagnosis of necrotizing gingivitis can be made simply based on clinical signs and symptoms.

e. **Typical Treatment Steps for Necrotizing Periodontal Disease.** Management of any form of NPD should focus on reducing patient pain/discomfort, arresting the destructive progression of the disease, restoring the form and function of the involved periodontal tissues, and preserving the stability of the periodontium following initial management.

1. To achieve these intended treatment outcomes, careful monitoring and treatment of the patient with necrotizing gingivitis should be done over several appointments. The following treatment sequence is recommended:
   a. Stage 1: Management of the acute signs and symptoms of the disease.
   b. Stage 2: Control of the preexisting condition(s).
   c. Stage 3: Corrective surgical treatment.
   d. Stage 4: Maintenance phase of treatment.
2. The key points of each stage are summarized below and outlined in **Box 9-7**. **Figure 9-14A** illustrates a case of a typical patient with early stages of necrotizing gingivitis. **Figure 9-14B** shows the same patient case following resolution of the acute phase and control of the underlying preexisting condition.

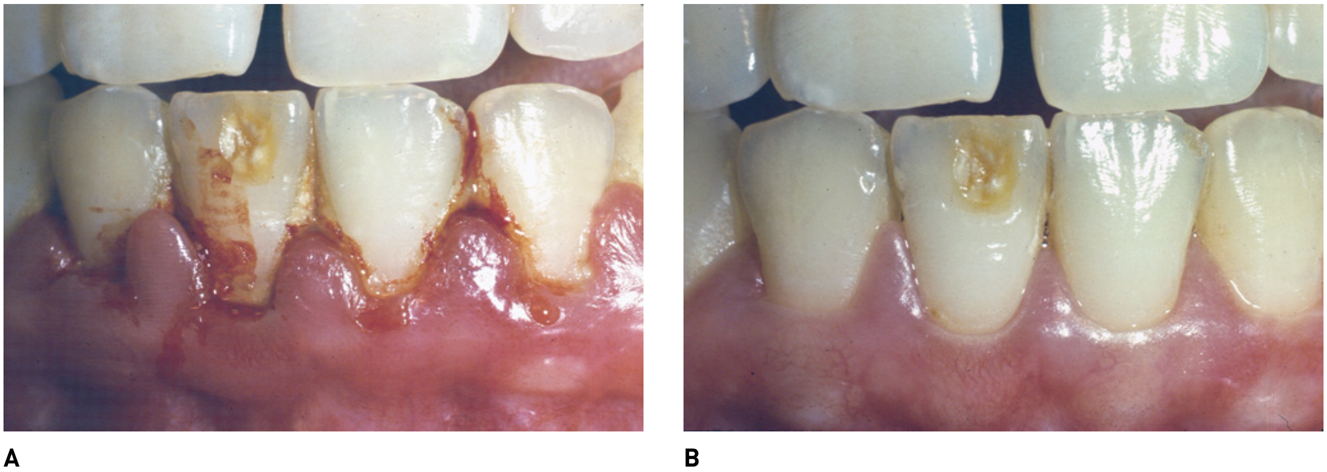

Figure 9-14 **Necrotizing Gingivitis. A.** Before treatment. **B.** Post treatment following resolution of the acute phase and control of the underlying condition.

Courtesy of Dr. Don Rolfs, Periodontal Foundations, Wenatchee, WA.

3. **Stage 1: Management of the Acute Stage**. As the initial stage of treatment, the clinician's primary goal should be to start the process of arresting the destructive nature of NPD and alleviating patient discomfort. So long as the acute phase persists, this stage of treatment may involve several appointments stretched out over several days (usually 2–5 days).
   a. On the first day of treatment (day 1), the clinician should:
      1. Carefully remove the pseudomembrane with irrigation and moist cotton.
      2. Gently perform supragingival periodontal instrumentation to remove the soft and mineralized deposits that have accumulated on day 1 of treatment. Ultrasonic instruments may be used in conjunction with hand instruments, applying minimal pressure over the ulcerated gingival tissues. However, the clinician should make sure that the patient is comfortable while the supragingival instrumentation is being rendered.
      3. Provide a self-care regimen that is specific to the needs of each NPD patient. The self-care regimen may involve gentle debridement with a toothbrush. Or, in some cases, the clinician may

        instruct a patient to limit mechanical oral hygiene measures because toothbrushing may impair tissue healing and induce even more gingival pain. To offset this, patients with necrotizing gingivitis may be instructed to use chemical plaque-control measures, such as 0.12% chlorhexidine gluconate mouth rinse or 3% hydrogen peroxide diluted in 1:1 warm water every 2–3 hours, to control plaque accumulation. This type of regimen contributes to mechanically cleansing the patient's oral cavity and provides an antibacterial effect of oxygen against anaerobic bacteria that may be associated with necrotizing gingivitis.[32]
        4. Counsel the patient to consume adequate amounts of fluids, get rest, and avoid excessive physical exertion and stress. The patient is also instructed to avoid tobacco and alcohol.
        5. For pain relief, the patient should be advised to use nonsteroidal anti-inflammatory agents.
        6. Some NPD patients may be unaware of their HIV status. In such a case, the patient should be counseled to seek medical evaluation for HIV screening as soon as possible.
    b. At the second visit (first follow-up appointment 2 days after initial visit):
        1. Subgingival periodontal instrumentation usually can be initiated at this appointment, provided that the patient's tolerance (to subgingival instrumentation) has improved.
        2. Further instruction in self-care should be reviewed, reevaluated, and reinforced at this visit.
        3. Attention should be paid to control the systemic predisposing factors associated with the onset of necrotizing gingivitis, such as enrolling the patient in a smoking cessation program or treating involved systemic conditions.
    c. At the third visit (second follow-up appointment approximately 5 days after initial visit).
        1. Subgingival instrumentation usually can be completed.
        2. Patient is evaluated for resolution of symptoms. Patient is further counseled on nutrition, smoking cessation, home care, and other factors that may have contributed to the onset of necrotizing gingivitis.
    d. In more severe cases of NPD that either fail to favorably respond to conventional mechanical therapy or have systemic involvement, systemic antibiotics may be needed. Note that local antibiotics are not recommended because the local drug may not achieve adequate subgingival concentration levels to have a significant antibacterial effect against the large concentration of bacteria present in the tissues.
4. **Stage 2: Control of the Preexisting Condition(s).** NPD often occurs over a preexisting gingivitis or periodontitis lesion. Thus, following resolution of the acute phase, the patient should be appointed for a comprehensive clinical assessment to identify and manage the underlying periodontal disease by performing more definitive treatment, such as prophylaxis or scaling and root planning, reinforcing oral hygiene, and addressing any other predisposing plaque-retentive local factors such as defective restorations or cavitated lesions.
5. **Stage 3: Corrective Surgical Management.** NPD often creates an altered gingival topography (e.g., gingival craters) that favor plaque retention and disease recurrence. Thus, in some cases, following complete resolution of the infection, some patients with necrotizing gingivitis may require periodontal surgery such as a gingivectomy to reestablish the natural contours of the gingiva.
6. **Stage 4: Maintenance Phase.** During the maintenance phase, the clinician should assess the periodontal status, reinforce self-care, control the predisposing factors, and perform the necessary periodontal instrumentation. The clinician should factor in patient compliance levels with health habits, psychosocial factors, and the interval of subsequent recall visits. Like the maintenance phase of treatment for gingivitis and periodontitis, the primary aim of this phase of treatment is to prevent NPD recurrence and monitor the patient's oral hygiene levels.

Box 9-7 Typical Treatment Steps for a Patient with Necrotizing Periodontal Disease

Stage 1: Management of the Acute Stage

- The pseudomembrane should be carefully removed.
- Supragingival periodontal instrumentation is performed. Instrumentation is limited because of the discomfort elicited by tissue manipulation.
- The patient is instructed regarding a gentle self-care regimen.

- A follow-up appointment is scheduled for 2 days after the initial visit.
    - Subgingival periodontal instrumentation usually can be begun at this appointment.
    - Further instruction in self-care should be included at this visit.

Stage 2: Control of the Preexisting Condition(s)

- Following the resolution of the infection, the patient should be appointed for a comprehensive clinical assessment to identify any underlying chronic periodontal disease.

Stage 3: Corrective Surgical Management

- Gingivectomy or a gingivoplasty can be done to reestablish a more harmonious and natural gingival contour that is more amenable to plaque control.

Stage 4: Supportive (Maintenance) Therapy

- After the disease has resolved, the patient should be enrolled in a supportive (maintenance) program. During each appointment, the clinician should assess the periodontal status, reinforce oral hygiene, control the predisposing factors, and perform the necessary periodontal instrumentation.

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2. **Necrotizing Periodontitis**
    a. **Overview of Necrotizing Periodontitis**
        1. **Necrotizing periodontitis** has the same clinical features as necrotizing gingivitis. However, the tissue necrosis observed in necrotizing periodontitis is not only confined to the interdental and marginal gingiva, but also spreads to affect the underlying periodontal attachment apparatus. If left untreated, this could potentially lead to bone loss and clinical attachment loss (**Figure 9-15**).
            a. The most distinguishing feature of necrotizing periodontitis is its destructive disease progression, which manifests as periodontal attachment loss and bone loss.[18] Necrotizing periodontitis may therefore be an extension of necrotizing gingivitis. As such, necrotizing periodontitis may occur in patients who previously had necrotizing gingivitis that was left untreated.
            b. One unusual finding in necrotizing periodontitis is that it can be accompanied by the formation of bone sequestra. A bony **sequestrum** (plural: sequestra) is defined as a piece necrotic (dead) bone that has been separated from the surrounding normal healthy bone. Sequestration is the process of forming a sequestrum. **Figure 9-16** illustrates a necrotizing periodontitis patient with a bony sequestration.

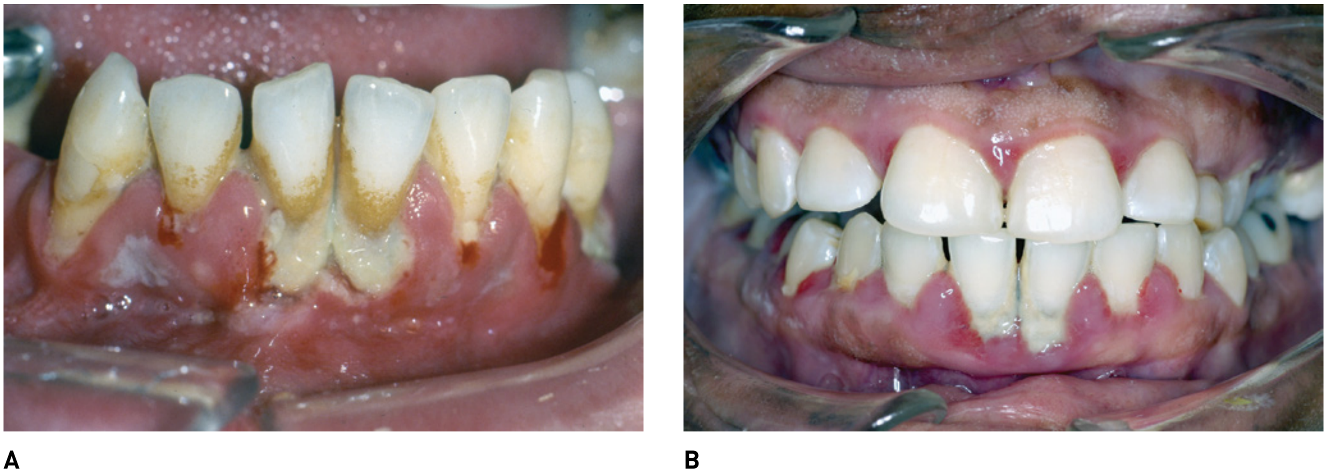

Figure 9-15 Necrotizing Periodontitis with Pseudomembranous Slough. Two cases of necrotizing periodontitis characterized by the appearance of a pseudomembranous slough that covers the crest of the interdental papilla and extends to the marginal gingiva of the lower central incisors. Note the clinical attachment loss in both cases.

A: Courtesy of Dr. Ralph Arnold, San Antonio, TX; **B:** Courtesy of Dr. Richard J. Foster, Educational Solutions and Design, Greensboro, NC.

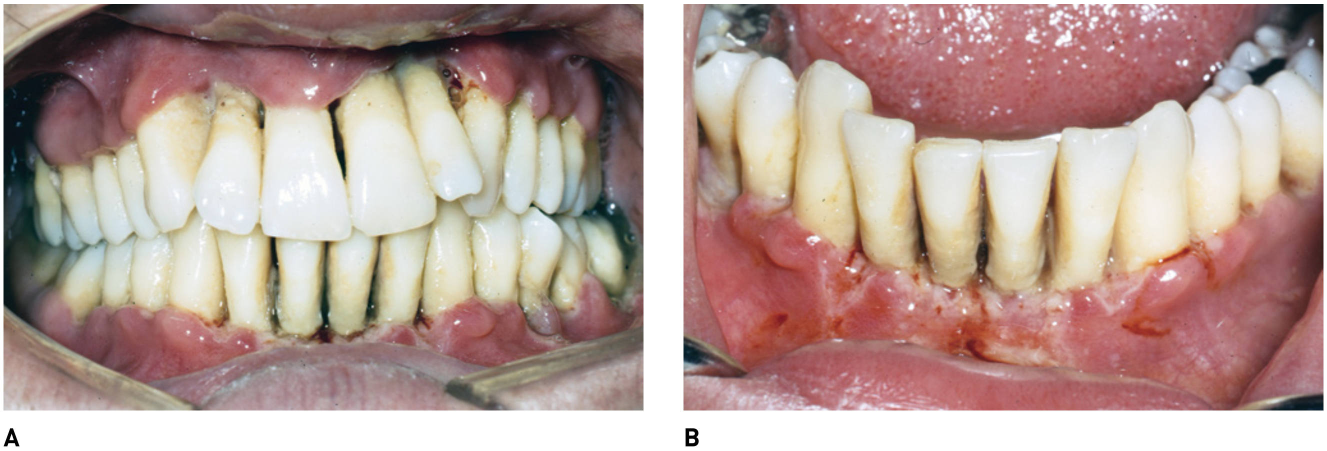

Figure 9-16 **Necrotizing Periodontitis. A** shows a patient with necrotizing periodontitis. **B** is a close-up of the same mandibular arch showing bone sequestration.

Courtesy of Dr. Don Rolfs, Periodontal Foundations, Wenatchee, WA.

2. **Typical Treatment of Necrotizing Periodontitis.** The treatment protocol for necrotizing periodontitis is similar to the sequence of treatment outlined above for any type of NPD. However, note that treatment of patients with necrotizing periodontitis may be more complex due to the extensive, irreversible tissue destruction that has taken place (i.e., clinical attachment loss and bone loss). Consequently, referral to a periodontist is recommended. Furthermore, close collaboration with the patient's medical practitioner is warranted as any form of NPD tends to occur in individuals with an underlying immunocompromising disorder (i.e., HIV). Medical management of the case must be done simultaneously with dental therapy.[20]

3. **Necrotizing Stomatitis.** **Necrotizing stomatitis** is an extension of either necrotizing gingivitis or necrotizing periodontitis where the necrosis progresses to deeper tissues beyond the mucogingival line, such as the lip or cheek mucosa.
   a. **Overview of Necrotizing Stomatitis**
      1. Clinical symptoms are similar to those of necrotizing gingivitis and necrotizing periodontitis. The most distinguishing feature of necrotizing stomatitis is the loss of soft tissue leading to the exposure of the underlying alveolar mucosa (known as bone denudation). This may result in an oral–antral fistula and osteitis (inflammation of the bone).
      2. Although it is a rare condition to encounter, necrotizing stomatitis is the most extensive and invasive form of NPD. Necrotizing stomatitis is associated with patients who have severe immunocompromised conditions, such as AIDS.
   b. **Typical Treatment of Necrotizing Stomatitis.** Because the disease is the most extensive and most invasive form of NPD, an immediate consultation and referral to an oral pathologist, oral maxillofacial surgeon, and physician is strongly recommended.

Section 5

Primary Herpetic Gingivostomatitis

1. **Overview of Primary Herpetic Gingivostomatitis**
   a. **Etiology of Primary Herpetic Gingivostomatitis.** Primary herpetic gingivostomatitis is a medical condition resulting from a viral infection (herpes simplex). It is listed here as a periodontal emergency condition because patients with this condition may first seek care in a dental office because of the nature of the oral symptoms.
   b. **Characteristics of Primary Herpetic Gingivostomatitis**
      1. **Primary herpetic gingivostomatitis** is a painful oral condition that can result from the initial infection with the herpes simplex virus (HSV).[33–35]
         a. There are two types of herpes simplex virus: oral herpes virus (HSV-1) and genital herpes virus (HSV-2). Primary herpetic gingivostomatitis is usually caused by initial infection with HSV-1, but, in rare cases, may be caused by initial infection with HSV-2.

b. In most patients, the initial infection with these viruses produces no noticeable clinical signs and can go undetected clinically. In other patients, however, the oral symptoms resulting from this initial infection can be quite severe, and it is these severe oral symptoms that are known as primary herpetic gingivostomatitis (**Figure 9-17**).

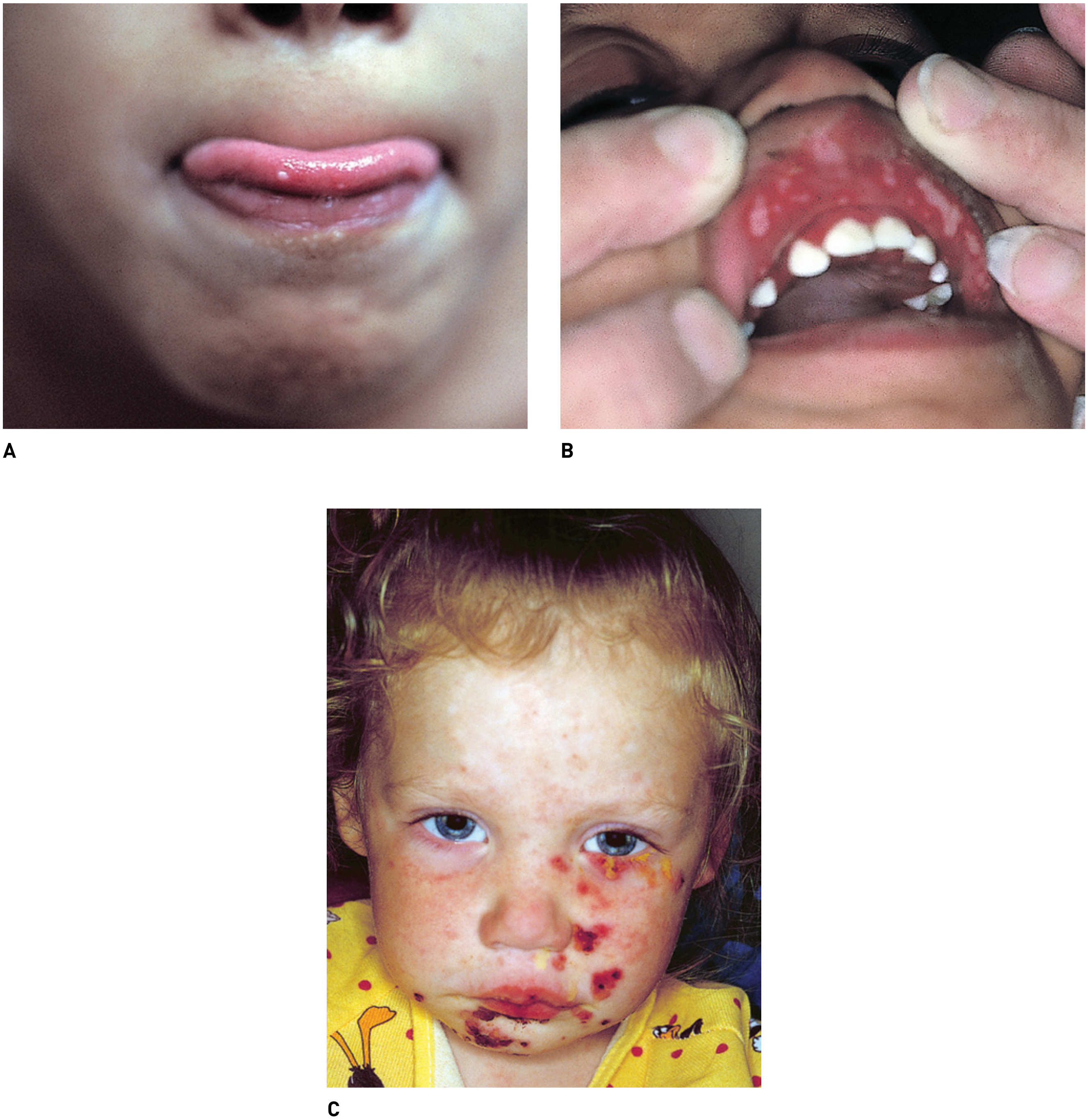

Figure 9-17 Primary Oral Infection with Herpes Simplex Virus. These three children demonstrate the spectrum of primary oral infection with the herpes simplex virus, which ranges from nearly asymptomatic to severe. The patient in **A** has a single vesicle on his tongue. The patient in **B** manifests widespread labial and gingival lesions. The parent's fingers are shown in the photograph; however, touching the infected area with bare fingers is not recommended. The dental hygienist should inform parents that physical contact with the open sores or saliva infected with HSV-1 virus without personal protective equipment (i.e., gloves) can spread the virus. The patient in **C** shows a severe infection with lesions on the face.

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   c. Primary herpetic gingivostomatitis usually occurs in infants or children younger than 6 years of age. However, individuals may acquire the disease later in life when they kiss an infected person during adolescence or adulthood.

2. Primary herpetic gingivostomatitis is contagious and requires careful attention to prevent its spread.
   a. HSV lesions proceed through several stages before healing. These stages are prodrome, macule, papule, vesicle, ulcer, scab, healed area with redness, and complete healing.
      1. Of all the stages of HSV, the two most important stages, from a dental management standpoint, are the vesicle and ulcer stages. The vesicle stage is characterized by fluid-filled blisters that are filled with actively reproducing HSV viral particles. When the vesicle ruptures (the ulcer stage), the HSV-filled fluid escapes and potentially spreads to other areas of the patient's mouth or other individuals, such as a dental practitioner.
      2. It is primarily for this reason that the American Academy of Oral Medicine recommends "delaying care until an HSV lesion is scabbed over or completely healed is prudent for minimizing recurrences and spread of the infection, and that the presence of an infectious HSV lesion orally or periorally can be a reason for deferral of care."[36]
   b. HSV-1 is primarily spread by direct contact through kissing, contact with open sores, or by contact with infected body fluids, such as saliva.
   c. HSV-1 can also spread from one part of the body to another, such as from saliva to the fingers, then to the eye. Transmission of HSV-1 to the eye can result in a painful and dangerous herpetic infection of the cornea (herpes keratitis), which may lead to permanent loss of vision, if left untreated. Herpetic whitlow is a painful herpetic infection of the hands and fingers that is initiated by exposure to HSV-1–infected body fluids (**Figure 9-18**).

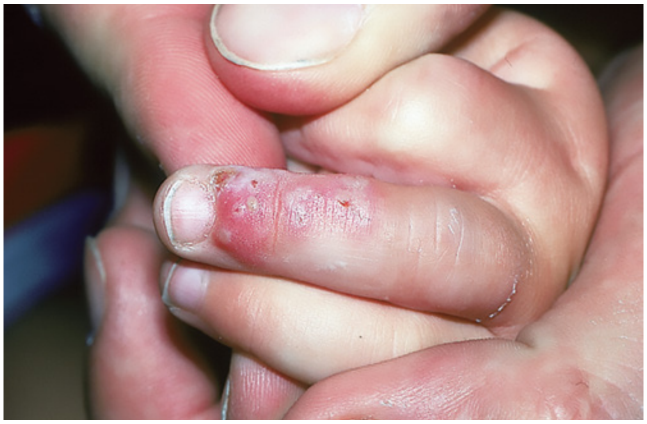

Figure 9-18 Primary Herpes Simplex Virus Infection in Infancy. Finger sucking likely caused the spread of the infection from the mouth to the hand of this infant (herpetic whitlow). The parent's fingers are shown in the photograph; however, touching the infected area with bare fingers is not recommended because of the risk of contracting the disease.

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3. The initial infection with HSV-1 usually occurs in children or in young adults, but it can occur at any age.[37]
4. Primary herpetic gingivostomatitis has a short duration that self-resolves within 2 weeks following onset. Despite the self-limiting nature of HSV lesions, the dental hygienist should know the procedures that reduce the risk of disease transmission and that provide pain relief for the patient.
5. Once a patient is infected with this virus, the virus travels to the trigeminal ganglion where it enters the latent stage (dormant state). Throughout the lifetime of the patient, the infection may reactivate ("wake up") periodically to cause recurrent HSV infection. Recurrent HSV infections are characterized

by either single or multiple vesicles that form on keratinized tissue (i.e., hard palate or gingiva) and rupture quickly to become ulcerative lesions. Recurrence can be triggered by a wide variety of factors, including as stress, trauma, sunlight, and fever. The most common manifestation of recurrent HSV-1 infection is herpes labialis (known commonly as the "cold sore"). Treatment protocols for recurrent HSV infections are the same as those recommended for treatment of primary herpetic gingivostomatitis.

2. **Clinical Signs and Treatment of Primary Herpetic Gingivostomatitis**
   a. **Clinical Signs.** As already mentioned, the clinical signs of primary herpetic gingivostomatitis can range from subclinical (no noticeable signs at all) to rather severe; the severe clinical signs are discussed below and outlined in **Box 9-8**.

Box 9-8 Clinical Signs of Primary Herpetic Gingivostomatitis

- Oral pain with difficulty in eating and drinking
- Edematous gingival tissues (swollen gingival tissue)
- Bleeding from gingival tissue
- Vesicles (blisters) and ulceration of the gingival tissue and sometimes the lips, tongue, and palate; ulcerations surrounded by red halo
- Elevated body temperature
- Malaise (vague feeling of discomfort)
- Swollen lymph nodes

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1. Severe oral pain can be associated with primary herpetic gingivostomatitis, and this discomfort results in difficulty in eating and drinking.
2. The gingival tissues appear swollen (edematous) and red (erythematous) and bleed quite easily when disturbed.
3. Primary herpetic gingivostomatitis is accompanied by painful oral ulcers. Careful inspection of the gingival tissue can reveal small clusters of fluid-filled blisters (vesicles) on the tissues that burst, leaving numerous, painful oral ulcers. The ulcers are surrounded by a red halo. It is important to reemphasize that when the fluid-filled blisters rupture, HSV-1 viral particles are released. This virus-filled fluid can serve as a source of infection and spread the disease to others.
   a. The ulcers can occur on lips, palate, and tongue as well as the gingival tissue.
   b. Pain caused by these ulcers can be such a major problem that it is difficult for the individual to eat and drink. Restricting fluids can even lead to dehydration, and dehydration in a child can be a serious medical emergency.
4. In the more severe clinical manifestation, this infection is associated with signs and symptoms such as elevated body temperature, a vague feeling of discomfort (malaise), headache, and swollen lymph nodes (lymphadenopathy).

b. **Treatment.** Treatment of patients with primary herpetic gingivostomatitis is primarily supportive (i.e., designed to keep the patient as comfortable as possible until the viral infection runs its course). Typical steps in the management of a patient with primary herpetic gingivostomatitis are discussed below and outlined in **Box 9-9**.

Box 9-9 Typical Steps in Management of Primary Herpetic Gingivostomatitis

- Keep in mind that this disease is highly contagious, especially in its vesicle and ulcer stages. Consequently, all elective dental treatment should be deferred until the HSV-1 lesion is scabbed over or completely healed.
- Primary herpetic gingivostomatitis regresses spontaneously in about 2 weeks.
- Control oral discomfort. Topical oral anesthetics can be used for temporary relief of oral discomfort so that the patient can eat and drink fluids.
- Recommend frequent fluid intake to avoid dehydration.
- Refer the patient to a physician if systemic symptoms are severe or if the patient is unable to tolerate fluid intake.

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1. The dental practitioner should keep in mind that primary herpetic gingivostomatitis is contagious, and any plan for periodontal instrumentation of the teeth should be postponed until the initial infection regresses.[38,39]
2. In young children, herpes simplex virus is transmitted primarily by contact with infected saliva. Precautions should be taken to protect others in the home, daycare center, or other environments in which the affected child may encounter other children. Contact should be avoided with the child's mouth, as a painful cross-infection of the virus can occur on the fingers or nail cuticle if infected oral membranes are touched (refer to herpetic whitlow description above). All members of the dental team must adhere to universal precautions for infection control, and parents or guardians should be informed as well.
3. Primary herpetic gingivostomatitis usually regresses spontaneously (goes away without treatment) in approximately 1–2 weeks. Controlling discomfort and ensuring fluid intake are the main focus for supportive treatment.
4. Topical oral anesthetics can be used to control oral discomfort temporarily to allow the patient to eat or to drink fluids. Examples of topical anesthetics that can be used are (1) lidocaine 2% viscous and (2) Orabase with benzocaine.[40–42]
5. In some patients, treatment will include antiviral medications (acyclovir or valacyclovir), medications to reduce fever (antipyretics), and systemic medications to control pain (analgesics).[40,43] Antivirals have their greatest effects when taken within 72 hours of lesion eruption.[22]
6. In an American study assessing the prevalence of HSV infection visits to hospital emergency departments (ED) from 2006 to 2013, approximately 116,000 visits to the ED were due to patients presenting with the signs and symptoms consistent with the diagnosis of herpetic gingivostomatitis. Hence, because a significant number of hospital ED visits are due to primary HSV-1 infections, dental practitioners may be the first health care provider to recognize the early signs of herpetic gingivostomatitis, manage the symptoms of the disorder, and refer patients suffering from these conditions to medical practitioners if the symptoms persist and are refractory. Likewise, early detection, management, and referral to medical practitioners is also crucial to managing any of the acute periodontal diseases that have been reviewed in this chapter.[44]

Chapter Summary Statement

Although not as common as dental biofilm–induced gingivitis or periodontitis, acute periodontal diseases demand urgent attention because of the spontaneous onset, rapid progression, and intense pain associated with these conditions. Thus, all members of the dental team shoulder the responsibility of being able to intercept these conditions promptly and appropriately.

Section 6

Focus on Patients

Clinical Patient Care

CASE 1

During a routine patient visit for periodontal maintenance, you note that there is swelling in the interdental papilla between a patient's two central incisors. The swelling is localized to the area between the incisors. As you manipulate the tissues, you note pus coming from the sulcus of one of the central incisors. You also note some mobility of one of the incisor teeth. When questioned, the patient informs you that she is aware that these tissues are swollen and that she has been flossing more in hope that the swelling would go down. In view of these clinical findings, how should you proceed at this maintenance visit?

CASE 2

A patient with necrotizing gingivitis is referred to you by the dentist for calculus removal. Your examination of the patient reveals ulceration of most interdental papillae with the typical punched-out papillae often seen with this disease. The necrotic tissue pseudomembrane is present covering the ulcerations, and heavy calculus deposits are evident. The patient is quite uncomfortable. How should you proceed with the calculus removal?

Evidence in Action

A concerned mother, who is one of your maintenance patients, brings her 3-year-old daughter to the office. An examination reveals that the child has primary herpetic gingivostomatitis. Her mouth is very painful and she has an elevated temperature. What patient education would you provide to the child's mother? What treatment recommendations should the dental team make?

Ethical Dilemma

You are in your last semester of dental hygiene school, and the clinic has booked your morning appointment. Your patient is Josh K, a 21-year-old senior pre-med student who attends your university. As you review Josh's health history, he informs you that he has been extremely stressed studying for his MCAT (Medical College Admission Test) as well as completing all of graduation requirements for his bachelor's degree in physics. He admits to eating poorly, due to his busy schedule, as well as feeling very fatigued. He is getting very little sleep, only about 3 hours per night, just trying to keep up and get everything done. He has scheduled his appointment today as his gums have become very sore and are bleeding. He tells you that he is just very uncomfortable.

Your clinical exam reveals that Josh's interdental papilla seems to have a "punched out" appearance. There also appears to be a gray-white layer covering his gingival tissue. During your examination, even the slightest manipulation of his gingival tissues causes bleeding. Josh also presents with swollen lymph nodes, an elevated temperature, and extreme halitosis.

Although the clinical dentist is on vacation this week, and did not examine Josh, you remember from your Periodontology class that these classic symptoms and appearance of his tissue appear to be consistent with a necrotizing periodontal disease. You excitedly tell Josh your diagnosis, and once you have finished his assessments, reappoint him for treatment next week.

You meet your roommate Maeve, who is also a dental hygiene student, for dinner in the cafeteria after clinic. You tell her all about Josh, and his classic textbook case of necrotizing gingivitis. You decide to become "Poster Presentation" partners, and necrotizing gingivitis will be your topic. You plan to take before and after photographs of Josh's gingiva for your poster.

1. How would you explain necrotizing periodontal disease to Josh?
2. What are the typical treatment steps for a patient with necrotizing gingivitis?
3. Are there ethical principles in conflict in this dilemma?

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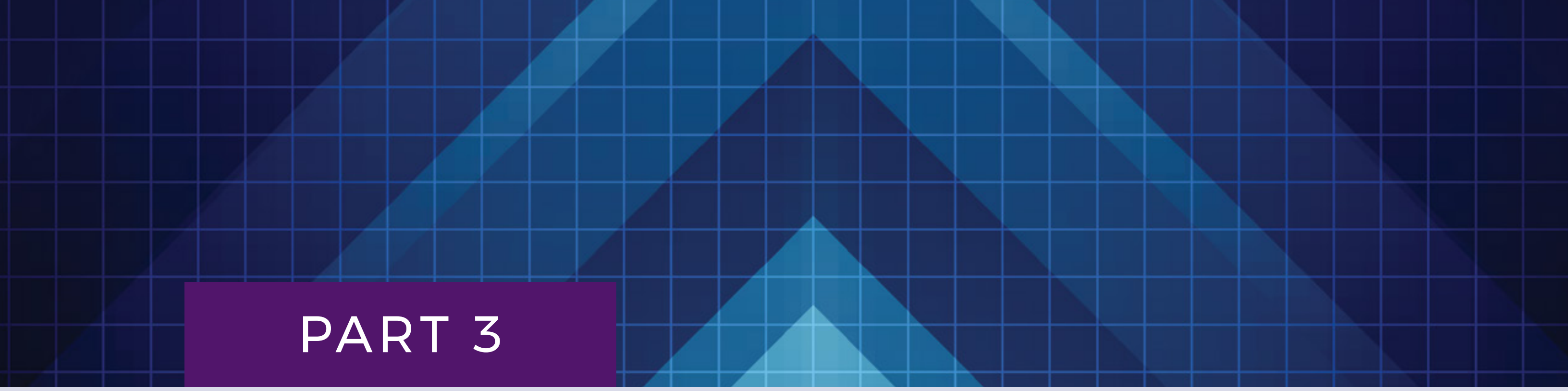

Etiologic Factors for Periodontal Diseases and Peri-Implant Diseases/Conditions

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CHAPTER 10

Risk Factors for Periodontal Disease

- Section 1 Risk Factors for Periodontal Disease
  - Local Risk Factors
  - Systemic Risk Factors
  - Tobacco Use
  - Medications
  - Other Risk Factors
- Section 2 Balance Between Periodontal Health and Disease
- Section 3 Periodontal Risk Assessment
- Section 4 Focus on Patients
  - Evidence in Action
  - Ethical Dilemma
  - Clinical Patient Care

CLINICAL APPLICATION

Why are certain individuals more at risk of developing periodontal disease compared to others? This is a central question that has befuddled dental practitioners for many decades. Although ongoing research has broadened our knowledge in the realm of periodontal disease etiology and pathogenesis, our current understanding to the above question remains unanswered because there is still a lot that we do not know as it pertains to *risk factors* for periodontal disease. The objective of this chapter is to identify potential periodontal risk factors and expound on the role risk factors play in the development and progression of periodontal disease.

LEARNING OBJECTIVES

- Define *risk factor* and provide various examples of risk factors.
- Define *etiologic factor* and distinguish etiologic factors from risk factors.
- Define *biologic equilibrium* and explain how certain factors can disrupt the balance between health and disease.
- Explain the differences between modifiable and nonmodifiable risk factors.
- Describe the significance of performing a thorough periodontal risk assessment for each patient in a clinical setting.
- In a clinical setting—for a patient in your care with periodontitis—explain to your clinical instructor the factors that may have contributed to your patient's disease progression.

KEY TERMS

Risk factor
Etiologic factor
Biologic equilibrium
Homeostasis
Risk assessment

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Section 1

Risk Factors for Periodontal Disease

It is widely accepted that periodontal disease is a bacterial-induced infection of the periodontium and that pathogenic bacteria are the primary etiologic factors in the initiation of periodontal disease.[1–4] However, the presence of pathogenic bacteria alone fails to explain why some individuals are more at risk of developing disease compared to others.[5–15] To put it another way, the existence of the cause (in this case, pathogenic bacteria within the oral biofilm) does not necessarily mean the event (in this case, disease onset) will happen. This means that the onset of periodontal disease is much more complex than we initially thought.

The concept of risk factors is very important to our understanding of disease. A **risk factor** is any attribute, characteristic, or exposure that is associated with an increased likelihood of developing a disease or injury. Key to understanding this concept is that risk factors are elements that are present in a patient *before* the onset of disease. For example, a smoker may not presently have periodontal disease; however, this individual is more at risk of developing the disease in the future due to the smoking habit. Smoking is therefore a risk factor because it increases the likelihood of disease in the future. Another key concept is that the term *risk factor* should not be confused with a similar, yet different, term: *etiologic factor*. An **etiologic factor** is any attribute, characteristic, or exposure that is known to *cause* the disease (as opposed to a risk factor which implies correlation but does not prove causation). In the context of this chapter, the focus will be on reviewing some of the common risk factors associated with periodontal disease (**Figure 10-1**). Subsequent chapters in this textbook will offer an in-depth discussion of systemic and local etiologic factors that lead to the initiation of the disease.

Risk factors are classified as either modifiable—things that can be controlled or changed—or nonmodifiable factors—things that can neither be controlled nor changed (**Figure 10-2**). Some examples of modifiable risk factors include smoking, diabetes mellitus, and psychological factors (e.g., stress). Examples of nonmodifiable risk factors include, but are not limited to, age, gender, genetics, and socioeconomic status.

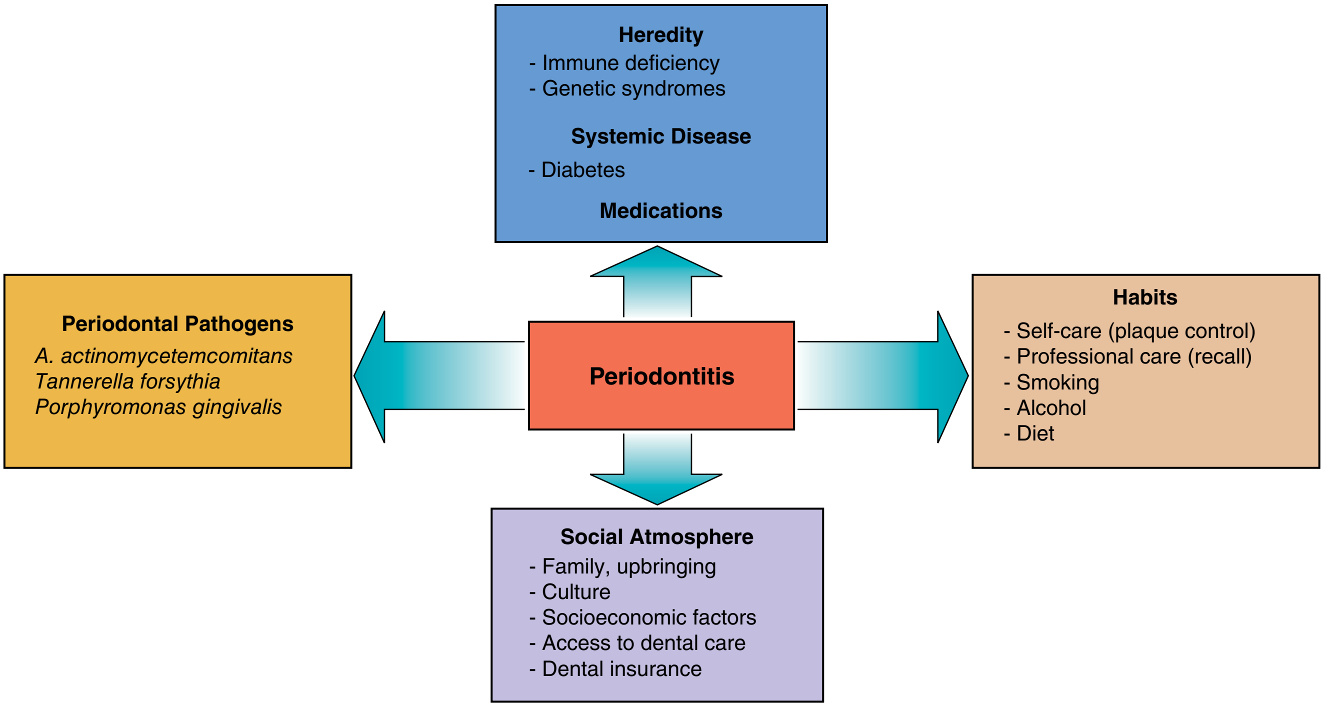
Heredity
- Immune deficiency
- Genetic syndromes
Systemic Disease
- Diabetes
Medications
Periodontal Pathogens
A. actinomycetemcomitans
Tannerella forsythia
Porphyromonas gingivalis
Periodontitis
Habits
- Self-care (plaque control)
- Professional care (recall)
- Smoking
- Alcohol
- Diet
Social Atmosphere
- Family, upbringing
- Culture
- Socioeconomic factors
- Access to dental care
- Dental insurance

Figure 10-1 Risk Factors for Periodontal Disease. Many risk factors have been identified that can make certain individuals more at risk of developing periodontal disease than others.

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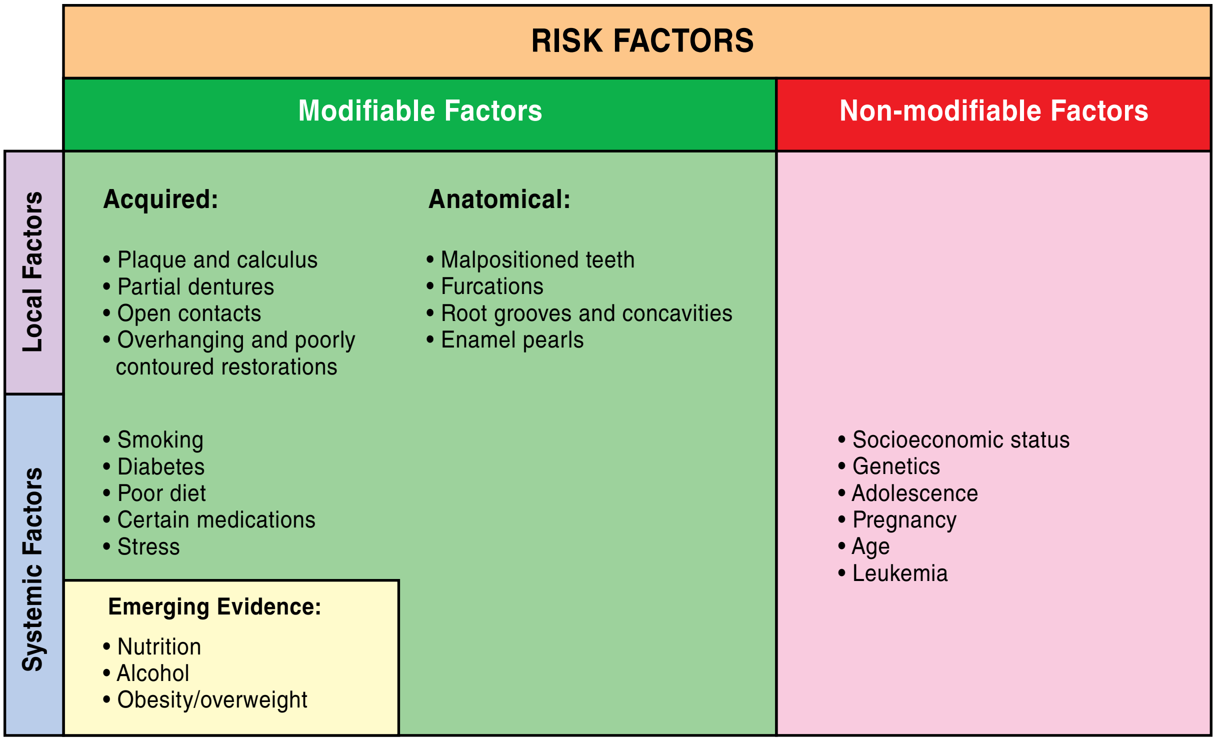

RISK FACTORS

Modifiable Factors

Non-modifiable Factors

Local Factors

Acquired:

- Plaque and calculus
- Partial dentures
- Open contacts
- Overhanging and poorly contoured restorations

Anatomical:

- Malpositioned teeth
- Furcations
- Root grooves and concavities
- Enamel pearls

Systemic Factors

- Smoking
- Diabetes
- Poor diet
- Certain medications
- Stress

Emerging Evidence:

- Nutrition
- Alcohol
- Obesity/overweight

- Socioeconomic status
- Genetics
- Adolescence
- Pregnancy
- Age
- Leukemia

Figure 10-2 **Modifiable and Nonmodifiable Risk Factors.** Risk factors for periodontal disease can be classified as modifiable or nonmodifiable factors. Modifiable risk factors are usually environmental or behavioral in nature, whereas nonmodifiable risk factors are usually intrinsic to the individual, and therefore cannot be changed.

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Local Risk Factors

Local risk factors can either be acquired or anatomic. Acquired local risk factors include, but are not limited to, calculus, overhanging restorations, and poorly contoured restorations. Examples of anatomic risk factors are malpositioned teeth, root grooves, concavities, and furcations.

Systemic Risk Factors

Several systemic diseases, states, or conditions can affect the periodontium. Examples that fall under this category include, but are not limited to, uncontrolled diabetes, stress, hormonal changes, systemic bone metabolic disorders (e.g., osteoporosis), and any neutrophilic disorder (e.g., Chediak-Higashi syndrome or cyclic neutropenia) that manifests as periodontitis and fall under the current diagnostic category of *periodontitis as a manifestation of systemic disease*.

Tobacco Use

The most significant known risk factor for periodontitis is cigarette smoking. Smoking has a profound impact on periodontitis development and treatment response. Because of its significance, tobacco as a risk factor for periodontal disease is discussed in detail in Chapter 17.

Medications

Certain medications have the unintended and undesirable side-effect of contributing to gingival enlargement. Calcium channel blockers (e.g., amlodipine and nifedipine), anticonvulsants, (e.g., phenytoin), and immunosuppressants (e.g., cyclosporin) are common medications that increase an individual's likelihood to developing gingival enlargement.

Section 2

Balance Between Periodontal Health and Disease

1. **Biologic Equilibrium.** The human body possesses a physiologic mechanism that functions to maintain a state of balance in the internal environment of the body. This is known as **biologic equilibrium** or **homeostasis**.
    a. **Periodontal Health**
        1. In health, there is a state of biologic equilibrium within the oral cavity between the biofilm bacteria and the host.
        2. For the periodontium to remain healthy, the bacterial challenge must be contained at a level that can be tolerated by the host.[2]
        3. The situation can be thought of as a balance scale, with the disease-promoting factors on one side of the scale and the health-promoting factors on the other (**Figure 10-3**). If the two sides of the scale are in balance, there will be no disease progression.

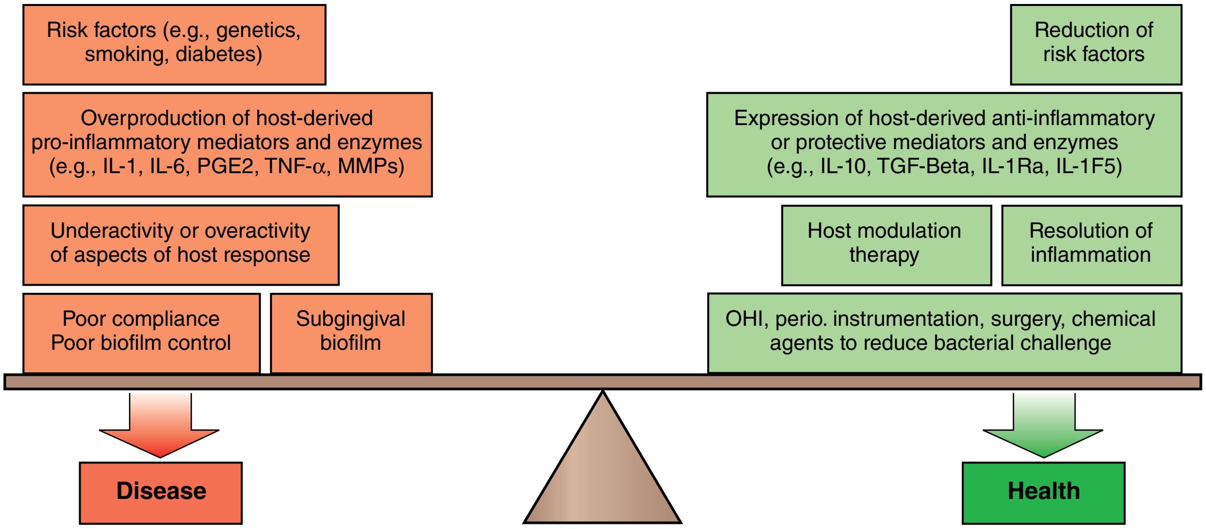
Risk factors (e.g., genetics, smoking, diabetes)
Overproduction of host-derived pro-inflammatory mediators and enzymes (e.g., IL-1, IL-6, PGE2, TNF-α, MMPs)
Underactivity or overactivity of aspects of host response
Poor compliance Poor biofilm control
Subgingival biofilm
Reduction of risk factors
Expression of host-derived anti-inflammatory or protective mediators and enzymes (e.g., IL-10, TGF-Beta, IL-1Ra, IL-1F5)
Host modulation therapy
Resolution of inflammation
OHI, perio. instrumentation, surgery, chemical agents to reduce bacterial challenge
Disease
Health

Figure 10-3 Periodontal Equilibrium. Equilibrium occurs when there is a balance between disease-promoting factors and health-promoting factors.

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    b. **Periodontal Disease**
        1. The presence of certain risk factors, however, can "tip" the balance, thereby leading to disease.
        2. Notably, a known risk factor present in one individual may not be a risk factor for another individual. Take for instance two individuals with diabetes, which is a known risk factor: the diabetic individual with poor glycemic control and poor plaque control may be at risk of developing periodontitis, whereas a diabetic patient with good glycemic control and good plaque control will not be at any greater risk to periodontitis.

2. **The Delicate Balance Between Health and Disease.** When active periodontal disease sites are present in the mouth, the goal is to return the oral cavity to a state of biologic equilibrium.
    a. **Periodontal Equilibrium and Dental Plaque Biofilm**
        1. Experienced dental practitioners will attest that there are differences in the way that individual patients respond to the plaque biofilm.
            a. Many patients return to the dental office year after year with generalized plaque biofilm. These patients exhibit gingivitis and yet, year after year, show no clinical signs of progression to periodontitis. For some reason, in these individuals, gingivitis never progresses to periodontitis. Perhaps these individuals have no systemic or acquired risk factors that disrupt the biologic equilibrium. So, if an individual's immune system can effectively deal with a mouthful of periodontal pathogens, there will be no destructive periodontal disease (**Figure 10-4**).

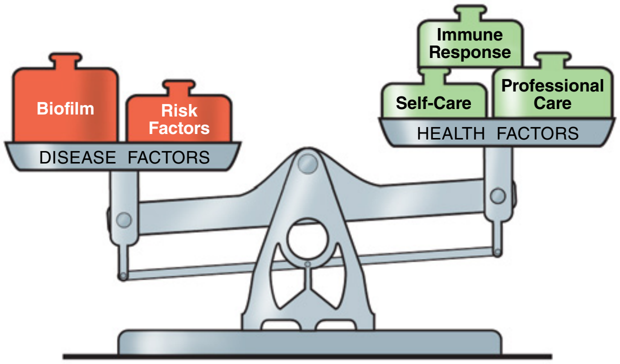

Figure 10-4 Gingivitis in the Presence of Plaque Biofilms. Patients with a low susceptibility to periodontitis may remain in the gingivitis state and never progress to periodontitis.

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b. In a few individuals, gingivitis progresses to periodontitis. It is theorized that in these individuals the body's immune response (host response) is responsible for the tissue destruction seen in periodontitis. In addition, some individuals possess systemic risk factors (such as genetic variables or systemic disease) that significantly increase their susceptibility to periodontitis.

2. There are many patients who are unable or unwilling to perform the thorough self-care necessary to control plaque biofilm. For these patients, it is necessary to increase the frequency of professional care to compensate for the inadequate level of self-care. Professional care at frequent intervals can be effective in restoring the balance between health and disease (**Figure 10-5**).

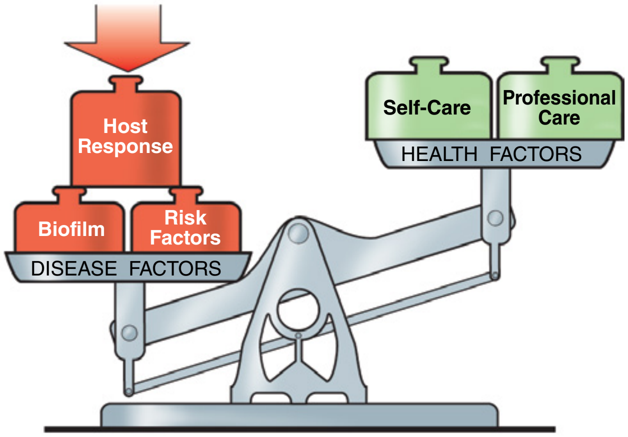

Figure 10-5 Periodontitis in the Presence of Plaque Biofilm. In susceptible individuals, the body's immune response (host response) results in damage to the periodontal tissues and progression from gingivitis to periodontitis.

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b. **Local Risk Factors**

1. In many cases, it is possible to eliminate a local risk factor. A faulty restoration is a good example of a local factor that can be corrected, restoring the balance between local disease–promoting and health-promoting factors at the site.
2. In other cases, it is possible to compensate for a local risk factor by improving the patient's self-care and/or increasing the frequency of professional care. For example, the patient may need to use tufted dental floss to clean around the abutment teeth of a fixed bridge. This situation can be compared to adding more weight on the health side of the balance to equal or exceed the weight on the disease side.

c. **Systemic or Genetic Risk Factors**

1. Certain systemic or acquired risk factors are possible to control or eliminate if the patient is willing to do so. For example, the individual can work with a physician to keep diabetes well controlled. A smoker may decide to stop smoking. In both cases, the individual has made a positive change that is health promoting, both systemically and for the periodontium (**Figure 10-6**).

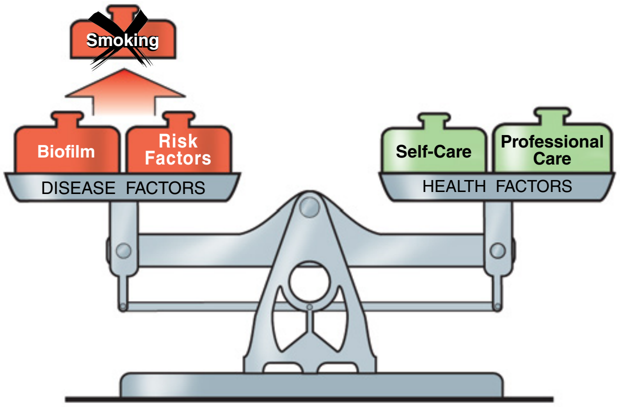
Smoking
Biofilm
Risk Factors
DISEASE FACTORS
Self-Care
Professional Care
HEALTH FACTORS

Figure 10-6 Eliminating a Systemic Risk Factor. Smoking cessation, combined with adequate self-care and professional care, restores the balance.

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2. In the case of a contributing risk factor that cannot be controlled, it is necessary to add weight to the health side of the scale. For example, some individuals have a genetic risk factor—such as abnormal neutrophil function—that causes them to be susceptible to severe periodontitis. At the present time, we are unable to eliminate or control genetic risk factors. It is possible, however, to assist the patient in maintaining health by increasing the extent of professional care. Frequent professional care will increase the weight on the health side of the scale (**Figure 10-7**).

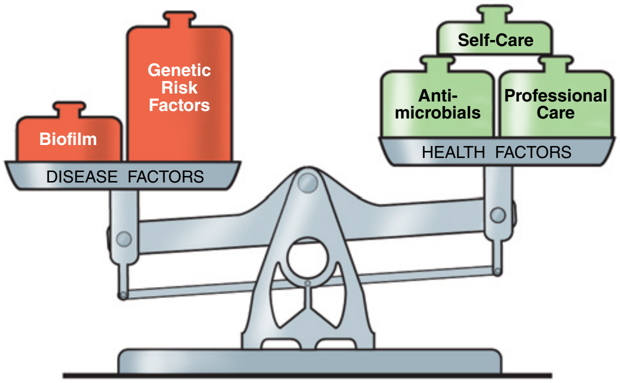
Genetic Risk Factors
Biofilm
DISEASE FACTORS
Self-Care
Anti-microbials
Professional Care
HEALTH FACTORS

Figure 10-7 Management of a Genetic Risk Factor. Currently, some risk factors cannot be eliminated or controlled. Professional care can help slow disease progression.

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Section 3

Periodontal Risk Assessment

Dental health care providers are interested not only in diagnosing and treating periodontal disease, but also in predicting which individuals are more likely to develop periodontitis. The process of identifying risk factors that increase an individual's probability of disease is called **risk assessment**.[2,7–10,14,16-18] The American Academy of

Periodontology (AAP) describes the risk assessment process as "increasingly important in periodontal treatment planning and should be part of every comprehensive dental and periodontal evaluation."[16]

1. **Assessment of the Individual**
   a. **An Individual's Risk Factors**
      1. It is becoming possible to consider an individual's risk factors for periodontal disease (e.g., systemic disease, genetic information, personal habits, and characteristics) and to classify patients into high- or low-risk groups.
      2. For example, smokers have a higher risk of periodontitis than nonsmokers.
   b. **Disease Prevention for the Individual**
      1. Clinicians also use the risk assessment process to prevent disease in susceptible individuals (e.g., identifying smokers and offering smoking cessation counseling before the onset of periodontal disease).
      2. Information concerning individual risk for developing periodontal disease is obtained through careful evaluation of the individual's demographic data, medical history, dental history, and comprehensive periodontal clinical examination (**Table 10-1**).

Table 10-1 Clinical Risk Assessment for Periodontal Disease

Demographic data	Age Duration of exposure to contributing risk factors Self-care (plaque biofilm control) Frequency of professional care Male gender Dental awareness Socioeconomic status
Medical history	Tobacco use Diabetes Osteoporosis HIV/AIDS Genetic predisposition to aggressive disease
Dental history	Frequency of professional care Family history of early tooth loss Previous history of periodontal disease
Clinical examination	Plaque biofilm accumulation and microbial composition Calculus deposits Bleeding on probing Loss of attachment Plaque retentive areas Anatomic contributing factors Restorative contributing factors

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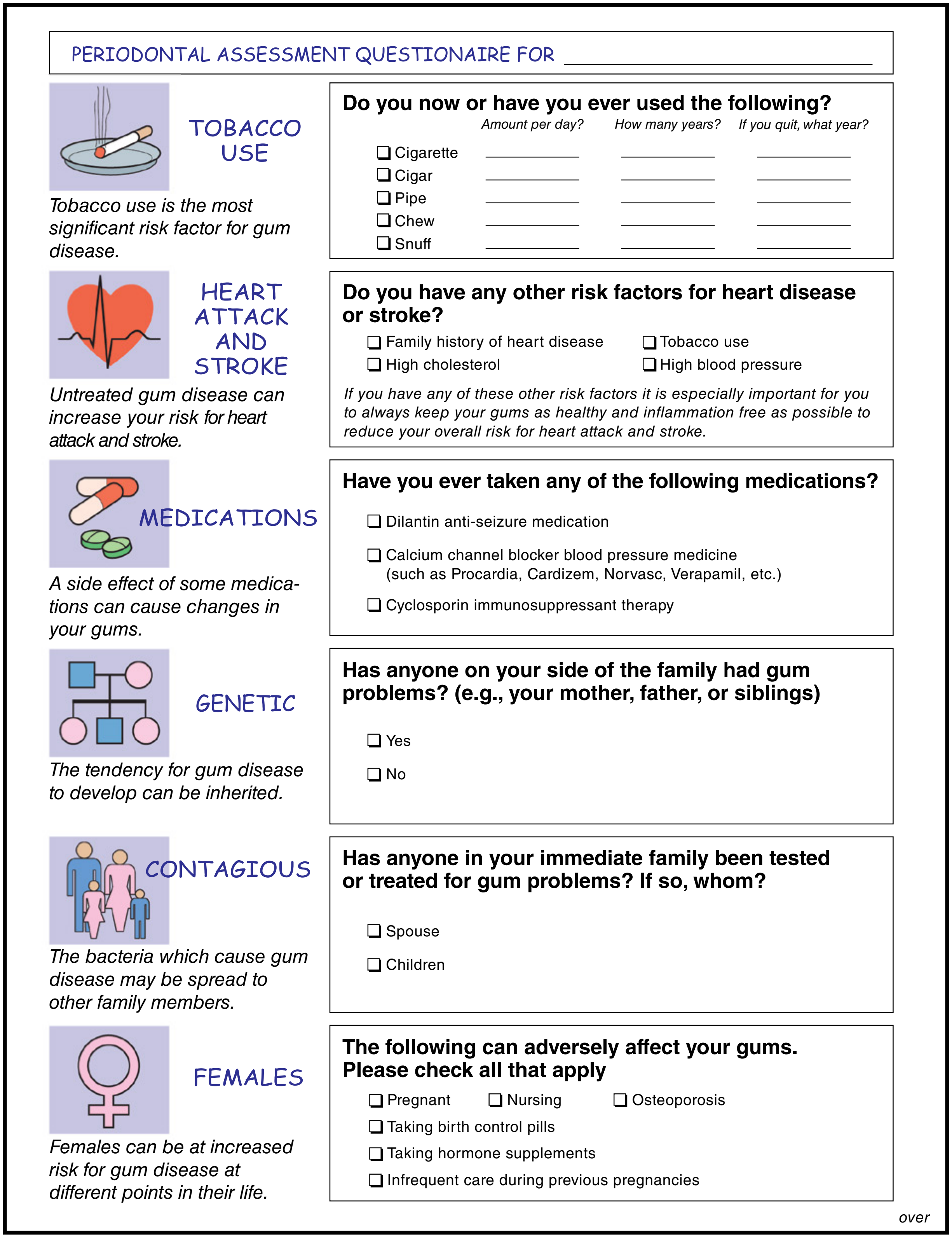
2. **Risk Assessment Systems**
   a. **Web-Based Tools.** Several commercially produced Web-based risk calculation tools are available.
      1. DentoRisk is a two-stage Web-based system for periodontitis risk assessment based on data collected on 20 separate factors.[19,20]
      2. Another system, the Periodontal Risk Assessment system, originally developed by Lang and Tonetti,[21] has been shown to be of practical value in clinical practice.
      3. The PreViser Periodontal Risk Calculator developed in 2002 is a Web-based system that aims to provide dental clinicians with an easy-to-use method to determine patients' periodontitis risk.[22–24]
   b. **Risk Questionnaires.** Risk assessment questionnaires are practical tools that can be helpful in identifying individuals who are at a high risk for periodontal disease. **Figure 10-8** shows an example of a simple two-page periodontal risk questionnaire that can elicit the presence of common periodontal risk factors. Dental hygienists can use risk questionnaires to initiate discussion with patients about periodontal risk factors.



PERIODONTAL ASSESSMENT QUESTIONAIRE FOR ____________________

TOBACCO USE

Tobacco use is the most significant risk factor for gum disease.

Do you now or have you ever used the following?

	Amount per day?	*How many years?*	*If you quit, what year?*
☐ Cigarette	______	______	______
☐ Cigar	______	______	______
☐ Pipe	______	______	______
☐ Chew	______	______	______
☐ Snuff	______	______	______

HEART ATTACK AND STROKE

Untreated gum disease can increase your risk for heart attack and stroke.

Do you have any other risk factors for heart disease or stroke?

☐ Family history of heart disease ☐ Tobacco use
☐ High cholesterol ☐ High blood pressure

If you have any of these other risk factors it is especially important for you to always keep your gums as healthy and inflammation free as possible to reduce your overall risk for heart attack and stroke.

MEDICATIONS

A side effect of some medications can cause changes in your gums.

Have you ever taken any of the following medications?

☐ Dilantin anti-seizure medication

☐ Calcium channel blocker blood pressure medicine (such as Procardia, Cardizem, Norvasc, Verapamil, etc.)

☐ Cyclosporin immunosuppressant therapy

GENETIC

The tendency for gum disease to develop can be inherited.

Has anyone on your side of the family had gum problems? (e.g., your mother, father, or siblings)

☐ Yes

☐ No

CONTAGIOUS

The bacteria which cause gum disease may be spread to other family members.

Has anyone in your immediate family been tested or treated for gum problems? If so, whom?

☐ Spouse

☐ Children

FEMALES

Females can be at increased risk for gum disease at different points in their life.

The following can adversely affect your gums. Please check all that apply

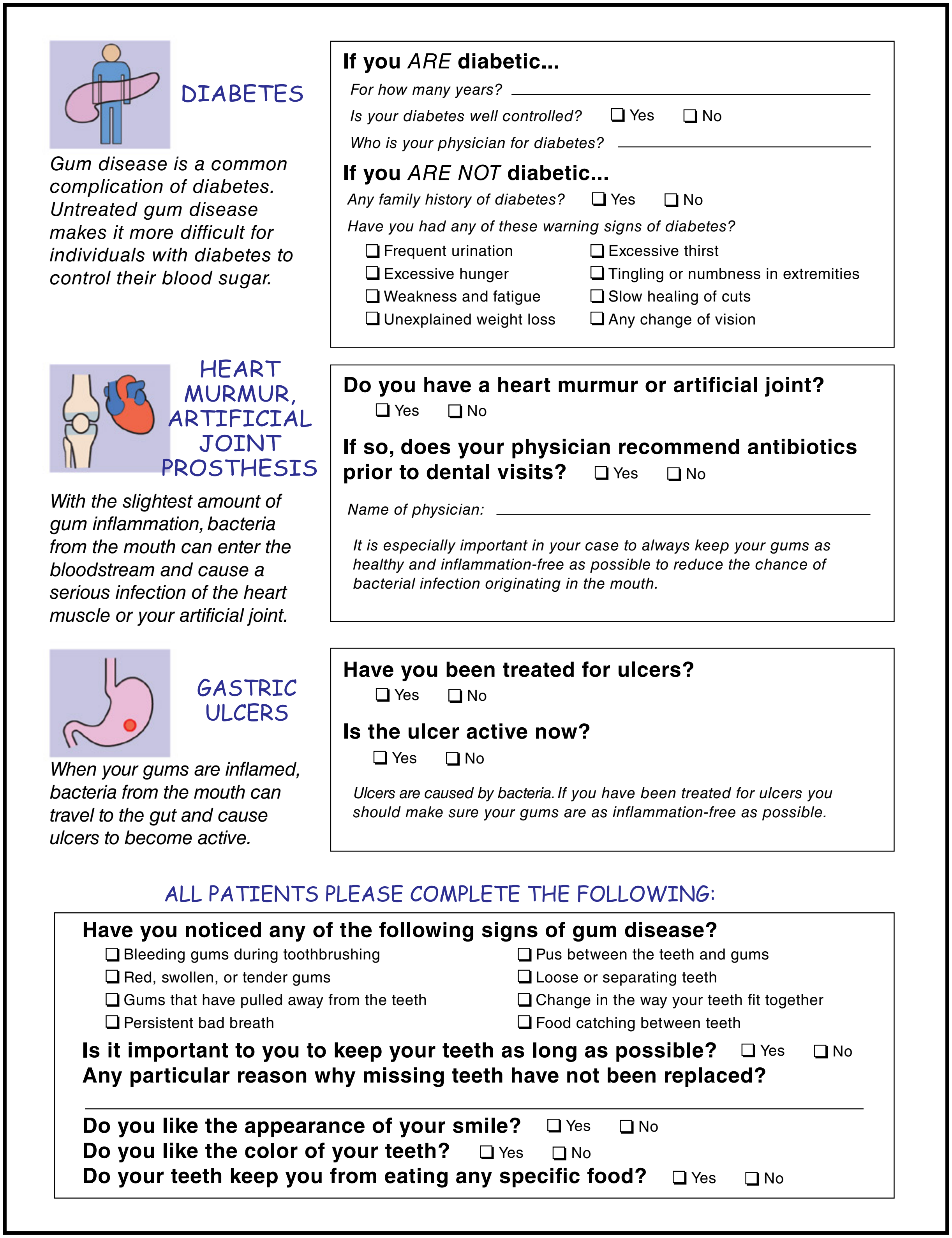
☐ Pregnant ☐ Nursing ☐ Osteoporosis
☐ Taking birth control pills
☐ Taking hormone supplements
☐ Infrequent care during previous pregnancies

over

A

Figure 10-8 Periodontal Risk Questionnaire. Risk assessment questionnaires are practical tools that can be helpful in identifying individuals who have a high susceptibility to periodontitis. **A.** Side 1 of the Periodontal Risk Questionnaire.

Courtesy of Timothy G. Donley, DDS, MSD, Bowling Green, KY.



DIABETES

Gum disease is a common complication of diabetes. Untreated gum disease makes it more difficult for individuals with diabetes to control their blood sugar.

If you *ARE* diabetic...

For how many years? ____________________

Is your diabetes well controlled? ☐ Yes ☐ No

Who is your physician for diabetes? ____________________

If you *ARE NOT* diabetic...

Any family history of diabetes? ☐ Yes ☐ No

Have you had any of these warning signs of diabetes?

- ☐ Frequent urination
- ☐ Excessive hunger
- ☐ Weakness and fatigue
- ☐ Unexplained weight loss
- ☐ Excessive thirst
- ☐ Tingling or numbness in extremities
- ☐ Slow healing of cuts
- ☐ Any change of vision

HEART MURMUR, ARTIFICIAL JOINT PROSTHESIS

With the slightest amount of gum inflammation, bacteria from the mouth can enter the bloodstream and cause a serious infection of the heart muscle or your artificial joint.

Do you have a heart murmur or artificial joint?
☐ Yes ☐ No

If so, does your physician recommend antibiotics prior to dental visits? ☐ Yes ☐ No

Name of physician: ____________________

It is especially important in your case to always keep your gums as healthy and inflammation-free as possible to reduce the chance of bacterial infection originating in the mouth.

GASTRIC ULCERS

When your gums are inflamed, bacteria from the mouth can travel to the gut and cause ulcers to become active.

Have you been treated for ulcers?
☐ Yes ☐ No

Is the ulcer active now?
☐ Yes ☐ No

Ulcers are caused by bacteria. If you have been treated for ulcers you should make sure your gums are as inflammation-free as possible.

ALL PATIENTS PLEASE COMPLETE THE FOLLOWING:

Have you noticed any of the following signs of gum disease?

- ☐ Bleeding gums during toothbrushing
- ☐ Red, swollen, or tender gums
- ☐ Gums that have pulled away from the teeth
- ☐ Persistent bad breath
- ☐ Pus between the teeth and gums
- ☐ Loose or separating teeth
- ☐ Change in the way your teeth fit together
- ☐ Food catching between teeth

Is it important to you to keep your teeth as long as possible? ☐ Yes ☐ No
Any particular reason why missing teeth have not been replaced?

____

Do you like the appearance of your smile? ☐ Yes ☐ No
Do you like the color of your teeth? ☐ Yes ☐ No
Do your teeth keep you from eating any specific food? ☐ Yes ☐ No

B

Figure 10-8 **B.** Side 2 of the Periodontal Risk Questionnaire. (*continued*)

Courtesy of Timothy G. Donley, DDS, MSD, Bowling Green, KY.

Chapter Summary Statement

In a healthy, nondiseased state, the oral cavity is in a delicate state of equilibrium between the bacteria (residing within the oral biofilm) and the host.

For the periodontium to remain healthy, the bacterial challenge must be contained at a level that can be tolerated by the host. The situation can be thought of as a balance scale, with disease-promoting factors on one side of the scale and health-promoting factors on the other. If the two sides of the scale are in balance, there will be no disease progression. Any disruption in the equilibrium can "tip" the scale toward the disease-promoting factor side. Notably, however, the presence of pathogenic bacteria alone, does not necessarily mean that an individual will develop the disease. There must be additional factors that tip the balance to favor disease.

A risk factor is any attribute, characteristic, or behavior that increases an individual's likelihood to *developing* disease. Risk factors such as systemic disease, smoking, and genetic factors can play a significant role in determining the onset and progression of periodontitis.

Despite the tremendous effort that the scientific field has made to find effective treatments, there remains no cure to periodontitis. Therefore, an alternative strategy to fight against periodontitis could be the prevention of the known modifiable risk factors and related mechanisms for the disease. Preventing or, perhaps more realistically, controlling these modifiable risk factors well before the disease has a chance to develop would have a substantial effect on disease prevalence. Therefore, there is an urgent need for clinicians to utilize a periodontal risk assessment so that potential patient-specific risk factors can be identified and addressed prior to disease onset. Additionally, the information gained by performing a thorough periodontal risk assessment may be a powerful tool for any dental professional to use to develop a treatment strategy that aims to prevent disease and promote health.

Section 4

Focus on Patients

Evidence in Action

Mr. and Mrs. Merkel are patients in the general dental practice where you have been the dental hygienist for 10 years. Mrs. Merkel has been diagnosed with generalized Stage III, Grade C periodontitis and receives treatment in your office and at a periodontal practice.

Mrs. Merkel is very compliant with her self-care and frequent professional care. Despite her best efforts, her periodontal disease is progressing.

During today's appointment, Mrs. Merkel breaks into tears. She tells you that she is so conscientious about her daily self-care and keeping all her dental appointments, yet her dental health continues to worsen. She cannot understand why her husband gives his teeth a quick brush and never flosses, yet he has good dental health (no gingivitis or periodontitis). She asks, "How this can be?"

Based on current dental research, how would you answer Mrs. Merkel's question?

Ethical Dilemma

Your first patient on Monday morning is Joyce Robbins. She is new to your practice. She has filled out a Periodontal Assessment Questionnaire to assist you in determining her risk assessment. She states that she has never filled one out before and is concerned that she has received poor-quality dental work as a result. Her medical history and Periodontal Assessment Questionnaire reveal the following:

Joyce is a 58-year-old college professor. She has stage I hypertension and takes 20 mg of lisinopril daily as a result. She reports a family history of cardiac disease, and her father developed type 2 diabetes at age 50. She takes simvastatin for high cholesterol. She is a borderline diabetic, and her blood sugar levels have been rising at each primary care visit. She suffers from osteoporosis of the spine and hip. She is presently a nonsmoker but did smoke while she was in college.

Her oral exam and radiographs reveal periodontitis. She flosses daily but has not had her teeth cleaned in 5 years. She frequently gets food stuck between her teeth and is increasingly becoming concerned about "the way her teeth look and feel."

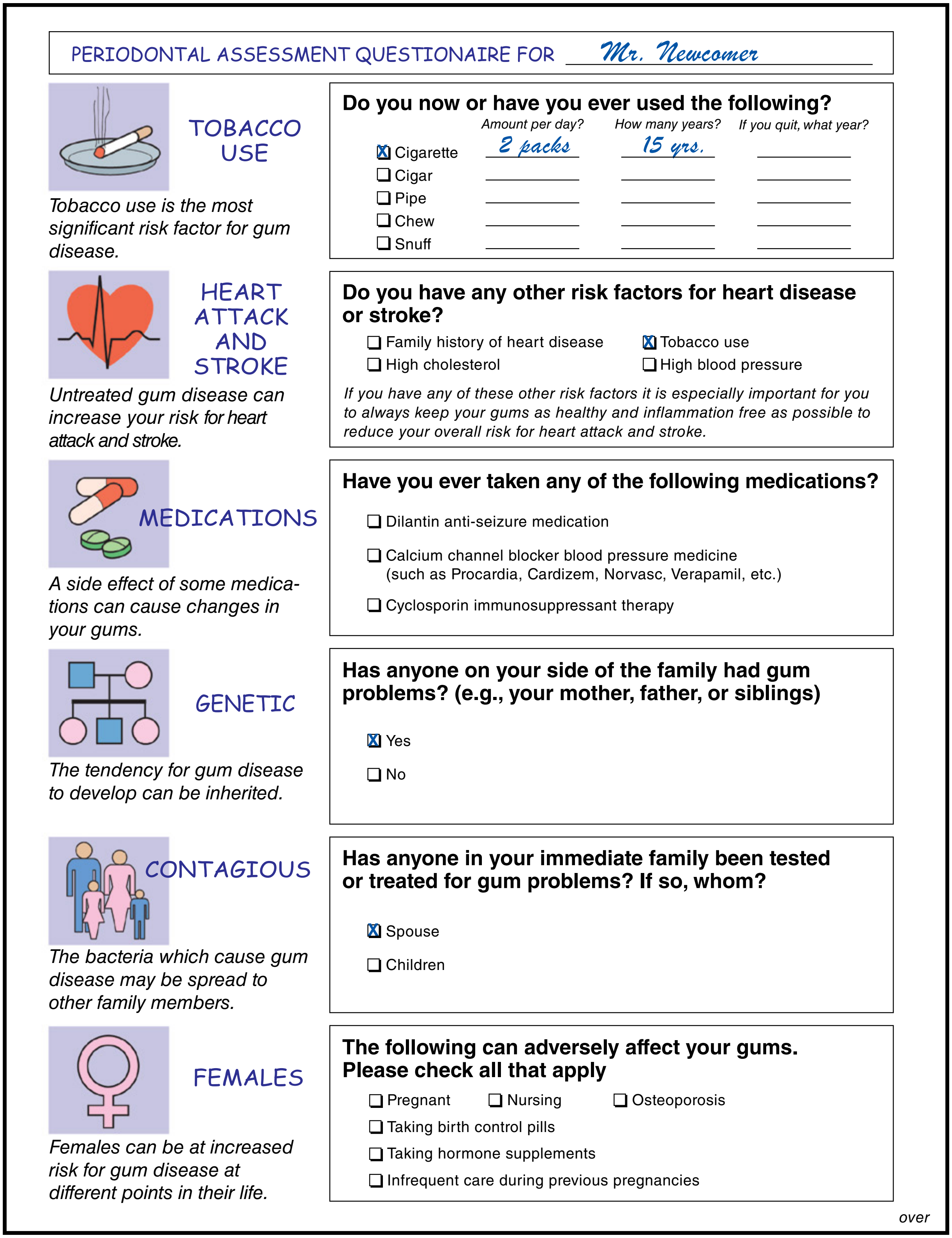
She wants to know what she can do to maintain her "pretty smile," and why no other office was as thorough with her care.

1. What do you think may be the cause of the patient's generalized recession?
2. What factors may have contributed to the patient's disease progression?
3. How will you discuss the balance between periodontal health and disease?
4. Is there an ethical dilemma involved?

Clinical Patient Care

CASE 1

Mr. Archie Newcomer is a new patient in your dental office. Mr. Newcomer is 35 years of age and reports that this is his first dental check-up in 5 or 6 years. Mr. Newcomer's completed Periodontal Assessment Questionnaire is shown in **Figure 10-9**. Review Mr. Newcomer's questionnaire, make a list of periodontal risk factors, and suggest strategies for managing these risk factors.



PERIODONTAL ASSESSMENT QUESTIONAIRE FOR Mr. Newcomer

TOBACCO USE

Tobacco use is the most significant risk factor for gum disease.

Do you now or have you ever used the following?

	Amount per day?	*How many years?*	*If you quit, what year?*
☒ Cigarette	2 packs	15 yrs.	
☐ Cigar			
☐ Pipe			
☐ Chew			
☐ Snuff			

HEART ATTACK AND STROKE

Untreated gum disease can increase your risk for heart attack and stroke.

Do you have any other risk factors for heart disease or stroke?

☐ Family history of heart disease
☒ Tobacco use
☐ High cholesterol
☐ High blood pressure

If you have any of these other risk factors it is especially important for you to always keep your gums as healthy and inflammation free as possible to reduce your overall risk for heart attack and stroke.

MEDICATIONS

A side effect of some medications can cause changes in your gums.

Have you ever taken any of the following medications?

☐ Dilantin anti-seizure medication

☐ Calcium channel blocker blood pressure medicine (such as Procardia, Cardizem, Norvasc, Verapamil, etc.)

☐ Cyclosporin immunosuppressant therapy

GENETIC

The tendency for gum disease to develop can be inherited.

Has anyone on your side of the family had gum problems? (e.g., your mother, father, or siblings)

☒ Yes

☐ No

CONTAGIOUS

The bacteria which cause gum disease may be spread to other family members.

Has anyone in your immediate family been tested or treated for gum problems? If so, whom?

☒ Spouse

☐ Children

FEMALES

Females can be at increased risk for gum disease at different points in their life.

The following can adversely affect your gums. Please check all that apply

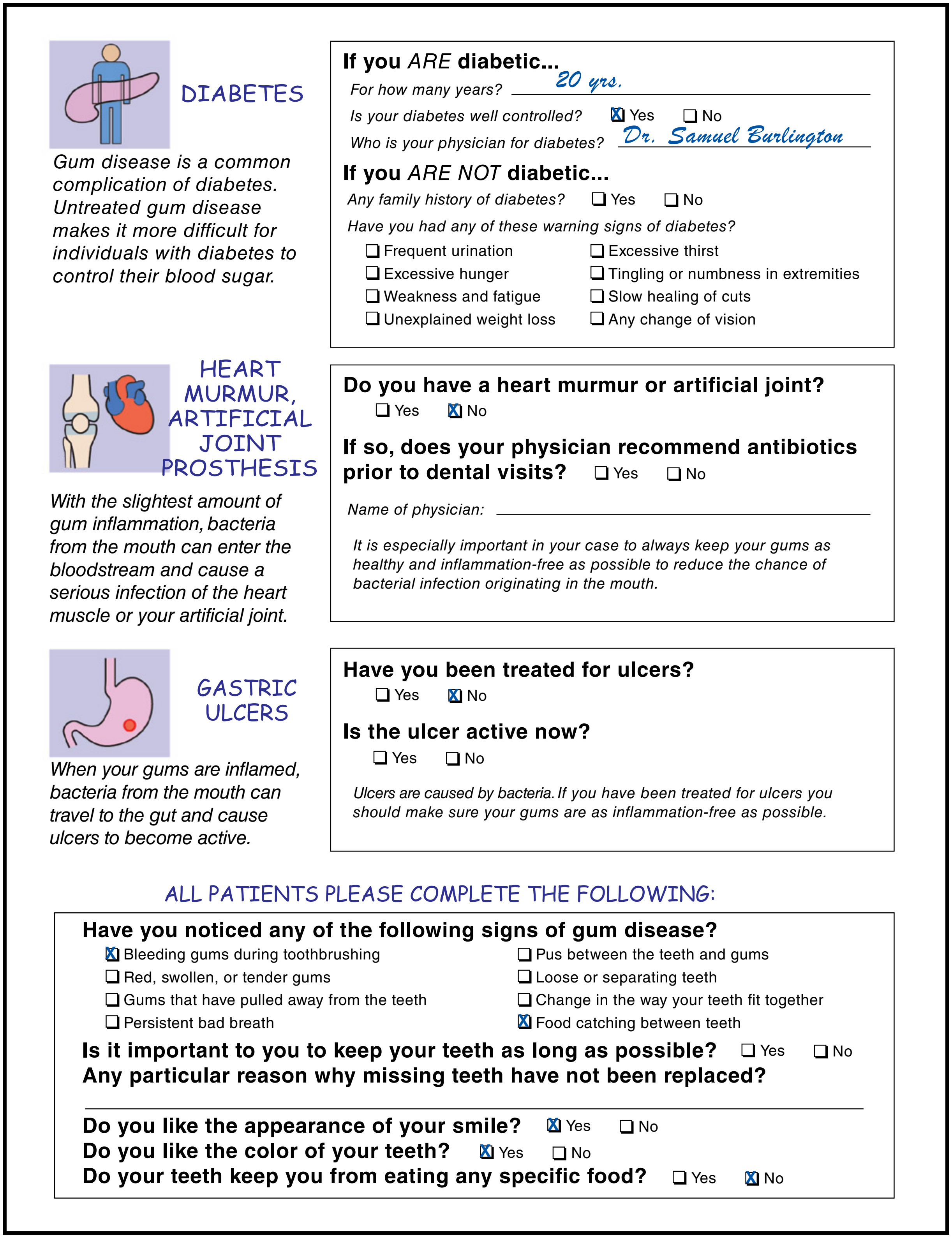
☐ Pregnant ☐ Nursing ☐ Osteoporosis
☐ Taking birth control pills
☐ Taking hormone supplements
☐ Infrequent care during previous pregnancies

over

A

Figure 10-9 **A.** Page 1 of Mr. Newcomer's Risk Questionnaire.

Courtesy of Timothy G. Donley, DDS, MSD, Bowling Green, KY, with modifications.



DIABETES

Gum disease is a common complication of diabetes. Untreated gum disease makes it more difficult for individuals with diabetes to control their blood sugar.

If you *ARE* diabetic...

For how many years? 20 yrs.

Is your diabetes well controlled? ☒ Yes ☐ No

Who is your physician for diabetes? Dr. Samuel Burlington

If you *ARE NOT* diabetic...

Any family history of diabetes? ☐ Yes ☐ No

Have you had any of these warning signs of diabetes?

- ☐ Frequent urination
- ☐ Excessive hunger
- ☐ Weakness and fatigue
- ☐ Unexplained weight loss
- ☐ Excessive thirst
- ☐ Tingling or numbness in extremities
- ☐ Slow healing of cuts
- ☐ Any change of vision

HEART MURMUR, ARTIFICIAL JOINT PROSTHESIS

With the slightest amount of gum inflammation, bacteria from the mouth can enter the bloodstream and cause a serious infection of the heart muscle or your artificial joint.

Do you have a heart murmur or artificial joint?
☐ Yes ☒ No

If so, does your physician recommend antibiotics prior to dental visits? ☐ Yes ☐ No

Name of physician: ____________

It is especially important in your case to always keep your gums as healthy and inflammation-free as possible to reduce the chance of bacterial infection originating in the mouth.

GASTRIC ULCERS

When your gums are inflamed, bacteria from the mouth can travel to the gut and cause ulcers to become active.

Have you been treated for ulcers?
☐ Yes ☒ No

Is the ulcer active now?
☐ Yes ☐ No

Ulcers are caused by bacteria. If you have been treated for ulcers you should make sure your gums are as inflammation-free as possible.

ALL PATIENTS PLEASE COMPLETE THE FOLLOWING:

Have you noticed any of the following signs of gum disease?

- ☒ Bleeding gums during toothbrushing
- ☐ Red, swollen, or tender gums
- ☐ Gums that have pulled away from the teeth
- ☐ Persistent bad breath
- ☐ Pus between the teeth and gums
- ☐ Loose or separating teeth
- ☐ Change in the way your teeth fit together
- ☒ Food catching between teeth

Is it important to you to keep your teeth as long as possible? ☐ Yes ☐ No

Any particular reason why missing teeth have not been replaced?

____

Do you like the appearance of your smile? ☒ Yes ☐ No

Do you like the color of your teeth? ☒ Yes ☐ No

Do your teeth keep you from eating any specific food? ☐ Yes ☒ No

B

Figure 10-9 B. Page 2 of Mr. Newcomer's Risk Questionnaire. (*continued*)

Courtesy of Timothy G. Donley, DDS, MSD, Bowling Green, KY, with modifications.

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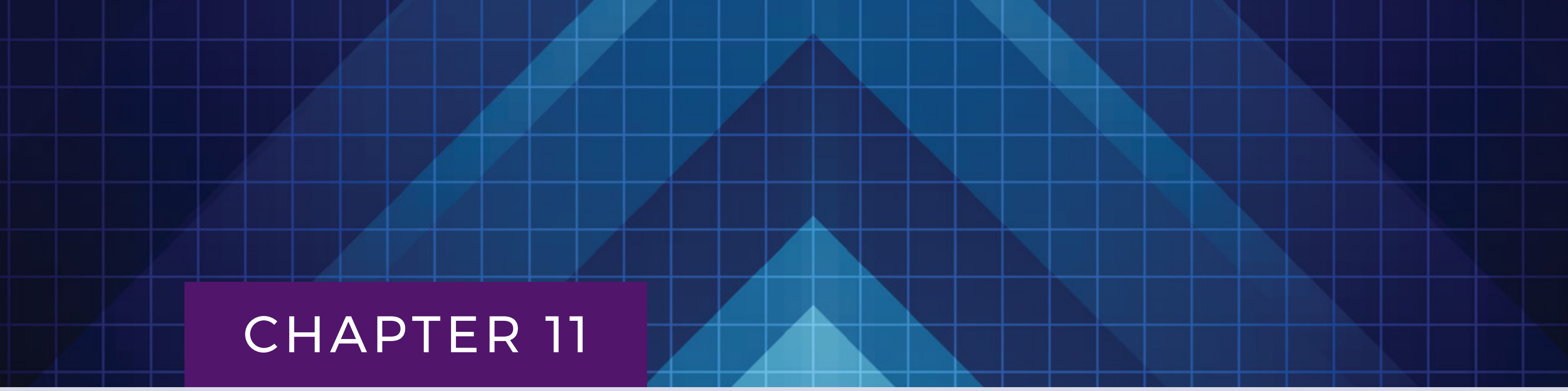

CHAPTER 11

Oral Biofilms

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  - General Characteristics of Bacteria
  - Microbial Communities
  - Oral Biofilms
  - Microbial Symbiosis Versus Dysbiosis
  - Species Capable of Colonizing the Mouth
- Section 2 The Structure and Colonization of Oral Biofilms
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  - Bacterial Attachment Zones
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CLINICAL APPLICATION

Caring for patients with periodontal diseases requires a comprehensive understanding of the concept of biofilms and how individual microbes interact with others within the biofilm community. This chapter discusses what is currently known about oral biofilms and sets the stage for the inevitable expansion of our knowledge about how these biofilms interact with the host (the human body).

LEARNING OBJECTIVES

- Describe the key structural features of Gram-positive and Gram-negative bacteria and explain the role of the cell envelope in the Gram-staining technique.
- Define the term *biofilm* and explain the benefits of bacteria residing in a biofilm.
- Describe the life cycle of a biofilm.
- Explain the significance of the extracellular protective matrix and fluid channels in a biofilm.
- Define *coaggregation* and explain its significance in bacterial colonization of the tooth surface.
- Define *quorum sensing* and explain its significance in coordinating and regulating microbial behavior and growth.
- Explain why systemic antibiotics and antimicrobial agents are not effective in eliminating dental plaque biofilm.

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- List several reasons why newer microbe detection methods have brought Socransky's microbial complexes model and the specific plaque hypothesis into question.
- Discuss the evolution of hypotheses to explain the role of bacteria in periodontal disease and how current hypotheses are distinct from the nonspecific plaque hypothesis and the specific plaque hypothesis.
- Discuss the hypothesis that plaque biofilm is necessary but not sufficient to cause destruction of the tissues of the periodontium and the implications for the treatment of individuals with periodontitis.

KEY TERMS

Microbe
Bacterium/bacteria
Cell envelope
Gram staining
Gram-positive bacteria
Gram-negative bacteria
Polymicrobial
Biofilm
Oral biofilm
Commensal bacteria
Symbiosis
Dysbiosis
Vertical transmission
Horizontal transmission
Acquired salivary pellicle
Coaggregation
Extracellular protective matrix
Extracellular polymeric substance
Microbial blooms
Mushroom-shaped microcolonies
Fluid channels
Quorum sensing
Tooth-associated plaque biofilms
Tissue-associated plaque biofilms
Unattached bacteria
Nonspecific plaque hypothesis
Specific plaque hypothesis
Ecological plaque hypothesis
Microbial homeostasis–host response hypothesis
Keystone pathogen–host response hypothesis

Section 1

Microbial Biofilms

Bacteria are everywhere in nature, including the oral cavity, which houses more microorganisms than there are people on the planet Earth. Further, it is estimated that 650 to 1,000 unique types of bacterial species populate the oral cavity.[1–5] These numbers can vary significantly from individual to individual and will inevitably increase as we discover previously unidentified microbial species using more advanced microbiological testing systems. Knowledge of several critical concepts of bacteria are fundamental to understanding the biological mechanisms/pathophysiology of periodontal disease.

The term **microbe** broadly refers to all types of microscopic organisms—namely bacteria, fungi, protozoa, and viruses. Frequently, the term *microbe* is inaccurately used to refer only to harmful and pathogenic microorganisms. However, it is a misconception that all microbes are pathogenic because some are innocuous. Key concepts discussed in this section include fundamental characteristics of bacteria, cell envelope structure, Gram staining, and the role of the biofilm in microbiology and microbial ecology.

1. **General Characteristics of Bacteria**
   a. **Description**
      1. **Bacterium** (plural, **bacteria**) are the smallest and simplest organisms which can only be viewed under a microscope.
      2. Although they are the smallest and simplest of all living organisms, bacteria are the most dominant living organisms—in terms of number—on the planet. In fact, there are between 10 million to 1 trillion species of bacteria, most of which are harmless to humans.
      3. Bacteria have existed on Earth far longer than any other organism and are the most abundant type of cell. Through millions of years of evolution, microorganisms have "learned" to adapt and survive in the oral environment.
      4. Bacteria reproduce quickly. This ability enables populations of bacteria to not only adapt rapidly even to sudden changes in their environment, but also copy and transfer valuable genetic information (i.e., DNA) to the next generation (offspring). The ability to adapt rapidly and transfer genetic information enhances bacterial survival and reproduction in the host (the human body).

   b. **Structure of the Bacterial Cell Envelope.** The bacterial **cell envelope** is a complex, multilayered structure that serves to protect the microorganism from the unpredictable and inhospitable external environment. One can imagine the bacterial cell envelope as being the "skin" of the microorganism. The physical structure of the cell envelope is the fundamental basis for a laboratory method known as **Gram staining**. Gram staining classifies bacteria into one of the two groups based on the structure of their cell envelope: Gram-positive microorganisms or Gram-negative microorganisms.

1. In 1884, Hans Christian Gram devised a staining technique to identify microorganisms under light microscopy. Today, this technique is known as Gram staining. Gram staining is a widely used diagnostic laboratory staining method that identifies bacteria based on the fundamental structural characteristics of the bacterial cell envelope. Depending on the permeability of the stain through the bacterial cell envelope, bacteria will appear either purple (Gram positive) or red/pink (Gram negative) under a light microscope.
2. The defining features of each type of bacteria are listed below and shown in **Figure 11-1**:

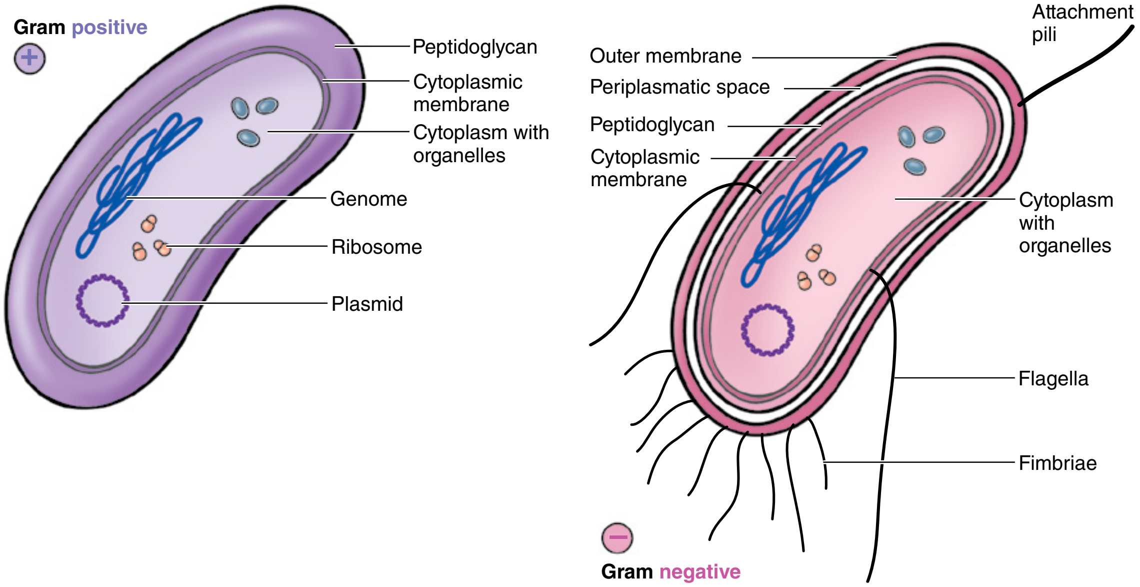

Figure 11-1 **Gram-Positive and Gram-Negative Bacterial Cell Envelopes.** Gram-positive bacteria have a thick peptidoglycan cell wall that retains the purple color when stained with crystal violet dye (the primary stain used in the Gram-staining technique). In contrast, Gram-negative bacteria have a thin peptidoglycan layer covered by an outer membrane that does not have the ability to readily absorb and retain the crystal violet dye. As a result, Gram-negative bacteria appear red or pink under the Gram-staining method. Gram-negative bacteria are believed to play an important role in periodontal disease.

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a. **Gram-Positive Bacteria (Purple Stain)**
   1. **Gram-positive bacteria** have a single, thick multilayered, mesh-like cell wall composed of peptidoglycan (a biological polymer composed of sugars and amino acids). This thick exterior layer lies above the cytoplasmic (plasma) membrane that faces the interior of the cell.
   2. It is this thick cell wall that binds and retains the purple color when stained with a dye known as crystal violet. Therefore, Gram-positive bacteria appear purple under a light microscope.

b. **Gram-Negative Bacteria (Red or Pink Stain)**
   1. **Gram-negative bacteria** have two membranes:
      a. The outer membrane faces the external environment. Structurally, it is composed of proteins and lipopolysaccharides (endotoxin). Lipopolysaccharides are important because they play a major role in the pathogenesis of Gram-negative bacterial infections.
      b. Below the outer membrane is an inner cytoplasmic (plasma) membrane that faces the interior of the cell.
   2. Sandwiched between the outer membrane and inner cytoplasmic membrane is a thin, single-layer cell wall composed of peptidoglycan. The thin cell wall does not readily absorb and retain the crystal violet dye. Therefore, the Gram-negative microbe appears red or pink under light microscope.

c. **Bacteria Are "Social Creatures"**
   1. For years, microbes were thought of as being dormant "bags" of enzymes. However, current research reveals that bacteria are "social creatures" that interact with one another and live together in complex microbial communities.
   2. In a similar manner to human cities, "microbial cities" contain different species of microbes (they are multicultural) that are spatially organized (properly designed and well structured) within the community.

Microbial Communities

In the past, microbes were studied as they grew artificially on culture plates in a laboratory. However, recent advances in microbiological testing have given us the ability to study microbes in their natural environment. These studies reveal that under natural conditions most microorganisms tend to live in complex communities attached to surfaces.

1. **Where Microbes Live.** Microbes rarely exist as planktonic (free-floating) forms. The majority live in complex **polymicrobial** (multispecies) biofilm communities attached to living surfaces (such as the root of a tooth) or nonliving surfaces (such as a prosthetic heart valve or a dental implant).
2. **Biofilms and Where They Form**
   a. **What Are Biofilms?**
      1. A **biofilm** is a complex and dynamic microbial community—containing a diverse array of many types of microbial species (bacteria, fungi, and viruses)—embedded within an extracellular self-protective matrix that adheres to a living or nonliving surface. These polymicrobial biofilm communities are niches for thousands of uniquely different species of microorganisms that interact with each other to thrive in the oral envrionment.[6]
      2. Within the biofilm, the microorganisms synthesize and secrete an extracellular protective matrix that is composed of sugars and proteins. This protective matrix surrounds the bacteria and makes them more tolerant of the harsh conditions of the oral cavity and resistant to antibacterial treatment.
      3. By some estimates, 65% of all diseases may be biofilm induced. Biofilm-induced diseases include tuberculosis, cystic fibrosis, subacute bacterial endocarditis, and periodontal disease.
   b. **Where Do Biofilms Form?**
      1. Biofilms are everywhere in nature (**Figure 11-2**). *Biofilm* may seem like a new term, but everyone encounters biofilms on a regular basis. The plaque biofilm that forms on teeth, the slime in fish tanks, and the slime deposit that clogs the sink drain are all examples of biofilms. Even the slimy rocks in a stream of water are coated with biofilm.

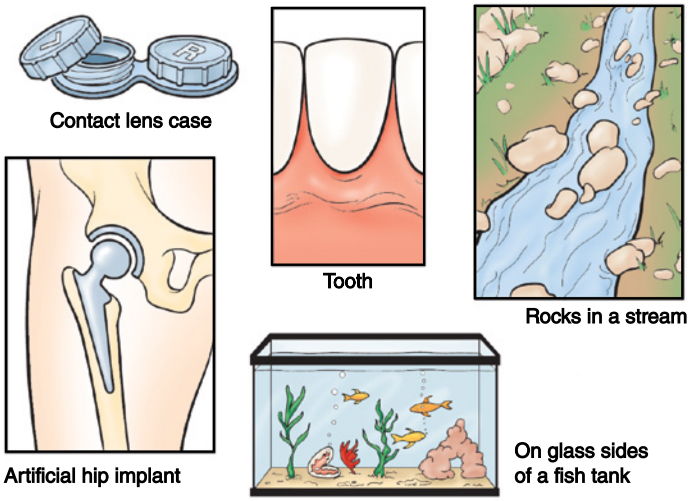

Figure 11-2 Biofilm Environments. Biofilms are found nearly everywhere in nature. They have a major impact on human health.

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2. Biofilms can exist on any solid surface that is exposed to a microbe-containing fluid. Biofilms may form on living or nonliving surfaces and can be prevalent in natural, industrial, and hospital settings. Biofilms thrive in dental unit water and suction lines and have been shown to be the primary source of contaminated water delivered by dental units.

c. **How Quickly Do Biofilms Form?** Experimental laboratory studies show that free-floating bacteria—for example, *Staphylococci* spp., *Streptococci* spp., *Pseudomonas* spp., and *Escherichia coli*—coalesce on a surface to form a biofilm very quickly.[7,8] A typical timeline for formation is as follows:
   1. Within minutes: Free-floating microbes attach to a surface.
   2. Within 2 to 4 hours: Microbes form strongly attached microcolonies.
   3. Within 6 to 12 hours: Microbes produce an initial extracellular protective matrix and become increasingly resistant to antiseptics and antibiotics.
   4. Within 2 to 4 days: The biofilm evolves into fully mature biofilm colonies that are extremely resistant to antibiotics; can rapidly recover from mechanical disruption and reform a mature biofilm within 24 hours.

d. **How Do Biofilms Mature?** As depicted in **Figure 11-3**, the first step is colonization of the tooth surface. Next, additional microbes join with the early colonizers to form a complex microbial community.

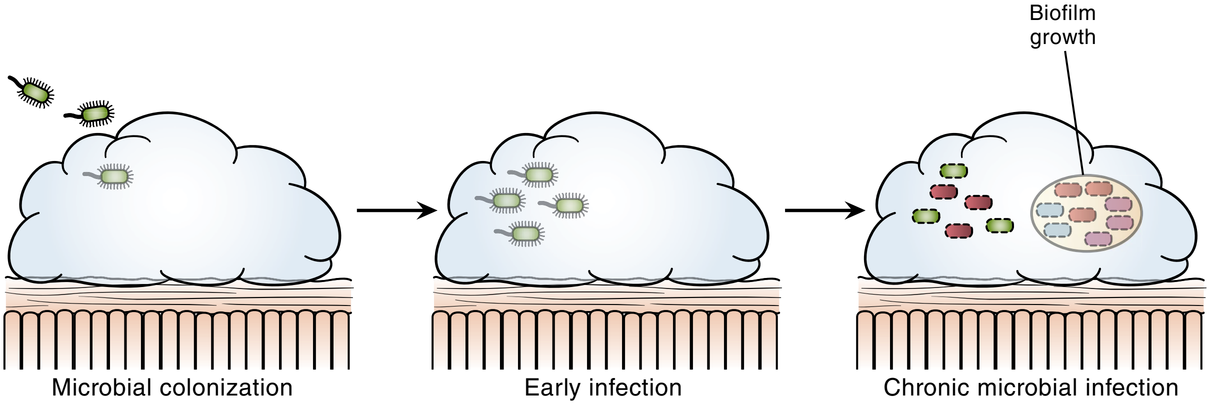

Figure 11-3 Biofilm Formation. Once microbes attach to the tooth surface, biofilms form quickly. Biofilm formation plays a major role in the onset of periodontal diseases.

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e. **How Do Mature Biofilms Protect Bacteria?** Biofilms greatly enhance the ability of inhabiting microbes to withstand the host's immune system, antimicrobials, and environmental stresses. Microbes in a biofilm can resist factors that would easily kill these same microbes when in a free-floating state.[9] The biofilm offers three inherent features that enable inhabiting microbes to tolerate various external stresses.
   1. **Blocking.** The extracellular protective matrix protects microbes by preventing large molecules (e.g., antibodies) and inflammatory cells from penetrating deeply into the biofilm matrix. Mature biofilms may even block small molecules such as antimicrobial agents.[10] Moreover, research shows that standard oral doses of antibiotics—which effectively kill free-floating bacteria—may not be able to fully penetrate through the biofilm, thereby having little to no antimicrobial effect against the same type of bacteria residing inside the biofilm.
   2. **Mutual Protection**
      a. A unique property of biofilms is the cooperative protective effects that different species of bacteria can provide to each other. For example, within the biofilm, antibiotic-resistant bacteria may secrete protective enzymes that shield neighboring non-antibiotic–resistant bacteria.[11]
      b. Microbes that are part of the normal (indigenous) flora of the oral cavity may offer natural protection for the host. Indigenous microbes may block pathogenic species from adhering to the existing biofilm and the mucosal surfaces. Thus, the biofilm can protect itself and the host from overgrowth of pathogenic bacteria.[12]

3. **Hibernation (Quiescence).** Another survival strategy that many bacteria in biofilms have developed is for a portion of the larger microbial population to hibernate (lay dormant) until environmental conditions become more favorable to support its growth. Because some antibiotics are only effective on metabolically active bacteria, hibernating bacteria in biofilms are unaffected by these types of antibiotics.[13,14]

Oral Biofilms

An **oral biofilm** is a polymicrobial, three-dimensional community embedded in a protective matrix that consists of microbial metabolic products and/or host components, such as salivary glycoproteins.[15] In health, the bacteria in the biofilm maintain a harmonious balance by keeping each other in check so that no one specific bacterial strain can dominate the biofilm community. However, if the biofilm remains intact (by not physically disrupting it) and is allowed to further develop and mature, an imbalance may occur within the biofilm community where certain pathogenic species become dominant while other species become weaker. **Figure 11-4** shows an artist's representation of a mature oral biofilm.

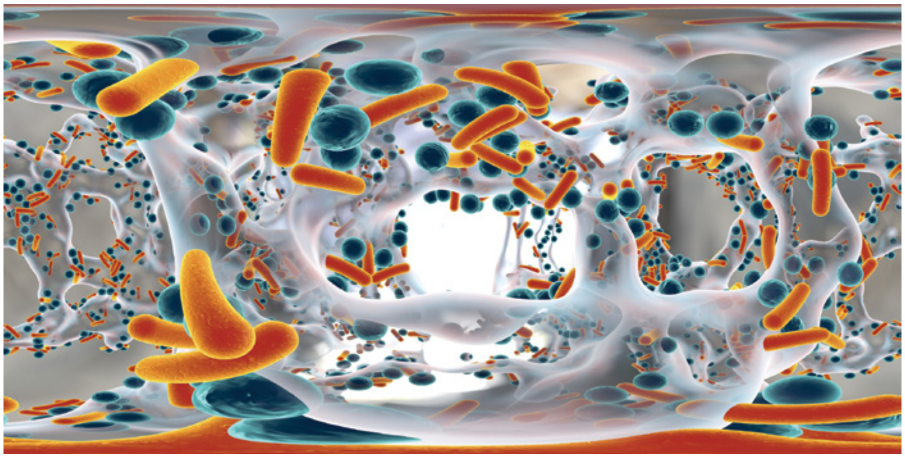

Figure 11-4 A Polymicrobial Biofilm. This three-dimensional illustration depicts a 360-degree spherical panorama view inside an antibiotic-resistant mature biofilm.

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Microbial Symbiosis Versus Dysbiosis

1. **Resident Microbes of the Oral Cavity.** A multitude of bacterial, viral, and fungal species inhabit the oral cavity. Many of these can associate to form biofilms, which are resistant to mechanical stress or antibiotic treatment.
   a. The oral cavity is inhabited by indigenous, resident bacteria, known as **commensal bacteria**. Commensal bacteria are part of the normal flora in the mouth, living in the oral cavity all the time and causing no problems. Further, as the natural, indigenous inhabitants of the oral cavity, commensal microorganisms play an important role in contributing to host defense by defending against colonization by opportunistic pathogenic bacteria.
   b. In a healthy state, all epithelial-lined surfaces (including the oral cavity) are colonized by a polymicrobial biofilm that is predominantly composed of commensal microorganisms.

2. **A Mutually Beneficial Relationship.** In health, a symbiotic (mutually beneficial) relationship exists between the host and commensal microorganisms.
   a. The commensal microorganisms contribute to host nutrition, maintain a robust immune system, and provide a cover over the mucous membranes (preventing the invasion and an overgrowth of pathogenic bacteria).[16]
   b. Conversely, the host provides the bacteria with nutrients and a stable environment in which to survive (**Figure 11-5**).[17]
   c. **Symbiosis** translates to living in harmony. In health, commensal microbes in the oral biofilm have a symbiotic relationship with the host.[18]

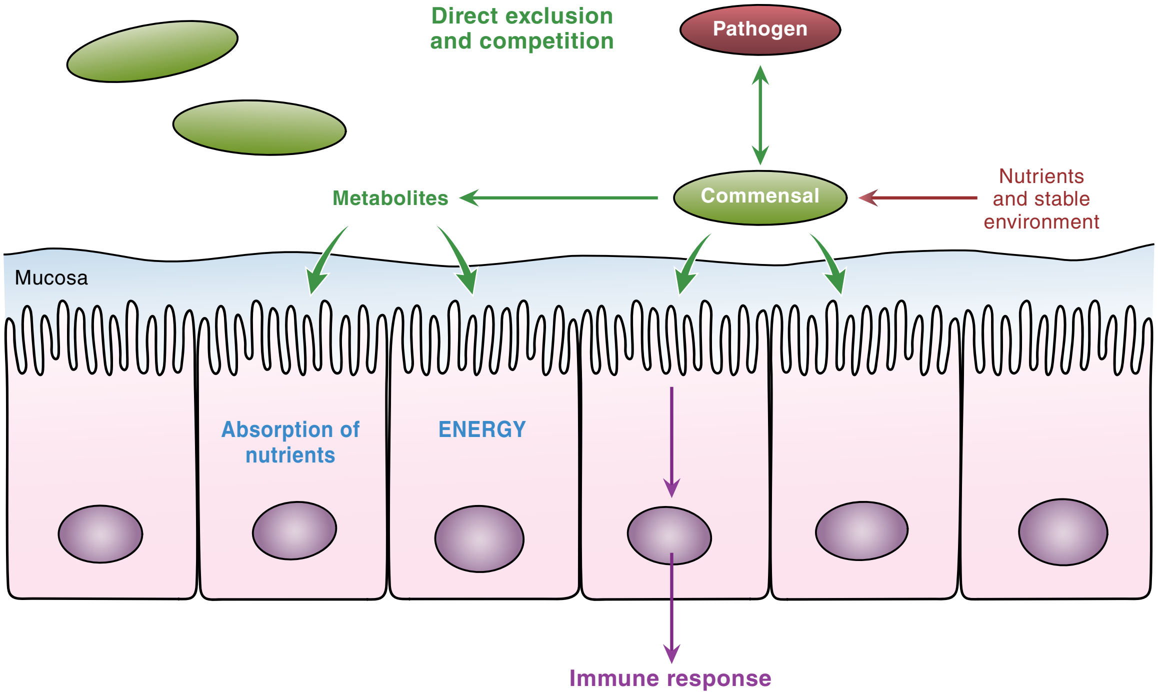

Figure 11-5 Commensal Bacteria Cross Talk with the Host. Commensal bacteria supply the host with essential nutrients and defend the host against opportunistic pathogens. In return, the host provides the bacteria with vital nutrients and a stable environment. This interaction facilitates a mutually protective relationship between the host and the commensal bacteria.

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3. **Microbial Imbalance: The "Bad Guys" Take Over the Biofilm**
   a. **Early Dysbiosis**
      1. Dysbiosis is the opposite of symbiosis. **Dysbiosis** is a term for a microbial imbalance on or inside the body.
         a. It essentially means there is an imbalance of microbial colonies. The bacteria maintain a harmonious balance in a healthy biofilm by keeping each other in check so no one specific strain can dominate.
         b. Dysbiosis can result if certain species of microbes become dominant while other species become weaker.
         c. If the oral biofilm is not disrupted frequently (through the daily use of a toothbrush and interdental cleaning aids) and is allowed to mature, the conditions within in it start to favor bacterial species that elicit a stronger host response. In turn, the stronger host response leads to the development of gingival inflammation.
         d. This state is known as initial dysbiosis or incipient dysbiosis because, in most individuals, gingivitis does not progress to periodontitis (**Figure 11-6**).

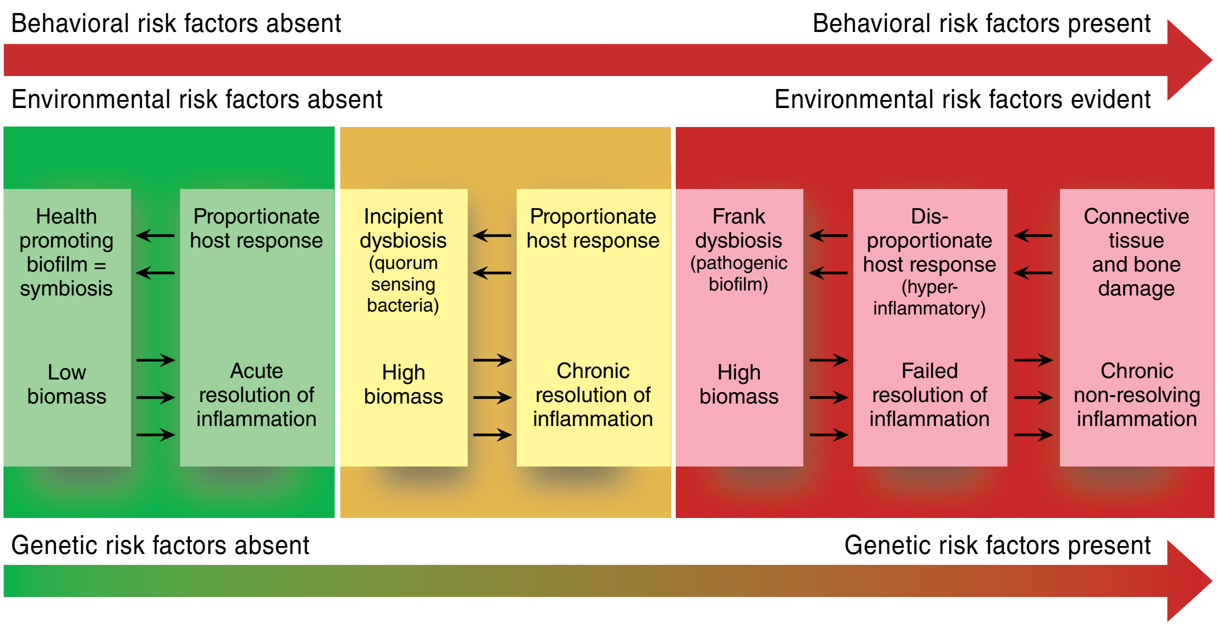
Behavioral risk factors absent
Behavioral risk factors present
Environmental risk factors absent
Environmental risk factors evident
Health promoting biofilm = symbiosis
Low biomass
Proportionate host response
Acute resolution of inflammation
Incipient dysbiosis (quorum sensing bacteria)
High biomass
Proportionate host response
Chronic resolution of inflammation
Frank dysbiosis (pathogenic biofilm)
High biomass
Dis-proportionate host response (hyper-inflammatory)
Failed resolution of inflammation
Connective tissue and bone damage
Chronic non-resolving inflammation
Genetic risk factors absent
Genetic risk factors present

Figure 11-6 **Model of Host–Microbe Interactions.** If the oral biofilm is not disrupted frequently and is allowed to further develop and mature, the conditions within it start to favor bacterial species that elicit a stronger host response, leading to the development of gingival inflammation. In susceptible individuals, dysbiosis triggers an exaggerated host response which results in progression from gingivitis to periodontitis.

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b. **Established Dysbiosis**
   1. Recent research has indicated that dysbiosis in the oral cavity can lead to periodontitis. The development of oral dysbiosis is likely to occur over an extended period of time, gradually changing the symbiotic host–microbe relationship to a pathogenic one.[18]
   2. In susceptible patients, dysbiosis triggers an inappropriate and excessive host response (to the unhealthy change in the oral biofilm) that results in irreversible periodontal tissue damage (progression from gingivitis to periodontitis).[19]

4. **Control of Microbial Growth Within a Mature Oral Biofilm**
   a. **Biofilm Bacteria Are Resistant to Antibiotics and Antimicrobial Agents**
      1. Bacteria living in a biofilm are unusually resistant to systemic antibiotics (in dentistry, usually administered in pill form) and antimicrobials (placed locally in the oral cavity or taken as an oral rinse).[20]
      2. Antibiotic doses that kill free-floating bacteria, for example, need to be increased as much as 1,500 times to kill plaque biofilm bacteria (and at these high doses, the antibiotic would harm the patient before the biofilm bacteria!).[21]
      3. Antimicrobial agents work best when used in conjunction with mechanical cleaning that removes or disrupts the structural integrity of the dental plaque biofilm.[22]
   b. **Physical Removal of Dental Plaque Biofilms Is Essential**
      1. Control of bacteria in dental plaque biofilms is best achieved by the physical disruption (by the means of daily toothbrushing, the use of an interproximal cleaning aid, and routine periodontal debridement) of the biofilm structure.
         a. It takes some time for the complex structure of a mature plaque biofilm to form.
         b. Mechanical cleaning physically breaks up the biofilm and forces the bacteria to start over with initial attachment, initial colonization, secondary colonization, and, finally, to become a mature biofilm.
         c. In areas that are cleaned regularly, a mature biofilm will not have enough time to develop and mature. Thus, the cleaner the tooth surface, the less complex the biofilm.
      2. Notably, as opposed to the mechanical removal of supragingival plaque biofilm, toothbrushes and interproximal cleaning aids (such as dental floss or toothpicks) cannot reach the *sub*gingival plaque biofilm (plaque biofilm residing inside a pocket). For this reason, frequent periodontal instrumentation of subgingival root surfaces by a trained dental clinician is an essential component in the treatment and management of periodontal disease.

5. **Transmission of Biofilm Bacteria**
   a. Molecular epidemiology techniques that isolate DNA provide evidence that bacteria in the biofilm are transmissible.[23]
   b. The term *transmissible* refers to the transfer of bacteria—that may or may not have the capability of producing an infection—from one individual to another. The transmission may occur either via direct contact, such as the transfer of saliva through kissing, or indirect contact via salivary substances on inanimate objects, such as the sharing of saliva-coated toys between children at a nursery school.
   c. The most common route of transmission is via the sharing of saliva between a caregiver and a child. This route of transmission is known as **vertical transmission** because it occurs between members of *different* generations (e.g., transmission from mother to infant). To highlight, *Aggregatibacter actinomycetemcomitans* and *Porphyromonas gingivalis* strains isolated from the saliva of parents and children within the same family exhibited identical restriction endonuclease DNA fragment patterns.[24–27] In contrast, a second, less common, route of transmission is via the sharing of saliva between individuals of the *same* generation (e.g., spouses). This is known as **horizontal transmission**. Kissing is the primary means by which saliva and its bacterial contents are transmitted via the vertical and horizontal routes.[26,28,29]
   d. All bacteria have the capability to be transmitted between individuals, but it does not necessarily always lead to infectious disease. In epidemiology, the term *infectious disease* is any disorder (or illness) that results from the transmission of the infectious agents (e.g., periodontal pathogen), followed by the eventual manifestations of the disease. With respect to periodontal disease, although intraoral transmission of periodontal pathogens can occur between individuals, periodontal disease per se is not an infectious disease. For example, if there is transmission of pathogenic-loaded saliva between two individuals, periodontal disease will not necessarily develop in the other individual. This could be attributed to differences in individual susceptibility to periodontal disease, host immune response, and the lack of pathogenicity of the transmitting microorganisms. Thus, although periodontal pathogens are transmissible, periodontal disease, itself, is not an infectious disease (**Figure 11-7**).
   e. **Table 11-1** provides a pronunciation guide to bacteria commonly associated with plaque biofilms.

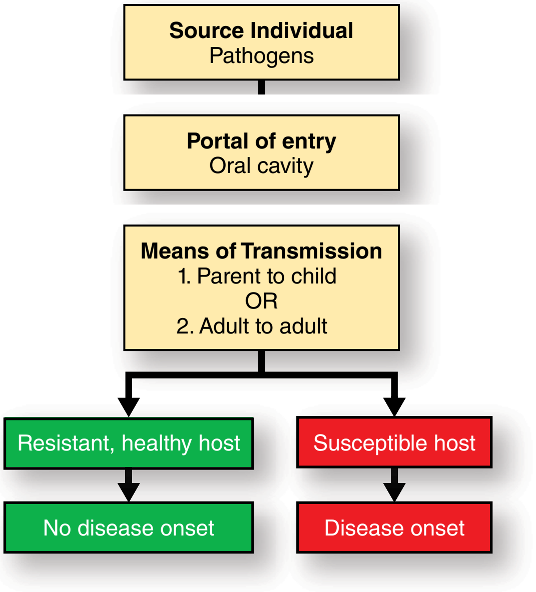

Figure 11-7 **Disease Transmission and Host Susceptibility.** The human mouth is a natural reservoir for many types of microorganisms that can be transferred from one host to another. Some individuals possess an immune response or certain genetic factors that makes them resistant to disease onset. Other individuals, unfortunately, may not have these factors and will be susceptible to disease. Note that, although periodontal pathogens are transferrable, this does not lead to infection of the host because of natural host defenses and bacterial antagonism with commensal microorganisms.

Table 11-1 **Pronunciation Guide to Bacterial Tongue Twisters**

Name	Pronunciation Guide
Aggregatibacter actinomycetemcomitans	ag-gre-gat-eee-bac-ter act-tin-oh-my-see-tem-comb-ah-tans
Tannerella forsythia	tann-er-ella fawr-sith-ee-uh
Fusobacterium nucleatum	fuse-so-back-tier-EEE-um nu-klee-ah-tum
Porphyromonas gingivalis	pour-fy-roh-mo-nas ging-jih-val-lis

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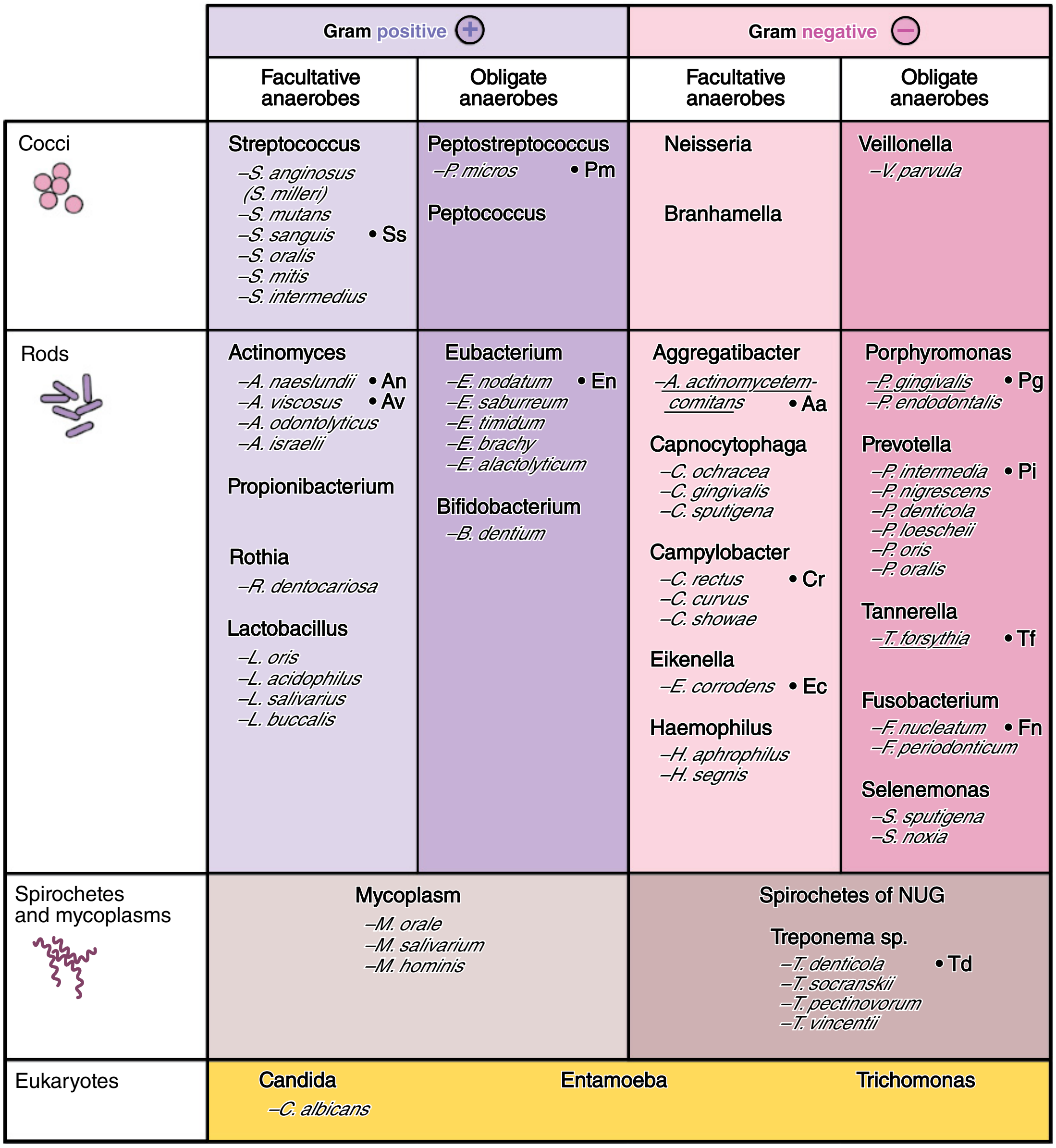
Species Capable of Colonizing the Mouth

Many different species and subspecies can colonize the mouth. **Figure 11-8** provides a few examples of the over 600 cultivable species.



	Gram positive +		Gram negative −	
	Facultative anaerobes	Obligate anaerobes	Facultative anaerobes	Obligate anaerobes
Cocci	Streptococcus –*S. anginosus* (*S. milleri*) –*S. mutans* –*S. sanguis* • Ss –*S. oralis* –*S. mitis* –*S. intermedius*	Peptostreptococcus –*P. micros* • Pm Peptococcus	Neisseria Branhamella	Veillonella –*V. parvula*
Rods	Actinomyces –*A. naeslundii* • An –*A. viscosus* • Av –*A. odontolyticus* –*A. israelii* Propionibacterium Rothia –*R. dentocariosa* Lactobacillus –*L. oris* –*L. acidophilus* –*L. salivarius* –*L. buccalis*	Eubacterium –*E. nodatum* • En –*E. saburreum* –*E. timidum* –*E. brachy* –*E. alactolyticum* Bifidobacterium –*B. dentium*	Aggregatibacter –*A. actinomycetem-comitans* • Aa Capnocytophaga –*C. ochracea* –*C. gingivalis* –*C. sputigena* Campylobacter –*C. rectus* • Cr –*C. curvus* –*C. showae* Eikenella –*E. corrodens* • Ec Haemophilus –*H. aphrophilus* –*H. segnis*	Porphyromonas –*P. gingivalis* • Pg –*P. endodontalis* Prevotella –*P. intermedia* • Pi –*P. nigrescens* –*P. denticola* –*P. loescheii* –*P. oris* –*P. oralis* Tannerella –*T. forsythia* • Tf Fusobacterium –*F. nucleatum* • Fn –*F. periodonticum* Selenemonas –*S. sputigena* –*S. noxia*
Spirochetes and mycoplasms	Mycoplasm –*M. orale* –*M. salivarium* –*M. hominis*		Spirochetes of NUG Treponema sp. –*T. denticola* • Td –*T. socranskii* –*T. pectinovorum* –*T. vincentii*	
Eukaryotes	Candida –*C. albicans*		Entamoeba	Trichomonas

Figure 11-8 **Examples of Microorganisms Capable of Colonizing the Mouth.**

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Section 2

The Structure and Colonization of Oral Biofilms

It can be very helpful for clinicians to develop an awareness of how biofilms form and mature on a tooth surface. The description below follows biofilm development starting with a recently cleaned tooth surface and progresses to a fully developed biofilm on that tooth surface.

Stages of Polymicrobial Biofilm Formation

Formation of the plaque biofilm begins with the attachment of free-floating microbes to the outer surface of the tooth pellicle. There are five stages of biofilm development: initial attachment, irreversible attachment, maturation I, maturation II, and dispersion.[30–33] These five stages are depicted in **Figure 11-9**.

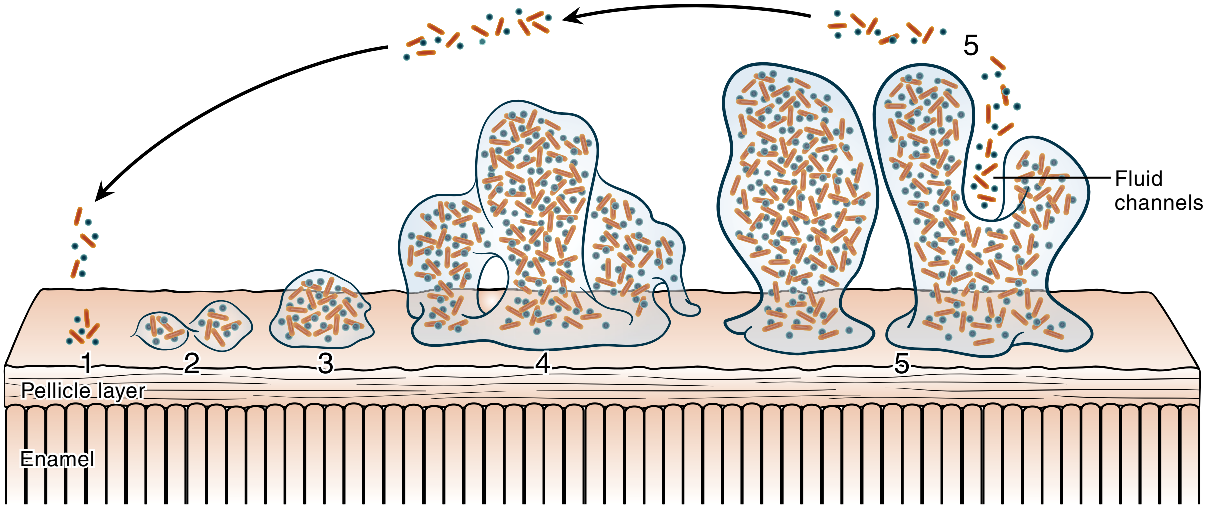

Figure 11-9 **Five Phases in the Formation of a Biofilm.** The five stages of biofilm development: (1) initial attachment, (2) irreversible attachment, (3) maturation I, (4) maturation II, and (5) dispersion.

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1. **The Five Stages of Plaque Biofilm Development in the Oral Cavity**
   a. **Stage 1—Initial Attachment of Microbes to Pellicle**
      1. Formation
         a. Within seconds to minutes following plaque removal, a salivary film immediately forms over the clean tooth surface. This film, known as the **acquired salivary pellicle**, is composed of a variety of salivary glycoproteins (mucins) and antibodies. The purpose of the acquired salivary pellicle is to protect the enamel from acidic activity (breakdown of the enamel from the low pH oral environment).
         b. Within a few hours after acquired salivary pellicle formation, free-floating (planktonic) microbes begin to attach to the outer surface of the pellicle. Under natural conditions, most bacteria are not free-floating, but tend to attach to surfaces. **Figure 11-10** shows a scanning electron micrograph of plaque-forming bacteria.
      2. **What Does It Take to Stick?**
         a. Motile bacteria use flagella to overcome hydrodynamic (fluid) forces within the oral cavity. One can think of the flagella as being a whip-like tail that gives the microorganism the capability to "swim" in fluid.
         b. Some bacteria possess attachment structures such as extracellular adhesive substances or extracellular hair-like, filamentous structures that extend from the surface of the bacteria and enable them to attach to the surface of the acquired salivary pellicle that coats the tooth surface. The hair-like, filamentous structures are termed *fimbriae*.

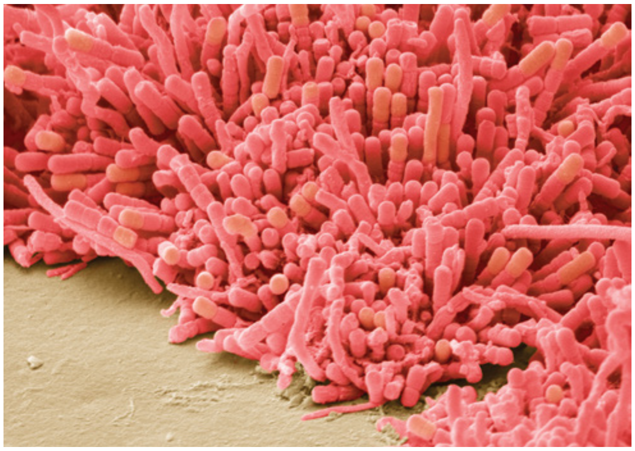

Figure 11-10 Plaque-Forming Bacteria. Scanning electron micrograph of plaque-forming bacteria growing on top of the outer surface of an acquired salivary pellicle.

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    c. The decision to "stick" to the tooth is not absolute; initial attachment is dynamic and reversible, during which the microbes can detach and rejoin the free-floating population if disturbed by hydrodynamic forces or in response to nutrient availability.

b. **Stage 2—Permanent Attachment**
  1. Permanent attachment is attained by microbes that can weather hydrodynamic (fluid) forces and maintain a steadfast grip on the tooth surface.
  2. At this stage, the microbes begin producing substances that stimulate other free-floating bacteria to join the community. Thus, the initial attachment of the microbes serves as the scaffolding to which other species may adhere.
  3. This process by which genetically distinct bacteria become attached to one another is commonly referred to as **coaggregation**. As a result, the composition of early colonizers determines which microbes colonize at later time points, consequently influencing the development of the biofilm.[34]

c. **Stage 3—Maturation Phase I: Self-Protective Matrix Formation**
  1. Once firmly attached, the bacteria begin to secrete an enveloping protective substance, known as an **extracellular protective matrix** (or an **extracellular polymeric substance**). **Figure 11-11** shows an example of microbes in an oral biofilm surrounded by an extracellular protective matrix.

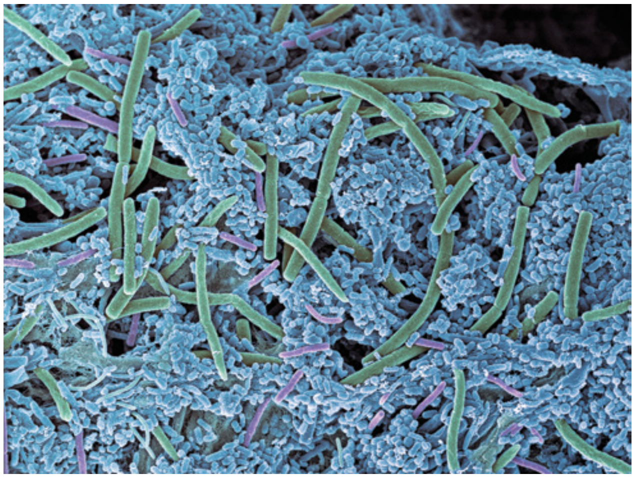

Figure 11-11 Protective Extracellular Matrix. Scanning electron micrograph of dental plaque biofilm showing bacteria [green stain] and the protective matrix [blue stain].

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   2. The exact composition of the extracellular protective matrix varies according to the microorganisms in the biofilm, but generally consists of proteins, glycolipids, and bacterial DNA.[11]
   3. Thus, the microbes are cocooned in a self-protective matrix. Inside the host, the matrix protects biofilm microbes from exposure to host immune defenses (such as phagocytosis) and antibiotic treatments. As a result, the biofilm persists, eventually establishing a chronic disease state (periodontitis).

d. **Stage 4—Maturation Phase II: Mushroom-Shaped Microcolonies**
   1. **Microcolony Formation.** At this stage, the biofilm continues to grow through a combination of cell division and recruitment. **Microbial blooms** are periods when specific species or groups of species grow at rapidly accelerated rates. The proliferating microbes begin to grow away from the tooth.
      a. The microbes cluster together to form **mushroom-shaped microcolonies** that are attached to the tooth surface at a narrow base. The result is the formation of complex collections of different microbes linked to one another.
      b. Each microcolony is a bustling community of microbes that actively exchanges essential nutrients and genetic information that play a pivotal role in creating a favorable living environment for the resident microbes.
         1. Environmental conditions within each microcolony vary radically. The environmental conditions among several microcolonies may include differences in oxygen concentration, pH, and temperature.
         2. The differing environmental conditions within a biofilm mean that the microbial population is very diverse—with each different species preferring a certain environment within the biofilm.
            a. This diversity helps to ensure the survivability of the plaque biofilm in widely varying oral conditions.
            b. If the plaque biofilm was composed of only one type of microbial species, it would be much more likely that a toxic agent or condition would destroy the biofilm.
   2. **Internal Organization of Mature Biofilm**
      a. **Layers and Layers of Microbes.** The biofilm develops by stacking one microbial species on top of another. A mature dental biofilm does not consist of only one species of microbe, rather it is polymicrobial.
      b. **Fluid Channels**
         1. As the plaque biofilm develops, a series of **fluid channels** are formed that penetrate the extracellular protective matrix.
         2. These fluid channels direct fluids into and around the biofilm, bringing nutrients and oxygen to the microbes and carrying waste products away.
         3. The fluids include everything from saliva to any beverages consumed.
      c. **Cell-to-Cell Communication System**
         1. Direct cell-to-cell interaction occurs among the microbes in the biofilm.
         2. The microcolonies use chemical signals to communicate with each other.
         3. This cell-to-cell communication also results in the transfer of genes among microbes.
      d. **Bacterial Signaling**
         1. Bacterial communication occurs when microbes within the biofilm release and sense small proteins (signaling molecules). This type of communication among microbes is termed **quorum sensing**.
         2. Microbes in the biofilm use quorum sensing to trigger events such as adhesion of additional microbes to the biofilm and formation of the extracellular protective matrix.
         3. In quorum sensing, individual bacteria "talk" with other bacteria to share information that is critical for their growth and survivability. In the oral environment, this is especially important because bacteria need to adapt rapidly to any sudden adverse change in environmental conditions, such as a sudden change in oral pH or in oxygen concentration. Continual back-and-forth communication permits microbes to coordinate their behavior collectively in order to adapt to changes in the local environment. This ensures survivability of all microorganisms living within the biofilm.
         4. Quorum sensing is not limited to bacteria from a single species communicating with bacteria from the same species. Rather, bacteria from one species can communicate with those from other species (interspecies communication) through the same process. The capacity for interspecies communication is critical for the growth, coordination, and development of the polymicrobial community residing within the oral biofilm.

5. Ongoing research is looking into treatment modalities that could interfere with the quorum sensing system. If this is possible, then there may be a way to therapeutically block the communication between microorganisms, thereby inhibiting the growth of the biofilm.

e. **Stage 5—Dispersion: Escape from the Matrix**

1. Dispersal of microbes from the biofilm colony is an essential stage of the biofilm life cycle. Dispersal enables biofilms to spread and colonize new tooth surfaces.

Bacterial Colonization and Succession of Oral Biofilms

1. **Colonization**

a. **Sequence of Bacterial Colonization**

1. Early bacterial colonizers of the tooth surface include many streptococcal species, such as *Streptococcus mitis* and *Streptococcus oralis*, that can attach to the tooth pellicle as well as to each other.[35,36]
2. The early bacterial colonizers release chemical signals that indicate to the next group of bacteria that conditions are favorable for them to join the biofilm. This best characterizes quorum sensing, which is essential in the coordination and regulation of the activities of a polymicrobial biofilm community.
3. Free-floating microbes cannot join the biofilm until the conditions are favorable. The succession of bacteria joining the biofilm is comparable to elementary school students who are asked to line up in alphabetical order as their teacher calls out their names. Students whose last names start with the letter "O" cannot get in line until all the students whose last names start with "M" and "N" have taken their place in line.
4. A mature plaque biofilm is a very complex collection of multiple microbial species. **Figure 11-12** illustrates the complex structure of a mature biofilm.

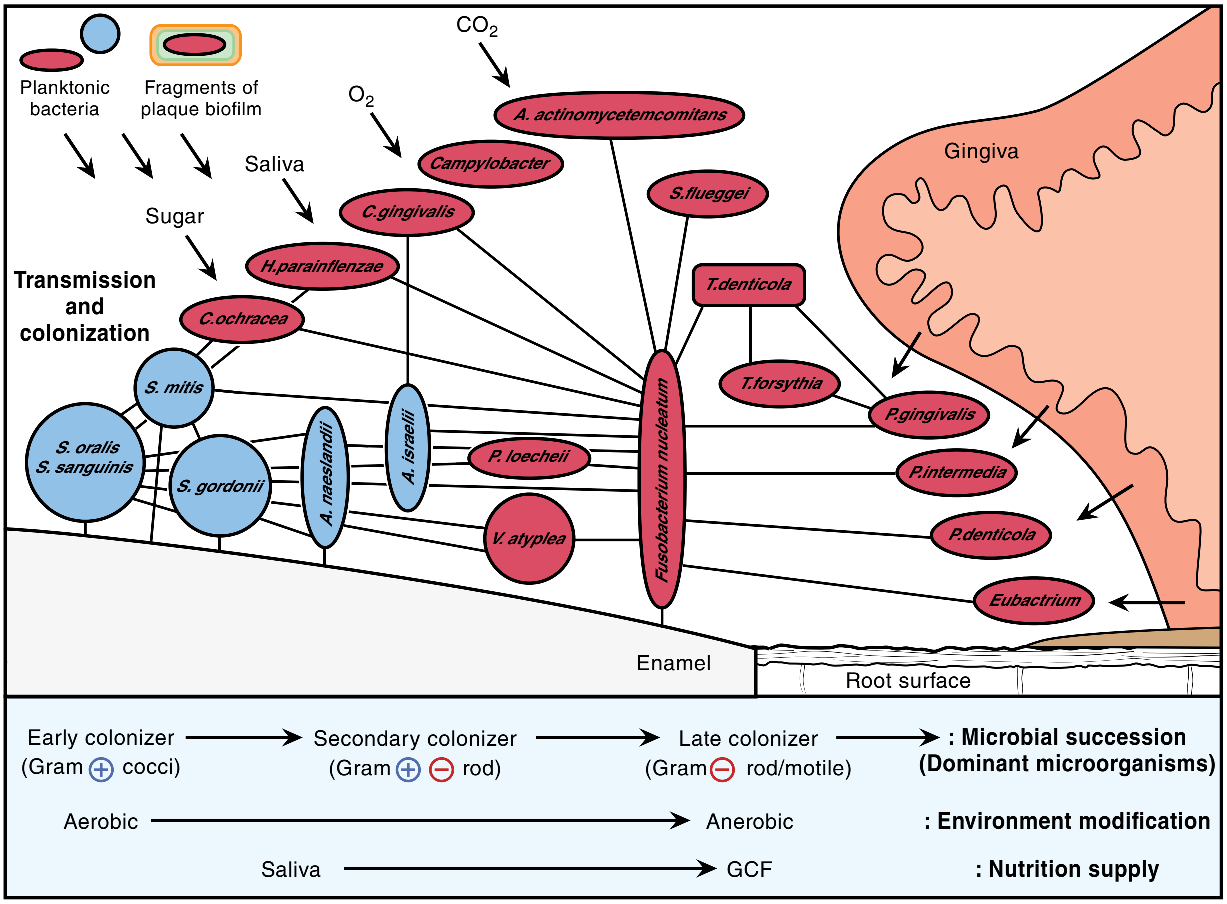
Planktonic bacteria
Fragments of plaque biofilm
CO$_2$
O$_2$
Saliva
Sugar
Transmission and colonization
A. actinomycetemcomitans
Campylobacter
C.gingivalis
S.flueggei
H.parainflenzae
C.ochracea
T.denticola
T.forsythia
S. mitis
S. oralis
S. sanguinis
S. gordonii
A. naeslandii
A. israelii
P. loecheii
V. atyplea
Fusobacterium nucleatum
P.gingivalis
P.intermedia
P.denticola
Eubactrium
Gingiva
Enamel
Root surface
Early colonizer (Gram + cocci)
Secondary colonizer (Gram + − rod)
Late colonizer (Gram − rod/motile)
: Microbial succession (Dominant microorganisms)
Aerobic
Anerobic
: Environment modification
Saliva
GCF
: Nutrition supply

Figure 11-12 Oral Biofilm. An oral biofilm is an organized, structural collective of different, genetically distinct microorganisms adhering to the tooth surface and/or attaching to each other (coaggregation).

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Bacterial Attachment Zones

The zones of subgingival bacterial attachment (**Figure 11-13**) are the tooth surface and the epithelial lining of the periodontal pocket. Bacteria also may attach to other bacteria that are attached to one of these surfaces. In addition to the attached bacteria, some bacteria remain free-floating in the gingival sulcus or pocket.

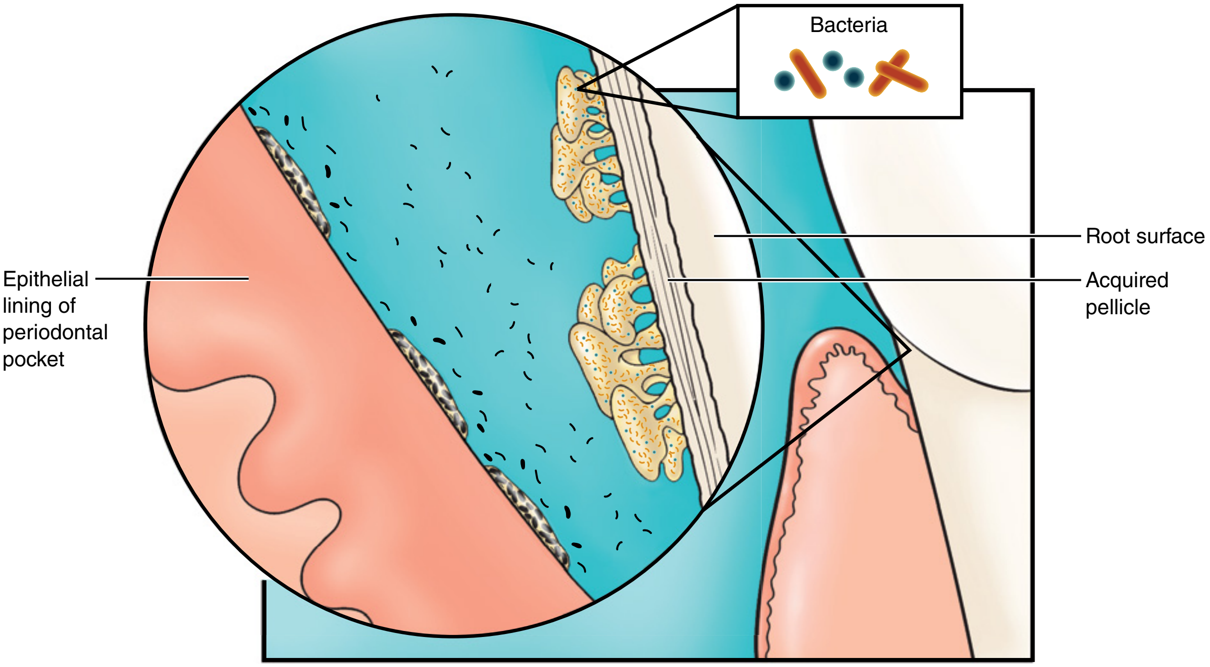

Figure 11-13 Subgingival Plaque Biofilm Attachment Zones. Within a periodontal pocket, bacteria attach to the tooth surface or the epithelial lining of the periodontal pocket.

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1. **Tooth-associated plaque biofilms**—bacteria that are attached to the tooth surface.
   a. Bacteria attach to an area of the tooth surface that extends from the gingival margin almost to the junctional epithelium at the base of the pocket.
   b. Subgingival bacteria appear to have the ability to invade the dentinal tubules of the cementum.
   c. Filamentous microorganisms, cocci, and rods, including *Streptococcus mitis*, *Streptococcus sanguis*, and *Actinomyces viscosus*, dominate the tooth-associated plaque biofilms.
2. **Tissue-associated plaque biofilms**—bacteria that adhere to the epithelium.
   a. Bacteria within the tissue-associated plaque biofilms are distinctly different from those of the tooth-associated plaque biofilms.
   b. The layers closest to the soft tissue wall contain large numbers of spirochetes and flagellated bacteria. Gram-negative cocci and rods also are present. Species such as *Streptococcus oralis*, *Streptococcus intermedius*, *Porphyromonas gingivalis*, *Prevotella intermedia*, *Tannerella forsythia*, and *Fusobacterium nucleatum* predominate.
   c. Bacteria in the tissue-attached plaque biofilms can invade the gingival connective tissue and be found within the periodontal connective tissues and on the surface of the alveolar bone.
3. **Unattached Bacteria**. In addition to the attached bacteria, the periodontal pocket also contains many free-floating **unattached bacteria** that are not part of the biofilm.

Section 3

The Role of Bacteria in Periodontal Disease

Changing Evidence for the Role of Bacteria

In 1683, Antonie van Leeuwenhoek (considered to be the father of microbiology), used a homemade microscope to first observe, describe, and illustrate the unique structural features of microorganisms. Since then, more than 700 bacterial species have been identified in the human oral cavity. Yet, despite more than 340 years of scientific investigation, microbiologists have yet to elucidate which bacterial pathogens cause periodontitis and what specifically triggers the onset of periodontitis in certain individuals but not others.[37] Over the years, five different and distinct hypotheses have emerged to explain the role of bacteria in periodontal disease: (1) the nonspecific plaque hypothesis, (2) the specific plaque hypothesis, (3) the ecological plaque hypothesis, (4) the microbial homeostasis–host response hypothesis, and (5) the keystone pathogen–host response hypothesis.

Historical Perspectives on the Role of Bacteria

Two hypotheses from the recent past focused on the role of the numbers of bacteria and the specific bacteria present in oral biofilms. The **nonspecific plaque hypothesis** postulated that the accumulation of bacterial biofilms leads to periodontal disease. A later theory, the **specific plaque hypothesis**, postulated that specific pathogenic bacteria and their products in the biofilm lead to periodontal disease. Although these hypotheses are now considered to be too simplistic, they served as important stepping stones to our current understanding of the role of bacteria in the development of periodontal disease.

1. **Historical Perspective: Nonspecific Plaque Hypothesis**
    a. **Hypothesis.** This hypothesis proposed that the overaccumulation of plaque biofilm—an overabundance of bacteria in the biofilm—adjacent to the gingival margin led to gingival inflammation and the subsequent tissue destruction.[37–39]
    b. **Limitations of the Nonspecific Plaque Hypothesis**
        1. The consensus among researchers is that this hypothesis is too simplistic and superficial.[19]
        2. A serious criticism of this hypothesis is that it fails to explain why most cases of gingivitis never progress to periodontitis. Some individuals with heavy amounts of plaque biofilm fail to develop periodontitis. Yet, puzzlingly, other individuals with very light amounts of biofilm experience periodontitis.[40]
        3. This hypothesis cannot clarify why some sites within the same individual exhibit considerable periodontal destruction while other sites are unaffected.
2. **Historical Perspective: Specific Plaque/Microbial Shift Hypothesis**
    a. **Hypothesis.** This long-standing paradigm suggests that as periodontitis develops, the oral microbiota shifts from one consisting primarily of beneficial microbes to one consisting of pathogens.[38,39,41] In the specific plaque hypothesis, the microbial composition of the oral biofilm—rather than the amount—is the deciding factor in the development of periodontal disease.
        1. An increase in the number of specific pathogens was thought to be associated with periodontitis.
        2. This model postulates that a microbial shift occurs in which the bacteria in the biofilm change from a predominantly Gram-positive aerobic community to one consisting mainly of groups of Gram-negative anaerobes.[41]
    b. **Socransky's Microbial Complexes.** Research efforts, over many years, focused on the hypothesis that specific microorganisms are the cause of various periodontal diseases and conditions.
        1. These studies identified specific groups of bacteria—*Tannerella forsythia*, *Porphyromonas gingivalis*, and *Treponema denticola*—that were significantly associated with periodontitis. It was noted that these bacteria were interdependent and often could not exist without the presence of the others.[42,43]
        2. Using advanced checkerboard DNA–DNA hybridization techniques, Socransky grouped microorganisms into "complexes" and assigned each complex a color.[43] **Figure 11-14** depicts Socransky's color designations for different microbial groups.
            a. Microorganisms assigned to the orange and red complexes are the species that were thought to be the major etiologic agents of periodontal disease.
            b. Microbes designated as the yellow, green, blue, and purple complexes were thought to be compatible with gingival health.
        3. The theory that microbes could be divided into nonpathogenic and pathogenic groups had a great deal of appeal because of its simplicity in understanding disease pathogenesis. The theory was widely accepted until newer molecular-based approaches to microbe detection brought its validity into question.
    c. **Limitations to the Specific Plaque Hypothesis.** The appeal of the specific plaque hypothesis resides in its fundamental concept that a single group of select pathogens (red-complex microorganisms) are the major cause in the tissue destruction seen in periodontitis. However, the specific plaque hypothesis has several weaknesses:

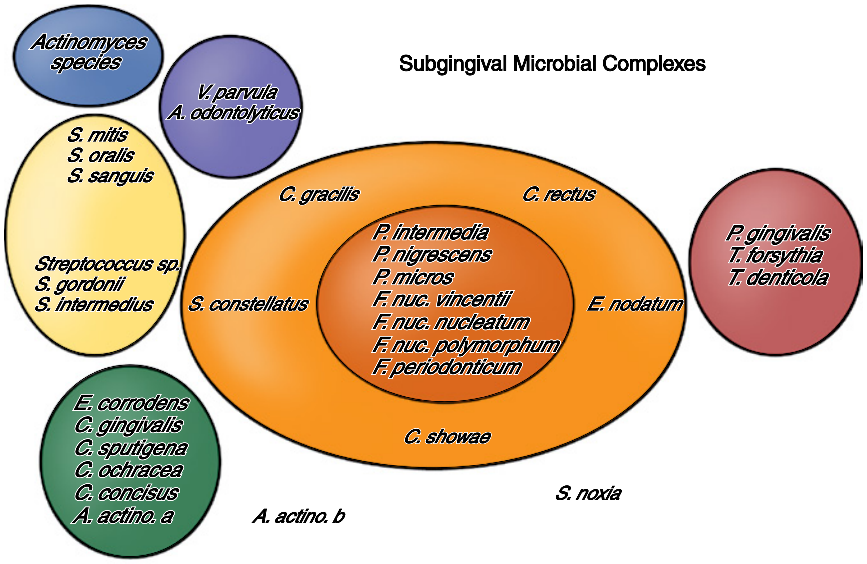
Subgingival Microbial Complexes
Actinomyces species
V. parvula
A. odontolyticus
S. mitis
S. oralis
S. sanguis
Streptococcus sp.
S. gordonii
S. intermedius
C. gracilis
C. rectus
S. constellatus
E. nodatum
C. showae
P. intermedia
P. nigrescens
P. micros
F. nuc. vincentii
F. nuc. nucleatum
F. nuc. polymorphum
F. periodonticum
P. gingivalis
T. forsythia
T. denticola
E. corrodens
C. gingivalis
C. sputigena
C. ochracea
C. concisus
A. actino. a
A. actino. b
S. noxia

Figure 11-14 **Socransky's Microbial Complexes.** Socransky organized bacteria into complexes and assigned each complex a color.

Reproduced from Socransky SS, Haffajee AD, Cugini MA, Smith C, Kent RL, Jr. Microbial complexes in subgingival plaque. *J Clin Periodontol*. 1998;25(2):134–144. doi:10.1111/j.1600-051X.1998.tb02419.x

1. Newer microbial assaying systems have detected red-complex microorganisms in healthy, nondiseased sites.[44,45] Hence, the presence of *Porphyromonas gingivalis* and *Tannerella forsythia* in healthy, nondiseased periodontal sites brings into question whether these bacteria are the direct cause of periodontal breakdown.[46–48]
2. The periodontal microbe population is more heterogeneous and diverse than previously thought.[49–51] More than 700 organisms are recognized as possible oral inhabitants, of which around 200 can be present in any one individual.[1] Many of these newly recognized organisms show good or better correlation with periodontal disease than the classical red-complex microorganisms.[46,52,53] The identification of so many more bacterial species in the oral cavity makes the concept of specific bacteria causing periodontitis even more unlikely.[12]
3. Contrary to the doctrine that Gram-negative bacteria dominate disease sites in periodontitis, Gram-positive anaerobic bacteria species can be found in deep periodontal pockets and have been detected in greater numbers than Gram-negative species in some studies.[53] As such, emerging evidence suggest that periodontitis may be more of a mixed infection, rather than an infection solely caused by specific Gram-negative bacteria.
4. It is evident that the concept of a three-species red complex as a primary causative factor in periodontitis requires refinement.[54]

Contemporary Perspectives on the Role of Bacteria

Rapid advances in the fields of microbiology and immunology have reshaped our previous notions about the role of bacteria in the pathogenesis of periodontal disease. We now recognize that (1) a pathogenic microbial biofilm is a prerequisite for periodontitis to develop, but the presence of a pathogenic oral biofilm alone is insufficient to cause the disease, and (2) although red-complex microorganisms are strongly associated with an inflammatory disease, the current evidence does not support the argument that the red-complex bacteria are potent initiators of the disease. As such, three relatively recent hypotheses have been proposed to reconcile the limitations of the nonspecific plaque hypothesis and the specific plaque hypothesis: (1) the **ecological plaque hypothesis**, (2) the **microbial homeostasis-host response hypothesis**, and (3) the **keystone pathogen-host response hypothesis**.

1. **Current Perspective: Ecological Plaque Hypothesis**
   a. **Hypothesis.** This theory combines key points from the specific plaque hypothesis and the nonspecific plaque hypothesis.[41]

1. The ecological plaque hypothesis postulates that the accumulation of nonspecific bacteria triggers the host inflammatory response. In turn, the host inflammatory response alters the local environment within the gingival sulcus (higher gingival crevicular fluid [GCF] flow, increased bleeding, raised pH, decreased oxygen concentration). **Figure 11-15** depicts the events postulated by the ecological plaque hypothesis.
2. As the local environment changes, the environment itself becomes more conducive to the growth of specific pathogenic bacteria, which leads to further changes in the environment, greater tissue destruction, and a growing predominance of specific periodontal pathogens.
3. Homeostasis is dependent on a balanced local environment. If there is a disruption in ecological homeostasis, it will change the environment that is favorable for specific microorganisms (Figure 11-15). Thus, according to this hypothesis, it is a shift in the local environment that drives the changes in microbial composition that results in pathologic disease, and not vice versa.[55]

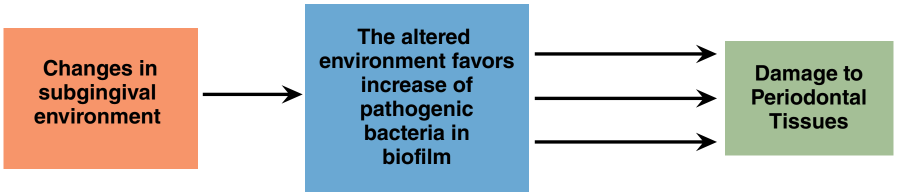
Changes in subgingival environment
The altered environment favors increase of pathogenic bacteria in biofilm
Damage to Periodontal Tissues

Figure 11-15 **Ecological Plaque Hypothesis.** This hypothesis postulates that a shift in the local environment drives the changes in microbial composition that lead to periodontal disease.

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b. **Support for This Hypothesis**
   1. Sites with bleeding upon probing and deeper probing depths are strongly associated with a higher GCF flow. In turn, the higher GCF flow alters the microbial ecology by favoring the growth of pathogenic microorganisms. This suggests that environmental factors—such as GCF flow, pH, temperature, and oxygen tension—are forces that drive dysbiosis in the oral cavity.[15]
   2. Subgingival periodontal instrumentation alters the subgingival ecosystem. This, in turn, reduces the number of pathogens. By reducing the inflammatory status of the gingiva, it is suggested that the flow of GCF decreases, thereby blocking a rich source of nutrients that is necessary for the growth of pathogenic bacteria.[56,57]

2. **Current Perspective: Microbial Homeostasis–Host Response Hypothesis**
   a. **Hypothesis**
      1. This hypothesis proposes that although plaque biofilms are the cause of the initial inflammatory response leading to gingivitis, the pathogenic bacteria are not the direct cause of the destruction of tissues seen in periodontitis.[58] **Figure 11-16** depicts the events postulated by the microbial homeostasis–host response hypothesis.

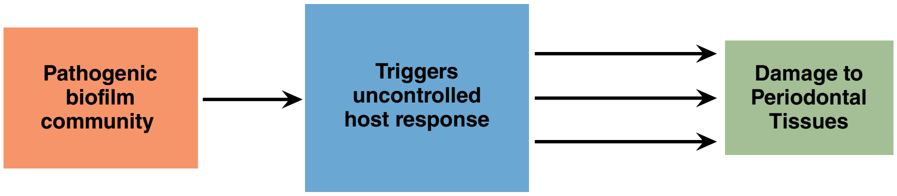
Pathogenic biofilm community
Triggers uncontrolled host response
Damage to Periodontal Tissues

Figure 11-16 **Microbial Homeostasis–Host Response Hypothesis.** This hypothesis postulates that a shift from beneficial to pathogenic bacteria triggers an uncontrolled host inflammatory response. It is this uncontrolled host response that is responsible for the tissue destruction seen in periodontitis.

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        2. The findings that bacteria are not the direct cause of tissue destruction led researchers to focus on the individual host and the complexities of the immune response rather than the bacteria.
        3. Host-related factors, such as genetic variations and the inflammatory immune response, and environmental factors, including smoking, stress, and systemic health, are all now recognized as major factors that contribute to the initiation and progression of periodontal disease.[59]
            a. Evidence for the role of host response in periodontal destruction first emerged in the landmark paper by Page and Schroeder.[60] Host immune response is discussed in detail in Chapters 12 and 13.
            b. Page and Schroeder noted that established gingivitis would not progress to periodontitis unless some other unknown factor tipped the delicate biofilm–host balance toward further tissue destruction.
            c. Thus, it is proposed that the shift from beneficial microbes to a pathogenic community triggers a potent host inflammatory response that contributes to the tissue destruction and alveolar bone loss characteristic of periodontitis.[61]
    b. **Support for This Hypothesis**
        1. The biofilm microbe population associated with periodontal health appears to remain stable over time and exists in a state of biologic equilibrium or homeostasis.
        2. While researchers have identified 6 to 17 potential periodontal pathogens,[62] decades of research have failed to provide evidence that bacterial pathogens are directly and solely responsible for the tissue destruction seen in periodontitis.
        3. Overwhelming evidence demonstrates that it is the uncontrolled host inflammatory and immune response—rather than pathogenic bacteria—that cause the tissue destruction seen in periodontitis.[63] (The shift to a pathogenic biofilm community triggers the host response, but it is the host response which destroys the tissue.) This makes it crucial to identify the subpopulation of susceptible individuals who display this type of uncontrolled immunoinflammatory response and are therefore at a higher risk of periodontitis.

3. **Current Perspective: Keystone Pathogen–Host Response Hypothesis**
    a. **Hypothesis.** This hypothesis builds upon the microbial homeostasis–host response hypothesis. As stated above, the microbial homeostasis–host response hypothesis theorizes that a shift from beneficial to pathogenic microbes triggers the host response responsible for the tissue destruction seen in periodontitis. The keystone pathogen–host response hypothesis expands upon this by postulating that a specific bacterial species is key in creating the shift from symbiotic microbes to dysbiotic microbes in the biofilm community. In turn, the dysbiotic biofilm community triggers the uncontrolled host response that results in damage to the periodontal tissues.[54] **Figure 11-17** depicts the events postulated by the keystone pathogen–host response hypothesis.

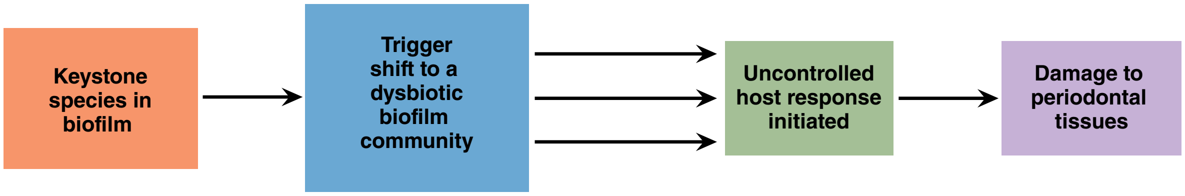
Keystone species in biofilm
Trigger shift to a dysbiotic biofilm community
Uncontrolled host response initiated
Damage to periodontal tissues

Figure 11-17 Keystone Pathogen–Host Response Hypothesis. The presence of certain microbial pathogens—even in low numbers—can cause a shift from beneficial to pathogenic microbes in the biofilm community. The key concept of this hypothesis is that, even at low levels, keystone pathogens can have a significant impact on the oral biofilm that initiates an uncontrolled host immune response.

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        1. According to Hajishengallis et al.,[54] *Porphyromonas gingivalis* is the "keystone species" in initiating this shift within the biofilm community. *Keystone species* is an ecological term used to describe a species that has a disproportionately large effect on its community relative to its abundance. In other words, although present in small numbers, a keystone species can exert a profound effect on the composition and pathogenicity of plaque biofilm.

2. Thus, this hypothesis postulates that a keystone species—*Porphyromonas gingivalis*—initiates the shift from beneficial to pathogenic microbes in the biofilm community that, in turn, triggers the inflammatory response in a periodontitis-susceptible individual. In other words, the transition to periodontitis requires both a polymicrobial dysbiotic biofilm community *and* a susceptible host.

b. **Support for This Hypothesis**
   1. Decades of research have failed to provide evidence that specific bacterial pathogens are the direct cause of the tissue destruction seen in periodontitis.
   2. Current evidence indicates that the uncontrolled host inflammatory and immune responses cause the tissue destruction seen in periodontitis.[63]

4. **Summation of Periodontal Disease Hypotheses.** Currently, each hypothesis falls short in fully unraveling the mechanisms that govern the change from periodontal health to disease. Nevertheless, current periodontal microbiology research is continuing to further investigate the diverse, polymicrobial ecosystem of the oral biofilm and the factors that drive the host inflammatory response. **Table 11-2** summarizes the evolution of periodontal disease hypotheses.

Table 11-2 Evolution of Periodontal Disease Hypotheses

Hypothesis	Status	Theory in Brief
Nonspecific plaque	Historical	An abundance of biofilm bacteria causes the tissue destruction seen in periodontitis.
Specific plaque	Historical	The presence of specific bacteria in the biofilm directly causes the tissue destruction seen in periodontitis.
Ecological plaque	Current	Changes in the subgingival environment can dictate or select the specific microbial composition of the biofilm; the pathogenic biofilm community causes the tissue destruction seen in periodontitis.
Microbial homeostasis–host response	Current	A shift from beneficial to pathogenic microbes triggers the host inflammatory response; the host response causes the tissue destruction seen in periodontitis.
Keystone pathogen–host response	Current	A keystone species initiates a shift from beneficial to dysbiotic microbes in the biofilm community that, in turn, trigger the host inflammatory response responsible for the tissue destruction seen in periodontitis.

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5. **Immune Status of Periodontal Tissue Between Healthy and Diseased Patients**
   a. Despite the difficulty in defining the precise etiology of periodontitis, one thing that is certain is the striking difference in the immune status of periodontal tissue between healthy and diseased patients. Details of the many immunological differences were recently discussed in a review by Darveau.[61]
      1. Clinically healthy periodontal tissue maintains a highly ordered, mild state of subclinical inflammation.[61,64,65]
      2. Clinically diseased periodontal tissue exhibits a marked histopathologic change characterized by a disordered state of severe inflammation.[61,66,67]
   b. It is proposed that the shift from beneficial microbes to a pathogenic community triggers a potent host inflammatory response in the susceptible host that contributes to the tissue destruction and alveolar bone loss characteristic of periodontitis.[61]
      1. Regardless of which disease hypothesis one subscribes to, one must fully appreciate the concept that plaque biofilm, itself, is necessary, but not sufficient to cause the tissue destruction seen in periodontitis.
      2. Instead, it is the uncontrolled host inflammatory and immune responses that cause periodontal tissue destruction.[63]

Chapter Summary Statement

A biofilm is a polymicrobial, three-dimensional community composed of numerous microbial species embedded in a matrix that consists of microbial metabolic products and/or host components, such as salivary glycoproteins. The protective biofilm matrix makes microbes extremely resistant to systemic antibiotics, antimicrobial agents, and the body's immune system, thereby enhancing the odds of survival in the hostile oral environment. Thus, the only way to control the growth and formation of the bacteria populating the biofilm is via mechanical (physical) removal of the biofilm itself.

To date, there is no definitive evidence that specific bacteria are directly responsible for the progression of periodontal disease. Plaque biofilm, itself, is necessary, but not sufficient to cause the tissue destruction seen in periodontitis. Considerable evidence indicates that it is more likely to be the host inflammatory and immune response to the plaque biofilm that leads to the tissue destruction seen in periodontitis. It is proposed that a shift from a symbiotic to a dysbiotic biofilm community triggers a potent host inflammatory response that leads to the tissue destruction and alveolar bone loss characteristic of periodontitis.

In addition, it should be noted that there is emerging evidence indicating that genetics, stress, smoking, diet, and general health play a major role in modulating the host response. These additional factors become more important to take into consideration as newer treatment regimens emerge that are more geared toward controlling and redirecting the host-mediated inflammatory response, rather than the mechanical disruption of the plaque biofilm alone.

Section 4

Focus on Patients

Clinical Patient Care

CASE 1

You have just completed periodontal instrumentation of a tooth surface. Describe the sequence of plaque biofilm formation that will occur on the tooth over the next few days if the patient does absolutely no further self-care of the tooth surface.

CASE 2

Imagine that you are holding an "interview" with the bacteria living in an oral biofilm. How might the bacteria respond to your question about advantages of living in a biofilm?

Evidence in Action

Mr. Smirnov is a new patient. His employer just began to offer dental insurance, so Mr. Smirnov has decided to take advantage of his dental insurance after not seeking dental care for 10 years.

A thorough periodontal assessment shows that Mr. Smirnov has generalized Stage II, Grade B periodontitis. The dental team explains the findings and treatment options to him. Mr. Smirnov asks, "Why can't I just take something to kill off all the bacteria in my mouth once and for all rather than me going to all the bother to remove them every single day from my teeth?" How would you respond to Mr. Smirnov?

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CHAPTER 12

Basic Concepts of Immunity and Inflammation

- Section 1 The Body's Defense System
  - Introduction to the Immune System
  - Components of the Immune System
  - Cells of the Immune System
  - The Complement System
- Section 2 Leukocyte Migration, Chemotaxis, and Phagocytosis
- Section 3 The Inflammatory Process
  - Major Events in the Inflammatory Response
  - Two Stages of Inflammation
- Section 4 Focus on Patients
  - Evidence in Action

CLINICAL APPLICATION

Understanding how the components of the immune system function and how each of these components fit together provides us with an insight of the intricacies of the immune system. This chapter presents a general overview of immunity and inflammation and lays the foundation to understanding the implications of these mechanisms to periodontal disease.

LEARNING OBJECTIVES

- Describe the function of the immune system.
- Compare and contrast innate immunity and adaptive immunity.
- Describe the role of polymorphonuclear leukocytes, macrophages, B-lymphocytes, plasma cells, T-lymphocytes, and NK (natural killer)-lymphocytes in the immune system.
- Compare and contrast a macrophage and a monocyte.
- List the five classes of antibodies (immunoglobulins) and describe the functions of each antibody class.
- Describe the three ways that antibodies participate in the host defense.
- Describe the complement system and explain the role it plays in the immune response.
- Describe the steps in the process of phagocytosis.
- Give an example of a type of injury or infection that would result in inflammation in an individual's arm. Describe and contrast the symptoms of inflammation that the individual would experience due to acute inflammation versus chronic inflammation.
- Define *inflammatory mediator* and provide several examples of inflammatory mediators of importance in periodontitis.

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KEY TERMS

Immune system
Host
Host response
Leukocytes
Granulocytes
Agranulocytes
Polymorphonuclear leukocytes
Neutrophil
Chemotaxis
Lysosome
Neutropenia
Neutrophilia
Monocytes
Macrophage
Lymphocytes
B-lymphocytes
Antibodies
Immunoglobulin
T-lymphocyte
Natural killer (NK)-lymphocyte
Cytokine
Complement system
Membrane attack complex
Opsonization
Transendothelial migration
Phagocytosis
Phagosome
Phagolysosome
Inflammation
Inflammatory biochemical mediator
Chemokines
Acute inflammation
C-reactive protein (CRP)
Homeostasis
Resolution process
Chronic inflammation

Section 1

The Body's Defense System

Humans are surrounded by millions of microorganisms, many of which can be deadly. Our hands alone harbor 2 to 10 million microorganisms. Fortunately, evolution has provided the human body with a multilayered, highly integrated defense system that is remarkably capable in safeguarding the host.

Introduction to the Immune System

1. **Description**
   a. **A Complex System of Responses**
      1. The highly coordinated, tightly integrated **immune system** involves an overlapping network of different types of leukocytes and proteins that work collaboratively in a well-choregraphed, preprogrammed manner to defend the host from invasion.
      2. Bacteria, viruses, and other disease-causing microorganisms bombard the human body over 100 million times a day. For this reason, an intact, robust immune system is necessary for survival.
      3. Broadly, the immune system is divided into two divisions: the innate immunity division and the adaptive (or acquired) immunity division. **Table 12-1** outlines the key characteristic features that distinguishes each division.[1]

Table 12-1 Innate Versus Adaptive Immunity

Innate Immunity	Adaptive Immunity
Present at birth	Develops throughout life following initial exposure to the antigen (over time, the immune system "learns" to recognize and best counterattack a specific pathogen. Thus, adaptive immunity is *acquired* over time through a process of repeated exposure and "relearning.")
Not antigen specific (exposure results in no immunologic memory)	Antigen specific (repeated exposure to a specific pathogen leads to immunologic memory)
Always present (immediate response to infection); innate immune cells are preprogrammed to respond to a broad category of pathogens.	Lag time between infection and response (develops in response to infection)

Innate Immunity	Adaptive Immunity
Does not improve with repeated exposure to an infectious agent	Memory develops, which may provide lifelong immunity to reinfection to the same infectious agent
Cells of innate immunity: ■ Neutrophils ■ Monocytes ■ Macrophages ■ Eosinophils ■ Basophils ■ Mast cells ■ NKT-lymphocytes Note: Physical barriers such as skin and mucosal membranes are key components of the innate immune system. The closed surface of the skin and mucosal membrane creates a natural barrier that protects the host from invading pathogens. Any breach of these physical barriers will trigger the immune response.	Cells of adaptive immunity ■ T-lymphocytes ■ B-lymphocytes

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a. The innate immune system—which humans are born with—is the first line of defense against invading organisms, whereas the adaptive immune system acts as a second line of defense and affords protection against re-exposure to the same pathogen.

b. The adaptive immune system—which develops throughout life following initial exposure to the antigen (a molecule that triggers the immune response)—requires some time to react to an invading organism, whereas the innate immune system includes defenses that, for the most part, are always present and ready to be mobilized immediately upon infection.

c. Adaptive immunity possesses two features that make it unique: (1) immunologic specificity and (2) immunologic memory. *Immunologic specificity* refers to its ability to target a particular infectious agent. *Immunologic memory* refers to its ability to "remember" that it has encountered an invading organism in the past, and then react more rapidly on subsequent exposure to the same organism. In contrast, the innate immune system demonstrates neither specificity nor memory.

d. Innate immunity and adaptive immunity are not mutually exclusive divisions that function independent of each other. Rather, both divisions work in tandem to mutually support each other and form an effective and formidable barrier to infection. For instance, upon encountering a microbe, an immune cell belonging to the innate immune response (such as a macrophage) will phagocytose the invading microorganism, process it, break it into peptides, and present it on its cell surface where it may interact with lymphocytes, thereby activating the adaptive immune response. At the same time, cells of the adaptive immune system, such as B- and T-lymphocytes, will release chemical signaling mediators, known as **cytokines**, to stimulate further recruitment and activation of cells of the innate immune system.

b. **Self Versus Nonself.** When the immune system encounters cells or molecules, it must determine whether these are self (part of the body) or nonself (foreign). Molecules might be harmless environmental molecules, such as pollen, or constitute part of a microorganism. Microorganisms, in turn, might be innocuous or pathogenic. Hence, the immune system must have the capability to differentiate self versus nonself antigens.

2. **Function**

a. **Primary Purpose**

1. The primary purpose of the immune system is to defend the life of the individual, the **host**, by identifying foreign substances in the body (e.g., bacteria, viruses, fungi, parasites, or foreign particles) and developing a formidable defense against them (**Figure 12-1**).[1,2]

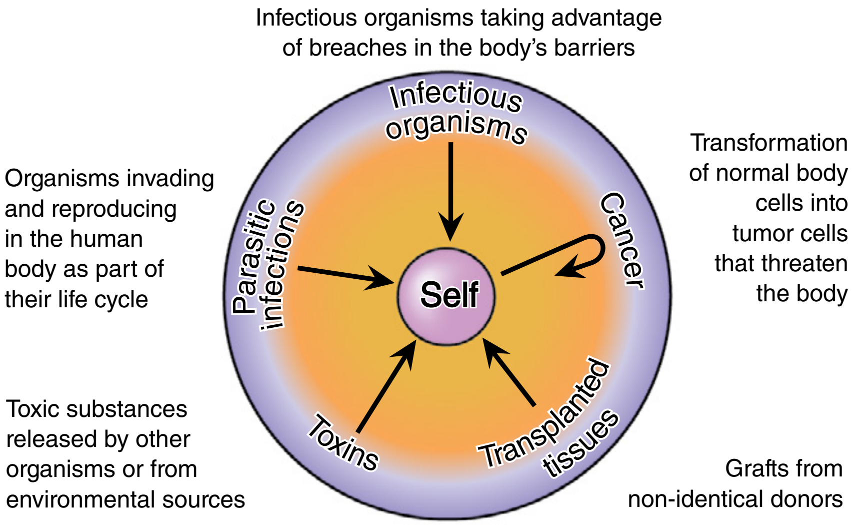
Infectious organisms taking advantage of breaches in the body's barriers
Infectious organisms
Transformation of normal body cells into tumor cells that threaten the body
Cancer
Organisms invading and reproducing in the human body as part of their life cycle
Parasitic infections
Self
Toxic substances released by other organisms or from environmental sources
Toxins
Transplanted tissues
Grafts from non-identical donors

Figure 12-1 The Immune Defense System. The immune system defends the body against invading microorganisms, as well as toxins in the environment. This includes protection against infectious organisms, parasitic infections, viral infections, toxins, cancerous cells, and environmental particles (e.g., pollen) that enter the body. Unfortunately, cells of transplanted tissues are recognized as "nonself," or invaders. For this reason, immunosuppressants are needed to keep the body's immune system from rejecting a transplant.

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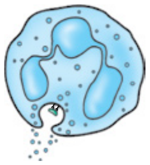
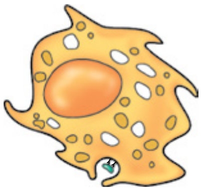
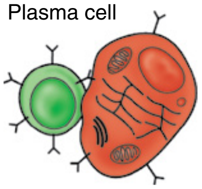
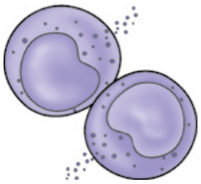
   2. The body recognizes bacteria, viruses, fungi, parasites, and environmental particles (such as pollen) that enter the body as something foreign to itself and responds by (1) deploying immune cells to target the invading microorganisms and (2) producing biochemical substances to amplify the immune response and counteract the foreign invaders.
   3. The way that an individual's body responds to an infection is known as the **host response**.
3. **Consequences of a Dysfunctional Immune Function.** A dysfunctional immune system can be deadly. An example is the human immunodeficiency virus (HIV), the virus that causes acquired immune deficiency syndrome (AIDS). HIV disables a specific group of immune cells (specifically, the T_H-lymphocyte) responsible for coordinating the immune response. Thus, HIV renders the immune system inoperable and makes HIV-positive individuals more susceptible to invading microorganisms that rarely cause infection in individuals with fully competent immune systems.
4. **Consequences of an Overactive Immune Response.** In certain susceptible individuals, the immune system may lead to unintended harmful effects. Rheumatic heart disease is an example of an overactive immune response precipitated by an invasion of the pharynx by *Streptococcus pyogenes*. The immune system recognizes *S. pyogenes* as being nonself molecules and acts against it by releasing cytokines and other inflammatory mediators. Unfortunately, certain surface peptides expressed on human heart tissue are structurally similar to the surface peptides expressed on streptococcal bacteria. As a result, the activated immune system not only attacks streptococci, but also cross-reacts against similar peptides expressed on heart tissue, such as cardiac valves. Prolonged and repeated attack against cardiac tissue will lead to chronic inflammation and irreparable scarring of the heart valves.

Components of the Immune System

Our multilayered, highly functioning immune system is composed of several components that coordinate their activities to mount an effective counterattack against invading microorganisms: (1) cellular defenders (phagocytes, lymphocytes) and (2) the complement system (**Table 12-2**).[1,2]

Table 12-2 **Components of the Immune System**

Component	Function
Polymorphonuclear leukocyte (PMN)  © Wolters Kluwer	■ Phagocytosis ■ Release of lysosomes ■ Release of powerful regulatory proteins (cytokines) that signal the immune system to send additional phagocytic cells to the site of an infection
Macrophage  © Wolters Kluwer	■ Phagocytosis ■ Release of lysosomes ■ Release of powerful regulatory proteins (cytokines) that signal the immune system to send additional phagocytic cells to the site of an infection
B-lymphocyte/ Plasma cell  © Wolters Kluwer	■ Production of immunoglobulns
T-lymphocytes  © Wolters Kluwer	■ Amplify the immune response
Immunoglobulins IgG, IgM, IgA, IgD, IgE © Wolters Kluwer	■ Neutralize bacteria or bacterial toxins ■ Coat bacteria to facilitate phagocytosis ■ Activate the complement system
Complement System © Wolters Kluwer	■ Lysis of cell membranes of certain bacteria ■ Recruitment of additional phagocytic cells to the infection site and clearance of immune complexes from circulation

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Cells of the Immune System

1. **Leukocytes.** **Leukocytes** (also called white blood cells) are colorless, nucleated cells that originate from hemopoietic stem cells in bone marrow, which subsequently differentiate into either bone marrow myeloid progenitor cells (which give rise to mature neutrophils, eosinophils, basophils, monocytes, macrophages, and mast cells) or lymphoid progenitor cells (which give rise to mature lymphocytic cells) (**Figure 12-2**).

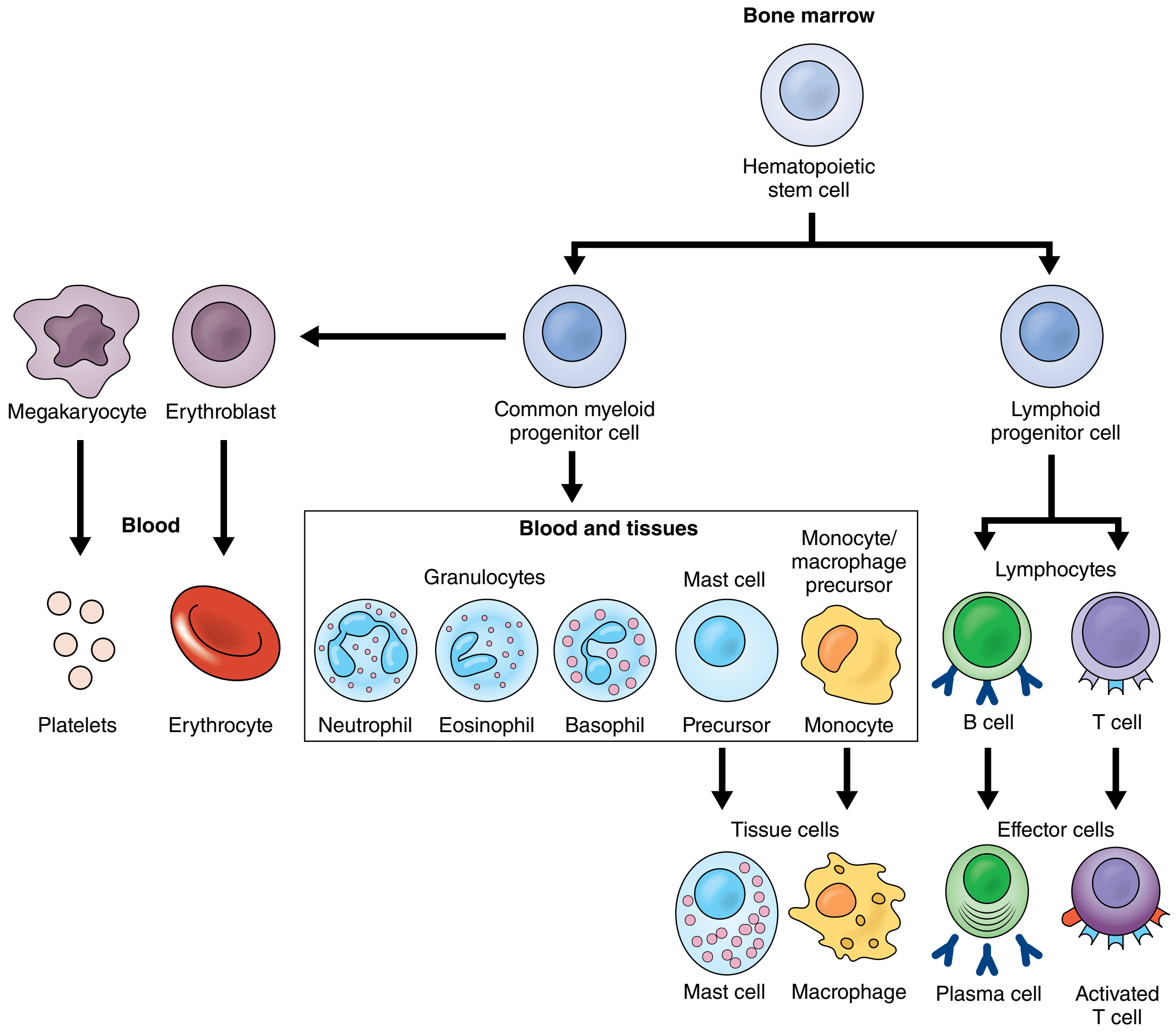
Bone marrow
Hematopoietic stem cell
Megakaryocyte
Erythroblast
Common myeloid progenitor cell
Lymphoid progenitor cell
Blood
Platelets
Erythrocyte
Blood and tissues
Granulocytes
Mast cell
Monocyte/ macrophage precursor
Neutrophil
Eosinophil
Basophil
Precursor
Monocyte
Lymphocytes
B cell
T cell
Tissue cells
Effector cells
Mast cell
Macrophage
Plasma cell
Activated T cell

Figure 12-2 **Cells of the Immune System.** All cells of the human body (immune and nonimmune cells) share a common origin: the pluripotent hemopoietic stem cell. The pluripotent hemopoietic stem cell branches off to either give rise to the common myeloid progenitor cell or the lymphoid progenitor cell. The common myeloid progenitor cell undergoes a series of biological processes to eventually differentiate into granulocytes, erythrocytes, and platelets. In contrast, the lymphoid progenitor cell differentiates into the **agranulocytes**.

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2. Leukocytes are generally categorized according to the presence or absence of cytoplasmic granules (membrane-bound intracellular compartments containing secretory antimicrobial peptides and other important immune-related chemical mediators needed for neutrophilic recruitment and chemotaxis).
   a. A leukocyte carrying granules within the cytoplasm is known as a **granulocyte**. Granulocytes are the most abundant immune cell type found in peripheral blood. Broadly, there are four types of granulocytes:
      1. **Neutrophils** are the most common polymorphonuclear leukocyte. Neutrophils play a vital role in the immune systems as they are the first immune cell to be deployed in response to an inflammatory signal. Because of their inherent capability to act as professional phagocytes, neutrophils can be thought of as the initial responders to the microbial invasion.
      2. Eosinophils—primarily play a role in combating parasitic infections. Secondarily collaborate with mast cells and basophils to regulate the allergic response.
      3. Basophils—largest but least common granulocyte. Primarily modulate the allergic response and coordinate physiologic activity against autoimmune diseases.
      4. Mast cells—tissue-resident immune cells that protect the host against pathogens and release key inflammatory mediators that modulate the allergic response. (Although their activities in modulating the

allergic response overlap with the activity of basophils, the key distinction between the two types of immune cells is that mast cells are fixed in tissues whereas basophils circulate in blood.)

b. An **agranulocyte** is a leukocyte that lacks granules. Lymphocytes and monocytes/macrophages are agranulocytes. Compared to granulocytes, agranulocytes have a larger rounded nucleus because of the lack of cytoplasmic granules.

3. **Polymorphonuclear Leukocytes (PML).** The term *polymorphonuclear* refers to a cell that has a nucleus divided by multiple, elongated lobes of varying sizes and shapes all strung together like sausages hung together on a string (**Figure 12-3**).

a. All members of the polymorphonuclear leukocytic family (neutrophils, eosinophils, and basophils) possess a nucleus that has a multilobulated shape and form.

b. Note that although a mast cell is classified as a granulocyte, it is not a polymorphonuclear leukocyte. Rather, mast cells are classified as *mono*nuclear leukocytes (described below) because they have a single-lobed elongated nucleus. Refer to Figure 12-2 to view the shape of the nucleus for each leukocyte.

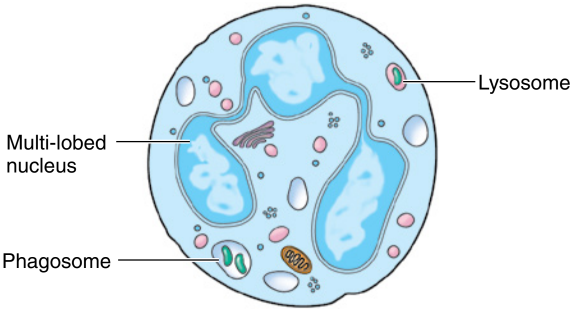

Figure 12-3 Morphology of a Polymorphonuclear Leukocyte. Morphologically, note the multilobulated shape of the nucleus. Also note that all polymorphonuclear leukocytes (neutrophils, eosinophils, and basophils) contain **lysosomes**, which are cytoplasmic compartments containing digestive enzymes that breakdown phagocytosed molecules.

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c. Although they are smallest of all granulocytes, the neutrophils are the most abundant leukocyte found in blood, accounting for 60–70% of all leukocytes. As such, neutrophils are the primary "defenders" of the host defense response. Moreover, neutrophils are one of two types of leukocytes (the other being monocytes/macrophages) that have the capability to phagocytose invading pathogens and foreign particles.

d. Neutrophils are the first line of defense against the initial microbial onslaught and are essential for the control of infections.

e. Although neutrophils typically reside within the bloodstream, they can cross through a capillary wall to reach the site of infection. It is theorized that the lobular arrangement of the nucleus gives the neutrophil greater flexibility to "wiggle" through small gaps in the blood vessels to migrate rapidly to sites of infection (a process known as **chemotaxis**).

f. The cytoplasm of a neutrophil has many granules that contain bactericidal digestive enzymes which break down and destroy the pathogens after phagocytosis.

g. Neutrophils are short-lived cells that die when they become engorged with the bacteria they phagocytize. The pus formed at sites of inflammation contains many dead and dying neutrophils. Neutrophils have a short lifespan, generally less than 1 day.

h. The bacteria associated with periodontal disease are most effectively phagocytized by neutrophils.

i. Normally, each milliliter of blood contains between 3,000 to 6,000 neutrophils. A neutrophil count of less than 1,000 cells/mL is called **neutropenia** and indicates an increased risk of infection. On the opposite side of the spectrum, a neutrophil count above this threshold is known as **neutrophilia** and may be an indication of a chronic inflammation, prolonged infection, or neoplastic condition.

4. **Monocytes/Macrophages**
    a. **Monocytes** are large phagocytes (about two to three times larger than the size of a red blood cell) with a single irregular, kidney-shaped nucleus and an absence of cytoplasmic granules; hence, they are classified as agranulocytes.
    b. A close relative to the monocyte is the **macrophage**, which is the largest leukocyte in terms of size—thus, the name *macrocyte* (**Figure 12-4**). In Greek, the term *macrophage* means "large eater." Macrophages are 5- to 10-fold larger than monocytes and contain more lysosomes. Like monocytes, macrophages are highly phagocytic cells that actively engulf and destroy microorganisms. Additionally, macrophages are agranulocytes.
    c. The shape of the monocyte nucleus differs from that of a macrophage. The monocyte nucleus has an irregular shape, whereas the macrophage has a more regular shape and rounder nucleus, possibly owing to this cell being further differentiated than a monocyte (**Figure 12-5**).
    d. Morphologically, a key distinction between the monocyte/macrophage leukocyte and the polymorphonuclear leukocyte is the number of lobes in the nucleus: the monocyte/macrophage has a single-lobed nucleus, whereas the polymorphonuclear leukocyte is multilobulated. Hence, in contrast to the multilobulated nucleus of a polymorphonuclear leukocyte, a monocyte/macrophage is a *mono*nuclear leukocyte because of the presence of a single lobed–shaped nucleus. Additionally, as mentioned previously, a mast cell is a mononuclear leukocyte because its nucleus has a more rounded, elongated shape and is not segmented into lobes.

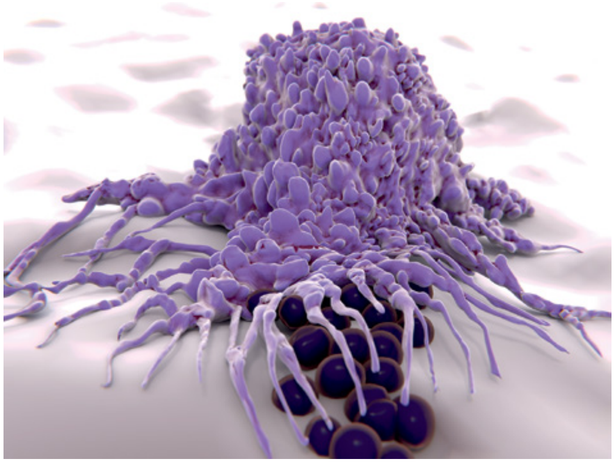

Figure 12-4 Macrophage Engulfing Bacteria (Cocci). Macrophages engulf and digest cellular debris and pathogens.

© Juan Gaertner/Shutterstock

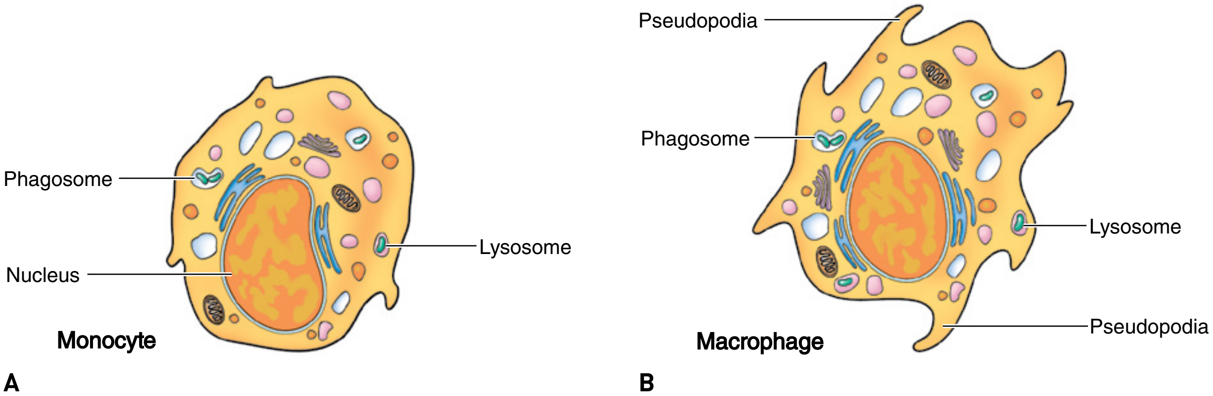

Figure 12-5 Morphology of a Monocyte and a Macrophage. These phagocytic leukocytes are monocytes (**A**) when found in the bloodstream and macrophages (**B**) when they are in the tissues. Structurally, the monocyte has a U- or kidney-shaped single-lobed nucleus, whereas the macrophage appears to have a more rounded single-lobed nucleus.

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e. Macrophages are slower to arrive at the infection site than neutrophils. The slower, long-lived macrophages are often the most numerous cells in chronic inflammation.
f. Macrophages primarily function as antigen-presenting cells. An antigen-presenting cell is any leukocyte that phagocytoses the pathogen (antigen), processes it into many little fragments, and then transports these digested fragments to its surface to be recognized by other immune cells. This, in turn, "informs" and "alerts" other leukocytes (such as T-lymphocytes) so that an effective counterattack can be mounted. The close interaction between cells of the innate immunity division (such as macrophages) and cells of the adaptive immunity division (such as T-lymphocytes) is critical in coordinating an effective host immune response (**Figure 12-6**).

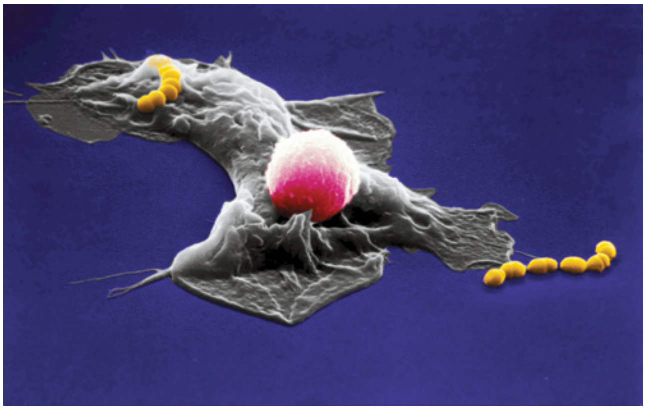

Figure 12-6 **The Antigen-Presenting Interaction: A Bridge Between Innate Immunity and Adaptive Immunity**. Scanning electron micrograph (SEM) of a human macrophage (gray) shows it approaching a chain of *Streptococcus pyogenes* (orange). Riding atop, the macrophage is a spherical lymphocyte. This image highlights the close interplay between cells of the innate immunity division (represented by the antigen-presenting macrophage) and cells of the adaptive immunity division (represented by the lymphocyte) working in tandem to mount an effective counterattack against foreign invaders.

Courtesy of James A. Sullivan, CELLS alive.

5. **Lymphocytes.** **Lymphocytes** are small mononuclear agranulocytes that play an important role in recognizing and neutralizing foreign invaders. As mentioned, because these cells exhibit both specificity and immunologic memory, lymphocytes belong to the adaptive immunity division. The three types of lymphocytes are B-lymphocytes (B-cells), T-lymphocytes (T-cells), and NK (natural killer)-lymphocytes.
   a. **B-Lymphocytes**
      a. **Description**
      b. **B-lymphocytes** are leukocytes that are smaller in size than neutrophils and monocytes/macrophages.
      c. B-lymphocytes originate from hematopoietic stem cells in the bone marrow, and then undergo further maturation and development in the bone marrow. Hence, these lymphocytes are termed *B*-lymphocytes.
      d. B-lymphocytes further differentiate into one of two subclasses of B-cells: plasma B-cells (a fully differentiated B-lymphocyte) and memory B-cells.
         1. Plasma B-cells contribute to the immune response by producing antibodies. Once a plasma B-cell is activated, it manufactures millions of antibodies that are released into the bloodstream (**Figure 12-7**). Thus, plasma B-lymphocytes play a critical role in providing the immune system with humoral immunity. (*Humoral* means "having to do with antibodies.")

2. In contrast, memory B-cells do not produce antibodies. Rather, these lymphocytes (1) "remember" previous exposure to a pathogen and (2) evoke a more rapid and more enhanced response to re-exposure to the same pathogen. Hence, memory B-cells give the immune system the capability to provide long-lasting protective immunity against recurring infectious agents.

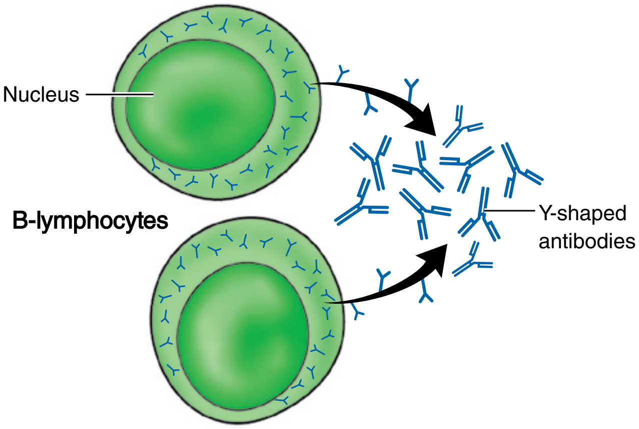

Figure 12-7 Plasma B-Lymphocytes. A plasma B-lymphocyte is notable for its capability to produce Y-shaped proteins, known as antibodies.

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1. **Antibodies**

   a. Structurally, **antibodies** are Y-shaped proteins composed of two fragments: the top-fragment is the antigen-binding region (known as the Fab—Fragment Antigen Binding), whereas the tail fragment (known as the Fc region—Fragment Constant) is the constant region that binds to immune cells and proteins of the complement system (to be reviewed in the next section) (**Figure 12-8**). Note that in most cases each antibody (with the exception of IgM—to be discussed below) has two antigen-binding regions.

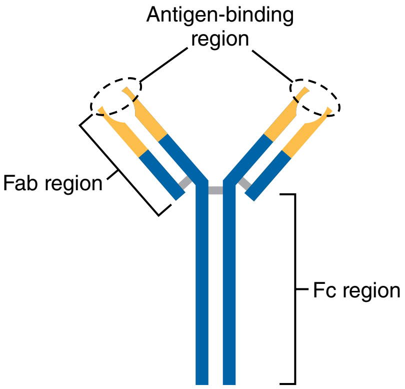

Figure 12-8 General Structure of Antibody. An antibody is a Y-shaped protein. The Fab region is the highly variable antigen-biding region (an antigen is any foreign molecule presence on the surface of a pathogen that can potentially induce harm on the body), whereas the Fc region is the same for all antibody classes and binds to immune cells and/or complement proteins.

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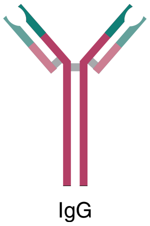
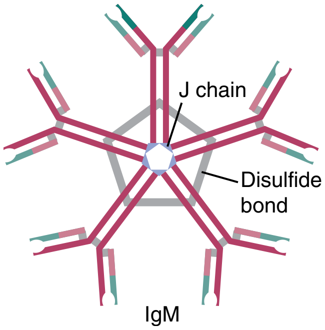
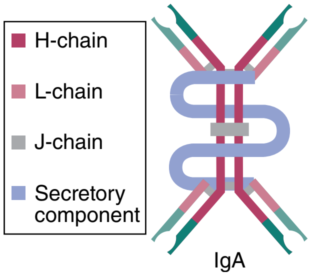
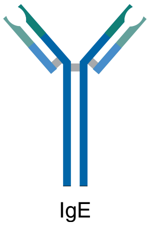
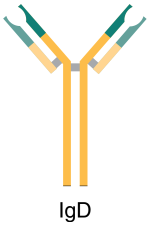
   b. Antibodies are known collectively as **immunoglobulins**. The five major classes of immunoglobulin are immunoglobulin M (IgM), immunoglobulin D (IgD), immunoglobulin G (IgG), immunoglobulin A (IgA), and immunoglobulin E (IgE). The Fc region is the same for all classes of immunoglobins, but the Fab region is highly variable and gives each antibody class its specificity for binding to specific antigens. **Table 12-3** lists the characteristic features of each immunoglobulin.

Table 12-3 Five Immunoglobulin Classes and Key Characteristic Features

Immunoglobin Class	Characteristic Features
IgG  © Jones & Bartlett Learning	■ Most abundant antibody found in the human body (accounts for 70–80% of all circulating antibodies). ■ Responds to invading pathogens such as bacteria and viruses. ■ Potent activator of the complement system. ■ Only immunoglobulin that can pass through the placental barrier, thereby providing protection to the fetus during development.
IgM  © Jones & Bartlett Learning	■ The largest of all antibodies by size. ■ First antibody to respond to initial exposure to antigen found in blood and lymph fluid. ■ Pentamer structure. Unlike other immunoglobulins, IgM has a pentameric structure (think of five immunoglobulins joined to each other via a J-chain). Consequently, IgM has 10 (not 2) antigen-presenting sites, which makes it more efficient in neutralizing the invading pathogens until sufficient quantities of IgG antibody have been synthesized. ■ Potent activator of the complement system.
IgA  © Jones & Bartlett Learning	■ Body's principal line of defense at mucosal barriers. This antibody bathes the mucosal surfaces of the oral cavity, gastrointestinal tract, and respiratory system. ■ The most prevalent form of IgA is secretory dimeric IgA (sIgA). Structurally, IgA looks like two immunoglobulins joined together via the J-chain (small polypeptide chains that attach two immunoglobulins together). Additionally, sIgA has a secretory component that protects the antibody from being degraded by highly active salivary proteolytic enzymes. The salivary component (see figure) physically wraps around the Fc region of each IgA. ■ Poor activator of the complement system. ■ Can be passed to a neonate through maternal breast milk. During the first few months of life, sIgA and IgG are the only immunoglobulins found in circulation until the newborn is able to build up a fully functioning immune system.
IgE  © Jones & Bartlett Learning	■ Powerful mediator of the host allergic response.
IgD  © Jones & Bartlett Learning	■ Least understood antibody, and least abundant antibody found in circulation.

c. Antibodies collaborate with other leukocytes and the complement system to eliminate the offending agent by:
   1. Coating the target to make it more readily phagocytosed. One can think of the antibody as tagging the target cell so that phagocytes can easily recognize and then neutralize the target.
   2. Activating the complement system (predominantly mediated by IgG and IgM).
   3. In certain individuals, it triggers the hypersensitivity immune reaction to a substance that is typically harmless or would not trigger an immune response in all individuals; this is known as the allergic response (predominantly mediated by IgE).

b. **T-Lymphocytes**
   1. Description
      a. A T-lymphocyte is another type of agranulocyte that plays a critical role in the adaptive immune response to pathogens.
   2. There are four important subsets of T-lymphocytes:
      a. T-helper (T_H)-lymphocytes are arguably one of the most important leukocytes of the adaptive immune response. Upon exposure to an antigen, T_H-lymphocytes have a "helper" role in (1) regulating the differentiation and maturation of B-cells into plasma cells so that antibodies are produced, (2) activating cytotoxic T-lymphocytes (T_C-lymphocytes) that kill the targeted agent, and (3) inducing macrophages to phagocytose the target agent. Structurally, the T_H-lymphocytes express a unique surface receptor known as the CD4 receptor. Hence, a T_H cell is commonly referred to as a CD4 cell. The significance of a functioning T_H-lymphocyte can be better appreciated when one considers the impact of HIV. HIV specifically targets the CD4 receptor expressed on the T_H-lymphocyte. This, in turn, (1) inactivates the protective effects of the T_H-lymphocyte and (2) disables the entire immune response.
      b. T-cytotoxic (T_C)-lymphocytes neutralize virally infected cells and tumor cells through direct or indirect means. Directly, T_C-lymphocytes neutralize the target cell by releasing perforin, a protein that binds to the target and creates pores in its membranes. Indirectly, T_C-lymphocytes mediate cytotoxic effects by secreting critical cytokines—specifically, tumor necrosis factor-alpha (TNF-α) and interferon-gamma (IFN-γ), which have antiviral and antitumor capabilities. Structurally, the T_C-lymphocyte is distinguished by a CD8 receptor expressed on the surface of its cell membrane. Hence, the T_C-lymphocyte is commonly referred to as a CD8 cell. The importance of functioning T_C-lymphocytes can be appreciated when one considers that impaired CD8 function is a well-known feature of many chronic autoimmune diseases, including rheumatoid arthritis, systemic lupus erythematous, Sjogren disease, myasthenia gravis, and multiple sclerosis.
      c. T-memory (T_M)-lymphocytes "remember" previous exposure to a specific pathogen. Upon re-exposure of the same pathogen, the T_M-lymphocyte responds rapidly by coordinating with T_H- and T_C-lymphocytes to mount a faster and stronger immune response. T_M-lymphocyte's "memory" serves as the basis for how vaccines provide long-term protection.
      d. NKT (Natural Killer T)-lymphocytes. Accounting for less than 1% of all T-lymphocytes, NKT cells are an unusual subset because they share the characteristics of both a T-lymphocyte and an NK-lymphocyte (described below). On the surface, NKT-lymphocytes originate from the same lymphoid lineage as T-lymphocytes and mature in the thymus. Functionally, however, NKT-lymphocytes are more like NK-lymphocytes: both are naturally endowed to "kill" the target. Upon activation, NKT-lymphocytes owe their "natural killing" instincts to their ability to unleash a massive quantity of inflammatory mediators, such as IFN-γ, IL-4, and other cytokines that modulate the effects of the immune response.
   3. T_H-,T_M-, and T_C-lymphocytes are part of adaptive immunity. However, NKT-lymphocytes are part of innate immunity because these naturally endowed "killer cells" are preprogrammed to be rapidly deployed against antigens following activation by other immune cells.

4. All lymphocytes have the same embryological derivation from the lymphoid lineage. Yet, unlike B-lymphocytes, T-lymphocytes mature and develop in the thymus. This is why these lymphocytes are referred to as *T*-lymphocytes.
5. **Table 12-4** summarizes the four subsets of T-lymphocytes and outlines their primary roles in the immune system.

Table 12-4 **Four Subsets of Lymphocytes**

Immunoglobin Class	Role in Immune System
T_H-lymphocyte (CD4 cell)	■ Plays a "helper" role by regulating the differentiation and maturation of B cells and activation of cytotoxic T cells and macrophages. ■ Does not directly neutralize or clear pathogens.
T_C-lymphocyte (CD8 cell)	■ Upon activation, the T_C-lymphocyte directly attacks viral-infected cells or tumor cells by releasing perforin, which punches holes through the plasma membrane of the target cell, thereby having direct cytotoxic effect. ■ Indirectly, the TC-lymphocyte produces antiviral and antitumor cytokines such as TNF-α and IFN-γ.
T_M-lymphocyte	■ "Memory" T-lymphocytes. Remember past exposure so that upon re-exposure the immune response is faster and stronger than the initial response.
NKT-lymphocyte (NKT)	■ Following activation, NKT-lymphocytes immediately commence with cytokine secretion. ■ The rapidity of their response makes NKT-lymphocytes important players in the very first lines of defense against some types of bacterial and viral infections.

c. **NK (Natural Killer)-lymphocytes** (also known as large granular lymphocytes) are an unusual form of lymphocytes because (1) they are noticeably larger than B- and T-lymphocytes; (2) their cytoplasm contains granules (containing proteases and perforin), whereas B- and T-lymphocytes do not have granules; and (3) they have the attributes of both innate immunity and adaptive immunity. Other distinguishing features of this type of lymphocyte are:

1. Like an NKT-lymphocyte, an NK-lymphocyte is a preprogrammed "natural killer" of virus-infected cells and tumor cells.
2. Although the names are similar, there are several key distinctions that distinguish NKT-lymphocytes from NK-lymphocytes.
   a. NK-lymphocytes are a separate and distinct category of lymphocyte that shares many characteristics with its brethren in the lymphocytic family. In contrast, NKT-lymphocytes are a subset of T-lymphocytes (**Figure 12-9**).
   b. Although NK-lymphocytes and NKT-lymphocytes originate from the same lymphoid progenitor cell lineage, NK-lymphocytes mature and develop in circulation, whereas NKT-lymphocyte precursors need to travel to the thymus to mature (just like the other T-lymphocytes).
   c. Although NKT-lymphocytes and NK-lymphocytes are both "natural killers," NK-lymphocytes do not require preactivation by other immune cells or by antibodies. This means that NK-lymphocytes can directly induce the death of a virus-infected cell or tumor cell in the absence of any specific coordination with another immune cell. Hence, NK-lymphocytes are true "natural killers"—in other words, these lymphocytes are naturally endowed cytolytic cells that can provide immediate defense, thereby giving NK-lymphocytes a feature consistent with innate immunity. At the same time, NK-lymphocytes also exhibit memory-like properties, which is a characteristic feature of adaptive immunity. Thus, a NK-lymphocyte has features that allow it to participate in both innate and adaptive immunity.

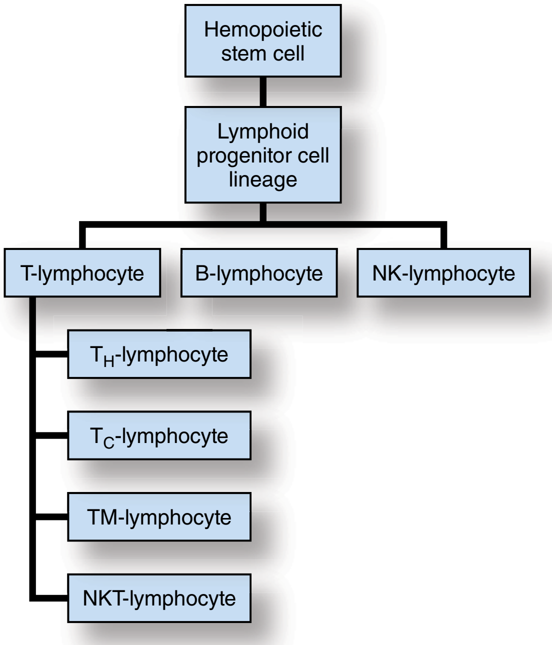


Figure 12-9 Lymphocytic Lineage. All lymphocytes originate from hemopoietic stem cells, which subsequently differentiate into lymphoid progenitor cells. These progenitor cells, in turn, give rise to B-lymphocytes, T-lymphocytes, and NK-lymphocytes. Note that an NK-lymphocyte is a distinct form of lymphocyte that matures in peripheral blood. In contrast, an NKT-lymphocyte is a subset of a T-lymphocyte and matures in the thymus, like all other forms of T-lymphocytes.

The Complement System

If the above cellular leukocytes are analogous to valiant defenders of the host, the complement system is akin to unmanned drones that work in concert with the cellular defenders to safeguard the host. The complement system offers an added layer of defense that is needed against pathogens that have the ability to evade and outwit the cellular defenders.

1. **Definition.** The **complement system** is composed of inactive, nonnucleated (lacking a nucleus) proteins that patrol the body for any potential invader. Upon exposure to a pathogen, the complement system undergoes a complex series of molecular events that lead to either the direct neutralization of the targeted pathogen (by punching holes in the bacterial cell membrane) or indirect neutralization of the target (by attracting neutrophils and monocytes/macrophages to phagocytose the target via a process known as opsonization). As the name suggest, the complement proteins work with ("complement") the antibodies and the phagocytes to neutralize the invading pathogen.
2. **Four Principal Functions of Complement.** After activation, the complement proteins undergo a highly regulated cascade that ultimately activates the system to carry out several defensive functions (**Figure 12-10**):
   a. **Destruction of Pathogens.** Components of complement can destroy certain microorganisms directly by forming pores in their cell membranes. To accomplish this task, the complement system creates a protein unit called the **membrane attack complex** that punctures the cell membranes of certain bacteria (lysis).
   b. **Opsonization of Pathogens.** The complement system facilitates the engulfment and destruction of microorganisms by phagocytes. This process, known as **opsonization** (from the Greek word *opsonein*, which means "to prepare for eating"), is the most important action of the complement system. Complement

proteins opsonize (bind and coat) the surface of the pathogen, thereby "marking" it for eventual engulfment by phagocytes.

c. **Recruitment of Phagocytes.** The complement system recruits additional phagocytic cells to the site of the infection. The complement proteins, themselves, are a potent chemoattractant that draw in more leukocytes to migrate to the infection site.

d. **Immune Clearance.** Finally, the complement system performs a "housekeeping" function by removing immune complexes from circulation.

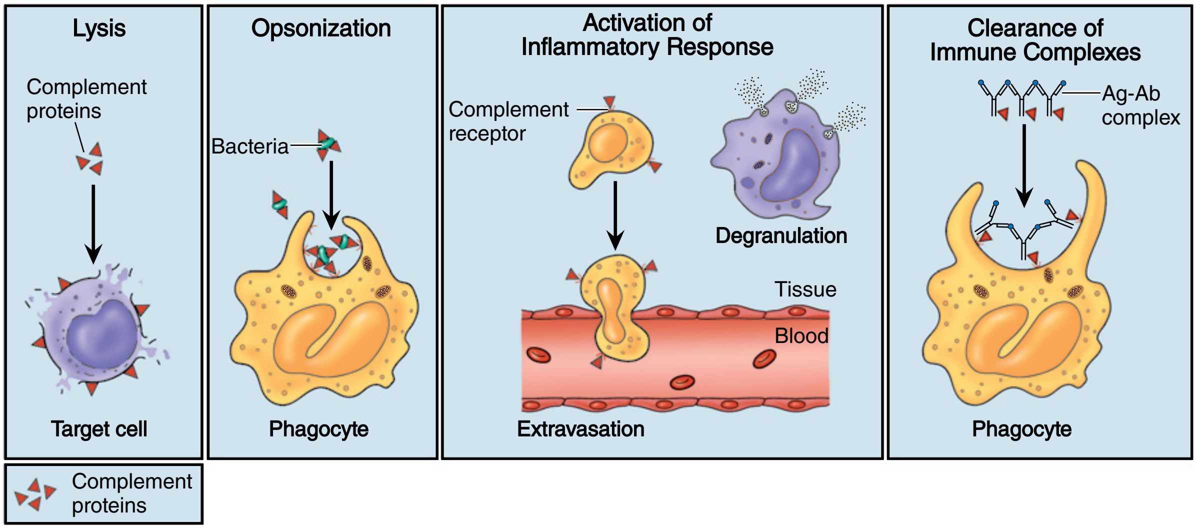

Lysis
Complement proteins
Target cell
Opsonization
Bacteria
Phagocyte
Activation of Inflammatory Response
Complement receptor
Degranulation
Tissue
Blood
Extravasation
Clearance of Immune Complexes
Ag-Ab complex
Phagocyte
Complement proteins

Figure 12-10 **Activities of the Complement System.** In this diagram, complement proteins are represented by small red triangles. Complement proteins facilitate several immune activities: (1) puncturing the cell membranes of certain bacteria (lysis), (2) phagocytosing pathogens (opsonization), (3) amplifying the inflammatory response by recruiting additional phagocytic cells to the infection site, and (4) clearing the immune complexes from circulation.

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Section 2

Leukocyte Migration, Chemotaxis, and Phagocytosis

1. **Leukocyte Migration from the Blood Vessels**
   a. **Transendothelial Migration**
      1. To fight an infection, tissue-resident leukocytes (i.e., macrophages) release cytokines and chemokines (potent signal protein molecules that draw leukocytes to the targeted infection site) into the connective tissue to recruit more leukocytes from the bloodstream to the site of the infection (**Figure 12-11**).
         a. Blood-borne leukocytes "sense" these potent signaling molecules, which activates further leukocytic recruitment to the site of infection and initiates the migration of more immune cells through the single-layered endothelial cells lining the blood vessels (extravasation) to enter the connective tissue.[3]
         b. The process of leukocytes squeezing their way through the thin endothelial wall of a blood vessel to enter the surrounding connective tissue is known as **transendothelial migration**.
      2. Defects in transendothelial migration are associated with severe forms of periodontitis, underscoring the importance of this process in pathogen elimination and tissue repair.
   b. **Chemotaxis**
      1. Once entering the connective tissue, leukocytes are drawn to the target by the released chemokines. This process is known as chemotaxis.

2. Chemotaxis is the process whereby leukocytes migrate toward the infection site in response to bioactive compounds released by leukocytes. A simple analogy is that chemokines act like a trail of breadcrumbs that guide the leukocytes to the intended target site.
3. As more neutrophils are directed to the infected tissue, a swarm-like migration pattern of neutrophils surrounds and neutralizes the invading pathogens; this is known as neutrophilic swarming. In entomology (the study of insects), neutrophilic swarming is analogous to how wasps protect their nest by swarming around an invading intruder.

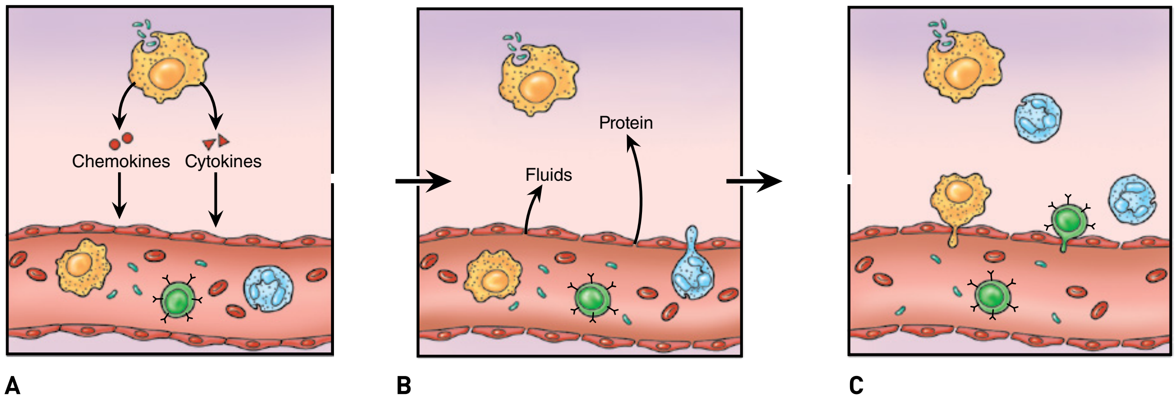

Figure 12-11 Leukocytic Migration to the Targeted (Infection) Site. At the site of infection, tissue-resident host immune cells release chemokines and cytokines (signaling protein molecules that activate the functions of the immune response) to recruit more leukocytes to the site of the infection. These potent signaling molecules stimulate the migration of blood-borne leukocytes into the connective tissue, thereby amplifying the host immune response and recruiting more leukocytes to the targeted site. **A.** Blood-borne leukocytes "sense" the cytokine signals, which then initiates the process of transendothelial migration. **B.** Leukocytes squeeze between the cells of the blood vessel wall to exit the bloodstream. **C.** Leukocytes enter the connective tissue and are drawn to the invading pathogens via the released cytokines and chemokines. (Key: yellow leukocyte = macrophage/macrophage; green leukocyte = plasma B-lymphocyte; blue leukocyte = neutrophil)

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2. **Phagocytosis**
   a. **Description. Phagocytosis** is the process by which neutrophils and monocytes/macrophages engulf and digest pathogens or nonself molecules.[4]
      1. **Steps in Phagocytosis**
         a. First, the phagocytic cell (such as a neutrophil or monocyte/macrophage) binds to the bacterium (**Figure 12-12**). The phagocytic cell extends amoeba-like projections (pseudopodia) that surround the bacterium.
         b. Next, within the cytoplasm of the phagocytic cell, a vesicle called a **phagosome** surrounds the ingested bacterium.
         c. Lysosome granules fuse with the vesicle to form a **phagolysosome**.
         d. The bacterium is digested within the phagolysosome.
         e. Finally, the phagocytic cell discharges the contents of the phagolysosome into the surrounding tissue.

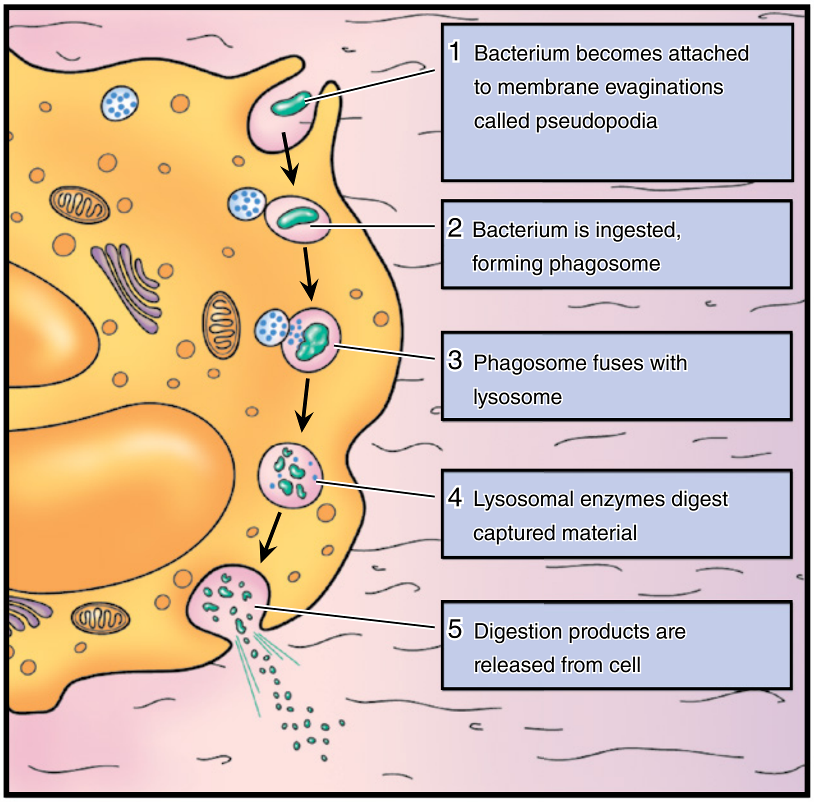
1 Bacterium becomes attached to membrane evaginations called pseudopodia
2 Bacterium is ingested, forming phagosome
3 Phagosome fuses with lysosome
4 Lysosomal enzymes digest captured material
5 Digestion products are released from cell

Figure 12-12 **Phagocytosis.** The steps involved in phagocytosis, the process by which leukocytes engulf and digest microorganisms.

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2. **Local Tissue Destruction from Phagocytosis**
   a. Lysosomal enzymes and other microbial products are released from a leukocyte after phagocytosis or when the leukocyte dies.
   b. Once released, the lysosomal enzymes cause damage to tissue cells in the same manner that they destroy bacteria.

Section 3

The Inflammatory Process

Inflammation is the body's protective response to pathogens, foreign bodies, or an injury. The inflammatory response concentrates host defense components at the site of an infection or injury to eliminate microorganisms and heal damaged tissue. Inflammation is characterized by (1) dilation of the blood vessels, (2) increased permeability of the blood capillaries, (3) increased blood flow, and (4) leukocyte migration into tissues.[1,2,5]

Major Events in the Inflammatory Response

1. The inflammatory response is triggered by the invasion of pathogens or tissue injury.
2. Immediately, mast cells (granulocytes located in the connective tissues near blood vessels) release cytokines (chemical signals) that dilate the capillaries and increase vascular permeability (**Figure 12-13**).
3. Minutes after tissue injury, there is an increase in blood flow to the area. Higher blood volume heats the tissue and causes it to redden. This increased blood flow is needed to deliver leukocytes to the targeted infection site.

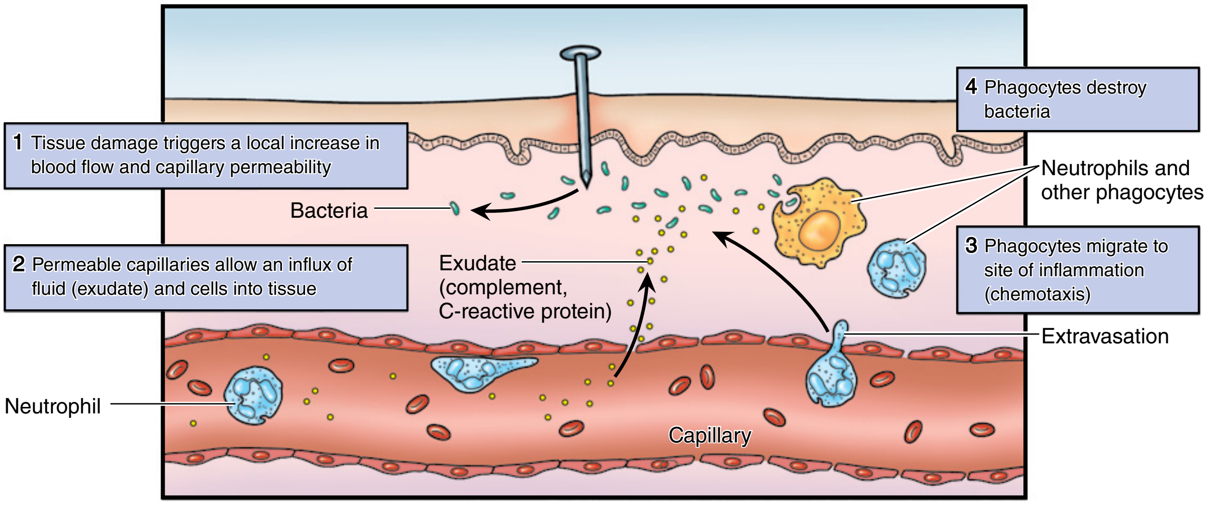
1 Tissue damage triggers a local increase in blood flow and capillary permeability
2 Permeable capillaries allow an influx of fluid (exudate) and cells into tissue
Bacteria
Exudate (complement, C-reactive protein)
Neutrophil
Capillary
4 Phagocytes destroy bacteria
Neutrophils and other phagocytes
3 Phagocytes migrate to site of inflammation (chemotaxis)
Extravasation

Figure 12-13 Inflammatory Response. In this illustration, bacteria have entered the body through a wound created by a nail puncture. The entry of bacteria initiates an inflammatory response that begins with the release of cytokines that attract phagocytic cells to the site of the bacterial invasion.

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4. Within hours, leukocytes pass through the walls of capillaries into the connective tissue (transendothelial migration). Plasma proteins leak from the capillaries and accumulate in the tissues.
5. The leukocytes phagocytose invading pathogens and release inflammatory biochemical mediators that further amplify the inflammatory response.
    a. **Inflammatory biochemical mediators** are biologically active proteins secreted by cells that activate the body's inflammatory response.
    b. Inflammatory biochemical mediators of importance in periodontitis are cytokines, prostaglandins, and matrix metalloproteinases.
        1. Leukocytes release cytokines that play a major role in regulating the behavior of immune cells.
        2. **Chemokines**, a major subgroup of cytokines, are signaling molecules that attract immune cells to the site of infection or injury.[4] Hence, **chemokines** are specially adapted cytokines that regulate chemotaxis (guiding of cell movement toward the target location).

Two Stages of Inflammation

1. **Acute Inflammation**
    a. **Description**
        1. **Acute inflammation** is a short-term, normal process that protects and heals the body following physical injury or infection (**Figure 12-14**).
        2. In the absence of inflammation, wounds and infections would never heal, and the progressive tissue destruction would threaten the life of the individual.
        3. The acute inflammatory process is achieved by the increased movement of plasma and leukocytes from the blood into the injured tissues.
    b. **Five Classic Symptoms of Acute Inflammation.** To *inflame* means "to set on fire," which makes us think of red, heat, and pain. Clinically, there are five classic symptoms of acute inflammation (**Box 12-1**) at the site of infection or injury:
        1. **Heat**—a localized rise in temperature due to an increased amount of blood at the site.
        2. **Redness**—the result of increased blood in the area.
        3. **Swelling**—the result of the accumulation of fluid at the site. The leukocytes and plasma that collect at the site cause the swelling (edema) associated with inflammation.
        4. **Pain**—the result of pressure from edema in the tissue. The excess fluid in the tissues puts pressure on sensitive nerve endings, causing pain.
        5. **Loss of function**—the result of swelling and pain. For example, inflammation of a finger (swelling and pain) would cause you to favor that finger and not use it in a normal manner.

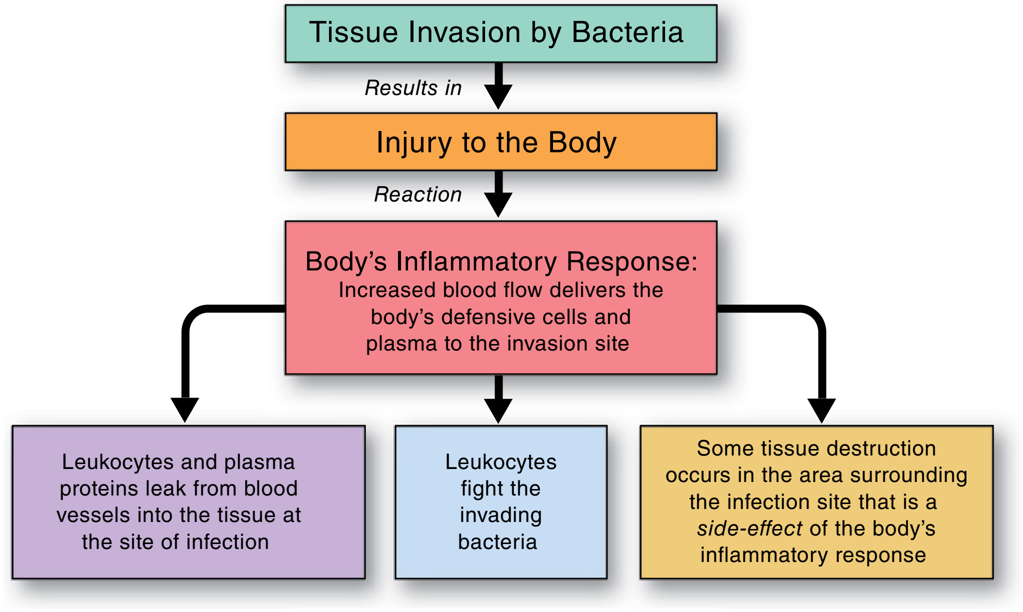
Tissue Invasion by Bacteria
Results in
Injury to the Body
Reaction
Body's Inflammatory Response: Increased blood flow delivers the body's defensive cells and plasma to the invasion site
Leukocytes and plasma proteins leak from blood vessels into the tissue at the site of infection
Leukocytes fight the invading bacteria
Some tissue destruction occurs in the area surrounding the infection site that is a *side-effect* of the body's inflammatory response

Figure 12-14 Major Events in the Body's Inflammatory Response. Inflammation is the body's response to injury or invasion by disease-producing organisms. This response focuses the body's defense mechanisms at the site of an injury or infection.

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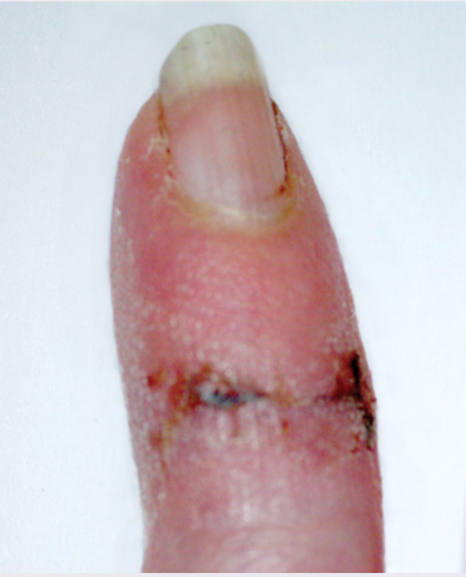
Box 12-1 An Everyday Example of Acute Inflammation



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Callie L. sustained a deep cut to her little finger. She applied an antiseptic cream and covered the wound with an adhesive bandage. A few hours later, the injured finger is quite painful. When Callie applies pressure to the area near the wound, it feels warm and the pressure of her touch is quite painful. The finger looks red and swollen.

What is the source of the redness?
The redness is due to increased blood flow at the injury site.

What is the primary source of the swelling?
The primary source of the swelling is the entry of fluid into the connective tissue. Cells entering the connective tissue also contribute to the swelling.

What is the cause of the warmth?
The warmth of an inflamed area results from increased blood flow to the area that brings with it the warmth.

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c. **The Acute Inflammatory Process**
   1. **Description.** Acute inflammation is initiated by the blood vessels near the injured tissue, which alter to allow the release of plasma proteins and leukocytes into the surrounding tissue.
   2. Neutrophils are the first leukocytes to arrive at the injured site.
      a. These cells phagocytose and kill invading microorganisms through the release of nonspecific toxins. These nonspecific toxins kill pathogens as well as adjacent host cells, sick and healthy alike.

   b. The neutrophils release proinflammatory cytokines, such as interleukin-1 (IL-1) and tumor necrosis factor-alpha (TNF-α).
   c. These proinflammatory cytokines, in turn, induce the liver to synthesize various plasma proteins called acute phase reactant proteins.
      1. The liver produces **C-reactive protein (CRP)**, a type of acute-phase reactant protein, during episodes of acute inflammation. The levels of CRP increase up to 50,000-fold in acute inflammation.
      2. Periodontitis, as well as other systemic diseases—diabetes, hypertension, and cardiovascular disease—are associated with elevated levels of CRP.[6,7]
      3. A study published in the *Journal of Periodontology* reports that the inflammatory effects from periodontal disease cause oral bacterial byproducts to enter the bloodstream and trigger the liver to make CRP that inflames the arteries and promotes blood clot formation.[8]
3. Neutrophils are short-lived and so are primarily involved in the early stages of inflammation.
4. If the body succeeds in eliminating all microorganisms, the tissue will heal and the inflammation will cease. The goal at this stage of the inflammatory process is to establish tissue **homeostasis** (the process of the body's tissue maintaining its optimal state of being). Immune cells leave the area, tissue structures return to normal, and blood flow is reduced with no damage to the tissues. In most cases of infection within the body, the acute inflammatory process eliminates the disease-producing organisms.
5. Resolution of the acute inflammatory process is a coordinated process. The **resolution process** uses cells to send "stop signals" that lead to shut down and clearance of immune cells.[9]
   a. Thus, the body attempts to eliminate bacterial invaders and, once this is accomplished, actively shuts down the inflammatory response to limit self-damage to host tissues.
   b. The process of shutting down the inflammatory response prevents tissue injury and progression of acute inflammation into chronic inflammation.
6. Inflammation is the body's first line of defense against injury and infection, but it is a double-edged sword. If the acute inflammatory response is not effective in eliminating the invading microorganisms or due to a defective/aggravated immune response, the inflammatory response does not shut down and becomes chronic (**Figure 12-15**).

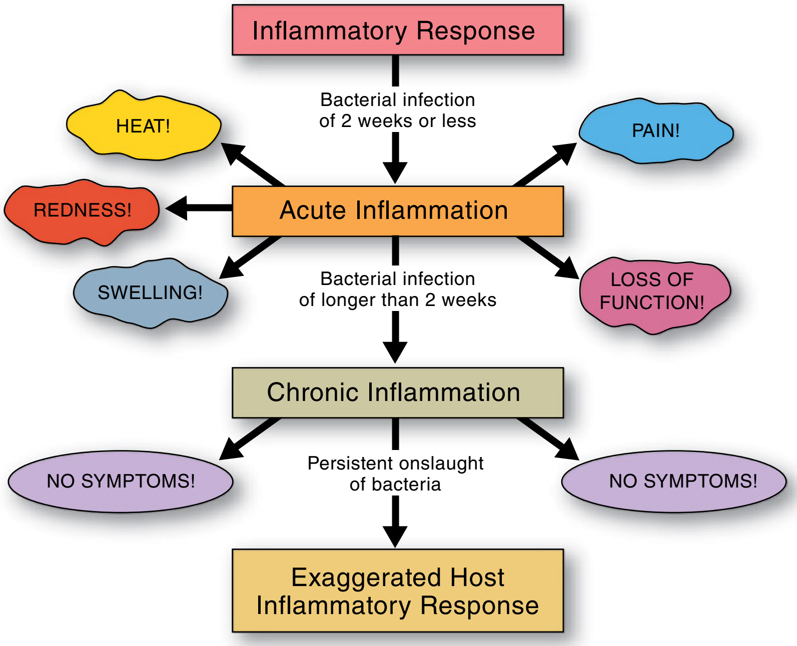
Inflammatory Response
Bacterial infection of 2 weeks or less
HEAT!
PAIN!
REDNESS!
Acute Inflammation
SWELLING!
LOSS OF FUNCTION!
Bacterial infection of longer than 2 weeks
Chronic Inflammation
NO SYMPTOMS!
Persistent onslaught of bacteria
NO SYMPTOMS!
Exaggerated Host Inflammatory Response

Figure 12-15 Two Stages of Inflammation. Acute inflammation is of short duration, whereas chronic inflammation is a long-lived inflammatory response.

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        7. Periodontal diseases are characterized by the dysfunction of the resolution pathways that shut down the acute inflammatory process. The result is a failure of the periodontal tissues to heal and a chronic, progressive, and destructive, nonresolving inflammation.[10,11]

2. **Chronic Inflammation**
    a. **Description**
        1. **Chronic inflammation** is a prolonged, out-of-control inflammatory response that continues for more than a few weeks and results in varying degrees of tissue injury.[12]
            a. It is a pathological condition characterized by active inflammation, host tissue destruction, and attempts at repair.
            b. The warning signs of acute inflammation are absent in chronic inflammation—such as periodontitis—and the problem may go unnoticed by the host (patient). Clinically, pain often is absent.
        2. The inflammatory response has one all-important goal: respond immediately to destroy infectious microorganisms in the damaged tissue before they can spread to other areas of the body.
            a. Chronic inflammation occurs when the body is unable to eliminate the infection. In this stage, the invading microorganisms are persistent and stimulate an exaggerated response by the host's immune system.
            b. In cases where inflammation becomes chronic, the inflammation can become so intense that it inflicts permanent damage to the body tissues. This is the case in periodontitis.
    b. **The Chronic Inflammatory Process**
        1. The accumulation of macrophages characterizes chronic inflammation.
        2. Macrophages engulf and digest microorganisms.
        3. Leukocytes release several different inflammatory mediators, including IL-1, TNF-α, and prostaglandins that perpetuate the inflammatory response (**Figure 12-16**).

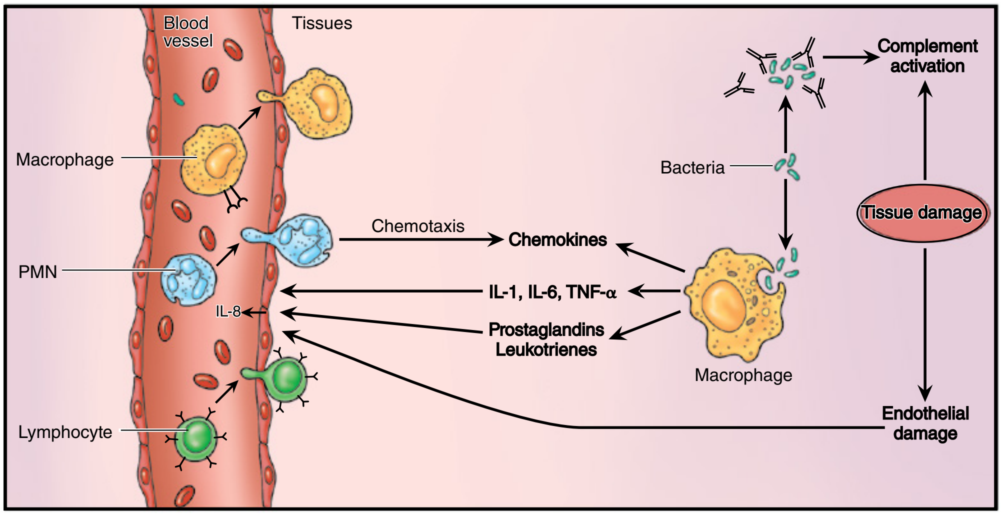

Figure 12-16 Cellular Defenders and Mediators in Inflammatory Response. The three primary cells involved in the inflammatory response are the polymorphonuclear leukocytes (i.e., neutrophils), macrophages, and lymphocytes. The first of these cells to migrate into the tissues are the neutrophils, followed by macrophages, and then the lymphocytes. In addition, this illustration shows some of the many chemical mediators that play a part in the inflammatory response. These include chemokines, IL-1, IL-6, TNF-α, prostaglandins, and leukotrienes.

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            a. One of the principal cytokines secreted by macrophages is TNF-α.[13] Evidence indicates that TNF-α contributes to the tissue destruction that characterizes chronic inflammation (**Table 12-5**).
            b. In fact, tissue damage is the hallmark of chronic inflammation.

4. If the infection persists, inflammation can last months or even years.
5. Chronic inflammation is abnormal and does not benefit the body. Chronic inflammation is an out-of-control response that can destroy healthy tissue and cause more damage than the original problem. As with other forms of host-mediated tissue injury—such as asthma, rheumatoid arthritis, diabetes, and atherosclerosis—periodontitis is characterized by unresolved, chronic inflammation that leads to ongoing tissue damage through continuous and recurring episodes of acute inflammation.[10,14]

Table 12-5 **Inflammatory Biochemical Mediators**

Name	Effects
IL-1	Increased vascular permeability T-cell and B-cell activation Fever Synthesis of proteins, such as C-reactive protein, by liver
IL-6	Increased vascular permeability T-cell and B-cell activation Increased immunoglobulin synthesis Fever Synthesis of proteins, such as C-reactive protein, by liver
IL-8	Attraction of Neutrophils to infection site
Leukotrienes	Allow leukocytes to exit the blood vessel and move into the connective tissue
Prostaglandins	Cause vasodilatation, fever, and pain
TNF-α	Increased vascular permeability Chemotaxis T-cell and B-cell activation Fever Synthesis of proteins, such as C-reactive protein, by liver Systemic effects of inflammation such as loss of appetite and increased heart rate

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Chapter Summary Statement

The human immune system is a robust multilayered defense system that functions remarkably to defend the host. At the forefront of this system are the leukocytes, which are derived from hematopoietic precursors in the bone marrow. Following formation, different types of leukocytes mature and develop in different parts of the body. All cells of the innate immune arm are descendants of a common myeloid progenitor cell that further differentiate in the bone marrow. These include neutrophils, eosinophils, basophils, mast cells, monocytes, and macrophages. In contrast, cells of adaptive immunity are descendants of the lymphoid progenitor cell. These include T-, B- and NK-lymphocytes. Notably, NK-lymphocytes have functional features of both innate immunity and adaptive immunity (**Figure 12-17**).

Leukocytes (which are akin to being the foot soldiers of the immune system) and the complement system (which is akin to unmanned drones that "complement" the fighting capabilities of the leukocytes) are the major components of the host immune system. To fight an infection, a highly choregraphed process involving multiple defenders converge to neutralize the infection: (1) tissue-resident leukocytes recognize invading pathogens, then subsequently release cytokines and chemokines; concomitantly, complement proteins act as chemical attractants that recruit more phagocytes to the infection site; (2) leukocytes "sense" these signaling molecules and

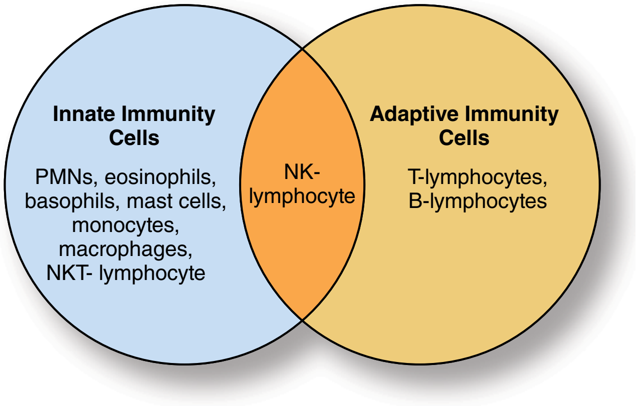
Innate Immunity Cells
PMNs, eosinophils, basophils, mast cells, monocytes, macrophages, NKT- lymphocyte
NK-lymphocyte
Adaptive Immunity Cells
T-lymphocytes, B-lymphocytes

Figure 12-17 Innate Immunity Cells vs. Adaptive Immunity Cells. The immune system is broadly divided into two categories: innate immunity and adaptive immunity. Innate immunity is the first line of defense and consists of the "first responders." In contrast, adaptive immunity response lags, but it offers specificity and immunological memory. Note that NK-lymphocytes have traits of both innate and adaptive immunity.

are drawn to the targeted infection site (chemotaxis) through a process known as transendothelial migration; (3) leukocytes—namely neutrophils and macrophages—reach the targeted site and phagocytose the invading pathogen.[4]

Inflammation is the body's reaction to injury or invasion by disease-producing organisms. The inflammatory response concentrates host immune components at the site of an infection to eliminate microorganisms and heal damaged tissue. It relies on both the physical actions of leukocytes and the biochemical compounds that these cells produce. Acute inflammation is a short-term, normal process that protects and heals the body. Chronic inflammation is a long-lived, out-of-control response that continues for more than a few weeks. In chronic inflammation, the immune system response can sometimes become so intense that it begins to harm the body that it is trying to protect. Tissue damage is the hallmark of chronic inflammation.

Periodontal disease is a bacterial infection that induces an inflammatory response in the periodontal tissues. Chapter 13 focuses on the host immune response to periodontal disease.

Section 4

Focus on Patients

Evidence in Action

Case 1: You injure your arm by accidentally stabbing it with an ice pick. Within minutes following the injury, you note some changes in the tissues around the injury. What changes in the tissues should you expect if your body responds with a typical inflammatory response?

Case 2: A new patient in your office reports a history of rheumatic heart disease at a very early age on her health questionnaire. The patient explains that her physician at the time thought that the rheumatic heart disease was a result of a streptococcal skin infection that she developed a few months prior to the discovery of the heart disease, but that the physician did not explain any of the details of these conditions to her. Explain how an infection of the skin could result in damage to the heart tissue.

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CHAPTER 13

Host Immunoinflammatory Response to Dental Biofilm

- Section 1 The Host Immunoinflammatory Response in Periodontal Disease
  - Factors Enhancing the Microbial Challenge
  - Factors Modulating the Host Immunoinflammatory Response
  - Inflammation: A Protective Host Response That Can Turn Harmful
  - Inflammatory Biochemical Mediators of the Host Response
  - Current Theory of Pathogenesis
- Section 2 Histologic Stages in the Development of Periodontal Disease
- Section 3 The Impact of the Host Immunoinflammatory Response to Bone Homeostasis
- Section 4 Focus on Patients
  - Clinical Patient Care
  - Evidence in Action
  - Ethical Dilemma

CLINICAL APPLICATION

Members of the dental team will encounter many patients with gingivitis and periodontitis. Recognizing the clinical features of these conditions will become second nature as clinicians gain more valuable experiences in clinical settings. However, understanding the body's underlying defense mechanisms that serve to protect the host, but also damage the periodontium, may not be as straightforward. This chapter explores some of the elements involved in the host immunoinflammatory response to microbial biofilm.

LEARNING OBJECTIVES

- Define the term *host response* and describe its primary function.
- Describe the role the host immunoinflammatory response plays in the pathogenesis of periodontal disease.
- Define the term *biochemical mediator* and list three of these mediators.
- Describe the role of cytokines in the pathogenesis of periodontitis.
- Describe the role of prostaglandins in the pathogenesis of periodontitis.
- Describe the effect of matrix metalloproteinases (MMPs) on periodontal tissues.
- Explain the phases of the bone remodeling cycle.
- Explain the significance of a balanced OPG-to-RANKL ratio.
- Describe the link between periodontitis and RANKL-mediated bone resorption.

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- For each of the histologic stages of gingivitis and periodontitis listed below, name one change in the host immune response likely to be encountered:
    - Bacterial accumulation phase
    - Early gingivitis phase
    - Established gingivitis phase
    - Periodontitis phase

KEY TERMS

Immunoinflammatory response
Virulence factor
Catabasis
Pro-resolving lipid mediators
Biochemical mediators
Cytokines
Prostaglandins
PGE
Matrix metalloproteinases (MMP)
Tissue inhibitors of matrix metalloproteinases (TIMP)
Pathogenesis
Dysbiosis
Bone remodeling
Osteoblasts
Osteoclasts
Receptor activator of nuclear factor kappa-B ligand (RANKL)
Osteoprotegerin (OPG)
Homeostatic condition
Bone resorption

Section 1

The Host Response in Periodontal Disease

As discussed in Chapter 12, the immune system is the defense system of the body that is well equipped to protect us. A hallmark of this defense system is the inflammatory response. When the body encounters an offending agent (e.g., viruses, bacteria, or toxic chemicals) or suffers an injury, it activates the immune system. The immune system sends out its first responders—inflammatory cells and cytokines—which stimulate more inflammatory cells. These cells begin an inflammatory response to trap bacteria and other offending agents or start healing injured tissue. The acute inflammatory response is extremely effective at containing the early spread of infectious microbes and promoting wound healing; however, it is extremely powerful and is not meant to be activated for long periods of time. Typically, acute inflammation resolves and tissue repair proceeds. A balanced inflammatory response will be strong enough to contain and eliminate pathogens, while resolving properly so that the tissue can heal and return to homeostasis. When a tissue does not go through the resolution phase, the result is chronic inflammation. Chronic inflammation can lead to extremely negative consequences such as collateral damage to host tissue.

Periodontal disease is a dental biofilm–induced infection that precipitates a complex, carefully orchestrated host **immunoinflammatory response** that involves the activation of leukocytes and the release of pro- and anti-inflammatory mediators. In some individuals, the immunoinflammatory response is proportionate to the microbial challenge; that is, the immunoinflammatory response can contain the microbial challenge with little to no permanent damage to the surrounding periodontal tissues. In other individuals, however, the immunoinflammatory response may be overwhelming and disproportionate; that is, in its attempt to contain the microbial challenge, the leukocytic infiltration and inflammatory mediators induce bone resorption and attachment loss.[1–3]

To highlight the prevailing thought that periodontal disease is an immunoinflammatory disease, growing evidence has emerged that although bacteria are essential for disease to occur, the presence of suspected periodontal pathogens alone is insufficient to cause the tissue destruction seen in periodontitis. Rather, it appears to be the host immunoinflammatory response, which is triggered by the presence of microbial biofilm, that is the direct cause of nearly all the destruction seen in periodontal disease.[1–4] In other words, the outcome of periodontitis-induced destruction of the periodontal tissues is determined not solely by the microbial challenge, but by the intensity of the individual host immunoinflammatory response. This response is essential for the normal resolution of infection, However, in certain individuals, the microbial challenge may provoke an uncontrolled, disproportionate immunoinflammatory response that leads to permanent tissue damage.

In the instance of periodontal disease, the immunoinflammatory system strives to defend the body against bacteria present in dental biofilm. The body's defenses are activated to eliminate the potential pathogens and limit the spread of infection, not to preserve the host tissues such as the tooth or its supporting attachment apparatus. **Figure 13-1** illustrates the natural chain of events that occurs following the biosynthesis (formation) of active inflammatory mediators that either trigger prolonged, unresolved inflammation (which leads to sustained chronic inflammation) or promote the return to homeostasis. Thus, close coordination of the host immune response to infection and inflammation is vital to an ideal response that does not lead to irreversible tissue loss.

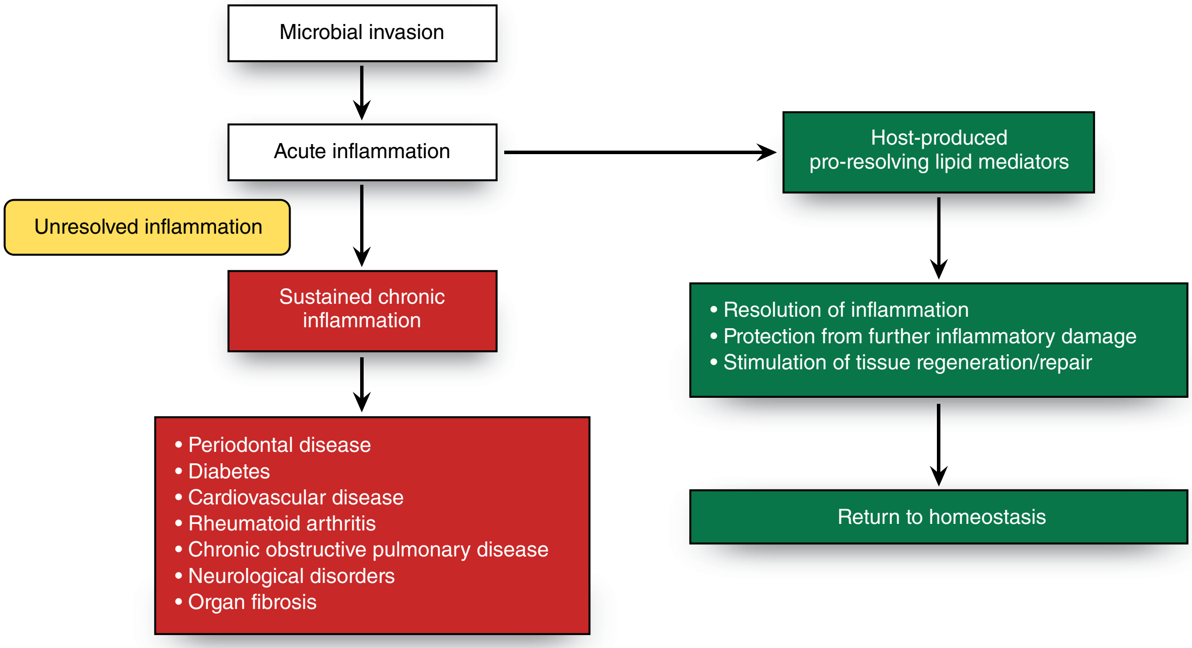
Microbial invasion
Acute inflammation
Host-produced pro-resolving lipid mediators
Unresolved inflammation
Sustained chronic inflammation
• Resolution of inflammation
• Protection from further inflammatory damage
• Stimulation of tissue regeneration/repair
• Periodontal disease
• Diabetes
• Cardiovascular disease
• Rheumatoid arthritis
• Chronic obstructive pulmonary disease
• Neurological disorders
• Organ fibrosis
Return to homeostasis

Figure 13-1 **Inflammatory Response and Resolution.** This flowchart outlines the possible cascade of events following an acute inflammatory response to an event triggered by bacterial challenge to the host. Note that there are two possible host responses: (1) resolution of the acute inflammation and return to a state of homeostasis or (2) lack of resolution to the acute inflammation and conversion to a chronic inflammatory response that can lead to irreversible tissue damage.

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Factors Enhancing the Microbial Challenge

The presence of bacteria is essential for the initiation and progression of periodontal disease. Certain bacteria can provoke the human immunoinflammatory response, which may subsequently lead to irreversible damage to the periodontium.[1,2]

The term **virulence factor** refers to the mechanisms that enable biofilm bacteria to colonize and damage the tissues of the periodontium. Virulence factors may be either structural proteins found on the cell membrane of bacteria or toxic substances that are produced by the bacteria, such as endotoxin. The following are the primary bacterial virulence factors that aggravate periodontal breakdown:

1. **Presence of Lipopolysaccharide (LPS).** Gram-negative bacteria are a component of mature plaque biofilm. Gram-negative bacteria are virulent because of lipopolysaccharide (LPS), an endotoxin present on the outer membrane of the bacteria. LPS can initiate inflammation in periodontal tissues.
2. **Ability to Invade Tissues.** There is good evidence that several subgingival bacteria can invade epithelial cells.[5] Some of the periodontal bacteria—such as *Porphyromonas gingivalis* (P.g) and *Aggregatibacter actinomycetemcomitans* (A.a)—can invade host tissues. To some degree, penetration of tissues may allow the invading bacteria to escape host defense mechanisms.

3. **Ability to Produce Enzymes.** Several periodontal bacteria produce enzymes, such as collagenases and proteases, that directly degrade host proteins which are a basic part of the structure of the periodontium.

Factors Modulating the Host Immunoinflammatory Response

Certain risk factors may modulate the host's susceptibility to periodontal disease by modifying the host response or tissue metabolism.[6,7] These factors include genetic factors, environmental factors, and acquired factors—all of which play important roles in periodontal disease pathogenesis.

1. **Genetic Factors.** Genetic factors appear to contribute to periodontal disease. Studies in twins and families indicate an association between periodontal disease and genetic factors.[8] Diseases with genetic origin such as Papillon–Lefèvre syndrome and leukocyte adhesion deficiency are associated with aggressive types of periodontal disease. Also, variations in genes controlling formation of biochemical mediators can modify the immune response to dental biofilm, increasing the susceptibility to periodontal disease.[9–12]
2. **Environmental Factors.** Tobacco smoking is a known risk factor for periodontal diseases. It can significantly impair the immune and inflammatory system. Smoking has been shown to decrease polymorphonuclear neutrophil (PMN) phagocytic capacity, decrease vascularity of gingival tissues, and affect both T- and B-lymphocyte response to periodontal pathogens.[13–17]
3. **Acquired Factors.** Diabetes mellitus is a known risk factor for periodontal diseases and its progression.[18–22] Abnormal blood glucose levels seen in diabetes mellitus affects the host response by reducing PMN function, increasing interleukin (IL)-1, tumor necrosis factor-α (TNF-α), and prostaglandin E2 (PGE_2) levels in gingival crevicular fluid and reducing growth and proliferation of periodontal ligament fibroblasts and osteoblasts in the periodontium.[7]

Inflammation: A Protective Host Response That Can Turn Harmful

1. **Acute Inflammation**
    a. **Host Response to Microbes.** When pathogenic bacteria successfully infect the periodontium, the body responds by mobilizing defensive immune cells and releasing biochemical mediators to combat the bacteria.
        1. Cells involved in immune and inflammatory processes include inflammatory cells, PMNs, antigen-presenting cells (macrophages and Langerhans cells), T- and B-lymphocytes, fibroblasts, and epithelial cells. The cellular components of the immune and inflammatory processes are discussed in Chapter 12.
        2. Acute inflammation has a host-protective effect. It serves as the body's first line of defense against microbial invasion, eliminates the harmful stimuli, replaces injurious host cells, and creates an environment favorable to tissue repair.
        3. After the microbial challenge has been eliminated, however, the host must be able to dampen (shutdown) the acute inflammatory response.
    b. **Resolution of Acute Inflammation Following Removal of Microbial Challenge**
        1. To maintain a healthy status, both the activation of acute inflammation and its resolution must work in tandem.
            a. It is not the extent of an acute inflammatory response, but rather how effectively the acute inflammation resolves that determines whether the inflammation is favorable or detrimental to the periodontium.
            b. Periodontitis is associated with unresolved inflammation. Indeed, uncontrolled or unresolved inflammation is now recognized as a major driver of human pathologies, including arthritis, asthma, cancers, cardiovascular diseases, and periodontitis.[23–25]
        2. Traditionally, resolution of inflammation was thought to be a passive process resulting from the reduction of biochemical mediators, an eventual disappearance of the inflammatory response, and a return to a state of tissue health. However, recent studies indicate that resolution of inflammation and return to a noninflammatory state is an actively regulated biologic process. This return to homeostasis is referred to as **catabasis**.
        3. Catabasis is thought to be a biologic process that is just as complicated as the onset of inflammation.[1,24,25]

a. **Proinflammation Mediators**
   1. In periodontitis, the lipid mediators of inflammation include prostaglandins, thromboxanes, prostacyclins, and leukotrienes.
   2. The mediators are associated with recruitment of PMNs, destruction of the connective tissue matrix, and resorption of alveolar bone. The over-recruitment or overactivity of PMNs can amplify the inflammatory process which result in tissue damage.

b. **Inflammation-Resolving Mediators**
   1. The resolution of inflammation is regulated by the activity of chemical mediators known as specialized **pro-resolving lipid mediators**.[1,26]
   2. Specialized pro-resolving lipid mediators are actively produced by the body during the resolution phase of acute inflammation. Specialized pro-resolving lipid mediators work in a programmed systematic process to (1) terminate PMN recruitment to the site, (2) stimulate macrophages to remove dead cells, (3) promote antibacterial activities, and (4) promote tissue repair and regeneration to achieve homeostasis.[25]
   3. Future research is focused on understanding the key steps that control the resolution phase of acute inflammation, rather than solely focusing on controlling the bacterial infection or suppressing the activity of pro-inflammatory mediators. This may lead to new treatment protocols.[1]

2. **Chronic Inflammation.** If the host is unable to tamp down the inflammation soon after removal of the microbial challenge, the acute inflammation will progress to uncontrolled and unresolved chronic inflammation that can have pathologic effects on host tissue. Figure 13-1 depicts the possible events in the inflammatory response and resolution of acute inflammation.

Inflammatory Biochemical Mediators of the Host Response

In response to a microbial challenge, host immune cells release biologically active compounds called **biochemical mediators**. These biochemical mediators are the "middlemen" sent by the host cells to activate the inflammatory response. Biochemical mediators of importance in periodontitis are cytokines, prostaglandins, and matrix metalloproteinases (MMPs) (**Table 13-1**).

Table 13-1 **Tissue Destruction by Pro-Inflammatory Biochemical Mediators in Periodontitis**

Mediators	Local Effects
Cytokine IL-1	Stimulates osteoclast activity resulting in bone resorption.[27–30] Induces breakdown of collagen matrix in gingiva, periodontal ligament, and alveolar bone.[28,31]
Cytokine IL-6	Stimulates bone resorption.[27,32] Inhibits bone formation.[27,33]
Cytokine IL-8	Stimulates connective tissue destruction.[27,34] Stimulates bone resorption.[27,29,35,36]
Cytokine TNF-α	Stimulates bone resorption.[27,30,37] Induces breakdown of collagen matrix in gingiva, periodontal ligament, and alveolar bone.[27,30,37]
Prostaglandin E2 (PGE_2)	Stimulates MMP secretion.[4,38] Stimulates bone resorption.[30,39–41]
MMP enzymes	Induce breakdown of the collagen matrix in gingiva, periodontal ligament, and alveolar bone.[30,40]
RANK (Receptor Activator of NF-KB)	The receptor found on the surface of osteoclasts. When bound by its ligand (RANKL), osteoclasts become activated, and release proteolytic enzyme's which destroy connective tissue in bone.[42]

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1. **Cytokines. Cytokines** are powerful regulatory proteins released by host immune cells that influence the behavior of other cells. The cytokine (literally "cell protein") is a molecule that transmits information or signals from one cell to another. When released by host cells, cytokines act as signaling molecules that alert and activate the immune system for help—that is, to send additional phagocytic cells to the site of an infection.
    a. **Sources of Cytokines.** Many different cells, including PMNs, macrophages, B-lymphocytes, epithelial cells, gingival fibroblasts, and osteoblasts, can produce cytokines in response to the bacterial challenge or tissue injury.
    b. **Functions of Cytokines**
        1. Cytokines bind to specific cell surface receptors expressed on the target cell. This is a necessary step for a cytokine to recruit immune cells, such as PMNs and macrophages, to the infection site.
        2. Paradoxically, cytokines have diametrically opposed roles—they can be tissue protective on the one hand, but tissue destructive on the other. Cytokines increase vascular permeability, allowing immune cells and complement to move deeper into the tissues at the infection site. However, cytokines have the potential to initiate and perpetuate irreversible tissue destruction in chronic inflammatory diseases, such as periodontitis.
    c. **Key Cytokines.** Cytokines that play an important role in periodontitis include IL-1, IL-6, IL-8, and TNF-α.[5–9]
2. **Prostaglandins. Prostaglandins** are a group of powerful biochemical mediators derived from fatty acids expressed on the cell surface of most cells. Biologically important prostaglandins are prostaglandins D, E, F, G, H, and I. Prostaglandins of the E series (**PGE**) play an important role in the bone destruction seen in periodontitis.
    a. **Sources of Prostaglandins.** The major source of PGE in inflamed periodontal tissues is the macrophage, although PMNs and gingival fibroblasts also produce them.
    b. **Functions of Prostaglandins**
        1. Prostaglandins increase the permeability and dilation of the blood vessels, which facilitates the rapid influx of PMNs and monocytes to the infected site.
        2. Prostaglandins can trigger osteoclasts to destroy alveolar bone. As stated above, prostaglandins are responsible for most of the alveolar bone destruction seen in periodontitis.
        3. Prostaglandins can promote the overproduction of destructive MMP enzymes.
3. **Matrix Metalloproteinases. Matrix metalloproteinases (MMPs)** are a family of at least 12 different proteolytic enzymes produced by various cells of the body. These enzymes act together to break down the connective tissue matrix.
    a. **Sources of MMPs.** PMNs, macrophages, gingival fibroblasts, and junctional epithelial cells can produce MMPs. The cells providing the major source of MMPs in periodontitis are PMNs and gingival fibroblasts.
    b. **Functions of MMPs**
        1. **MMPs Effects in Health.** In the absence of disease, MMPs facilitate the normal turnover of the periodontal connective tissue matrix. The overactivity of MMPs is tightly regulated by host-derived **tissue inhibitors of matrix metalloproteinases (TIMP)**. The MMP–TIMP balance is critical in maintaining the integrity and the health of the connective tissue. The balance also plays an important role in regulating normal turnover of the extracellular matrix. Any disruption in the MMP–TIMP balance will result in excessive, uncontrolled pathologic breakdown of the connective tissue matrix.
        2. **MMPs Effects in Chronic Infection and Inflammation**
            a. In the presence of chronic bacterial infection, the host immune system responds by releasing elevated levels of cytokines and prostaglandins. These mediators, in turn, stimulate leukocytes and fibroblasts to release MMPs.
            b. As the inflammation becomes more intense, leukocytes and fibroblasts produce excessive amounts of MMPs. The elevated MMP levels soon overwhelm the capacity of TIMPs to regulate the activity of MMP.
            c. In the presence of increased MMP levels, extensive collagen destruction occurs in the periodontal tissues. Note that collagen provides the structural framework of all periodontal tissues. Without collagen, the tissues of the gingiva, periodontal ligament, and supporting alveolar bone degrade. This degradation results in gingival recession, pocket formation, periodontal attachment loss, root exposure, and tooth mobility.

d. Periostat (20-mg doxycycline). A subantimicrobial dose of doxycycline is a host modulating agent that can be prescribed to patients with periodontitis to take orally. At such a low dose, doxycycline does not have an antimicrobial effect. Rather, it functions by inhibiting the activity of MMPs. When prescribed, it should be used as an adjunct to periodontal instrumentation. Chapter 29 offers a more detailed review of host modulating agents.

Current Theory of Pathogenesis

In summary, the chain of events—**pathogenesis**—that leads from health to gingivitis to periodontitis is complex and multilayered. For example, the microbial infection leads to the host immunoinflammatory response, whereas genetic, environmental, and acquired risk factors modify the response. Mediators, such as cytokines and antibodies, are produced by the cells of the immunoinflammatory response. Finally, advances in knowledge about resolution of inflammation have influenced our thinking about the pathogenesis of periodontitis. **Figure 13-2** depicts the interaction of the various factors involved in the pathogenesis of periodontal disease.

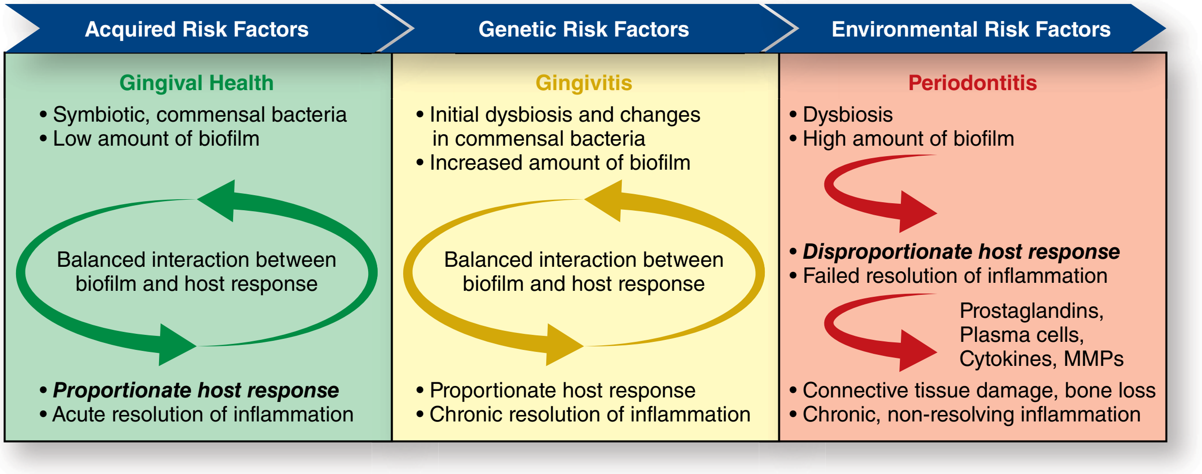
Acquired Risk Factors
Genetic Risk Factors
Environmental Risk Factors
Gingival Health
• Symbiotic, commensal bacteria
• Low amount of biofilm
Balanced interaction between biofilm and host response
• *Proportionate host response*
• Acute resolution of inflammation
Gingivitis
• Initial dysbiosis and changes in commensal bacteria
• Increased amount of biofilm
Balanced interaction between biofilm and host response
• Proportionate host response
• Chronic resolution of inflammation
Periodontitis
• Dysbiosis
• High amount of biofilm
• *Disproportionate host response*
• Failed resolution of inflammation
Prostaglandins, Plasma cells, Cytokines, MMPs
• Connective tissue damage, bone loss
• Chronic, non-resolving inflammation

Figure 13-2 Theory of Pathogenesis. It is well accepted that the development of periodontitis is a multifactorial process through which a bacterial-induced inflammation, modified by acquired, genetic, and environmental risk factors, leads to a disproportionate destructive host response.

Data from Meyle J, Chapple I. Molecular aspects of the pathogenesis of periodontitis. *Periodontol 2000*. 2015;69:7–17. doi:10.1111/prd.12104

1. Gingival health is associated with health-promoting, symbiotic biofilm (i.e., bacteria associated with gingival health).[43] This is mediated by a proportionate immunoinflammatory response to the symbiotic biofilm with subsequent resolution of the inflammation and maintenance of the homeostatic balance.
2. Traditional pathogenic bacteria continue to accumulate when bacterial biofilm is not disturbed; a decrease in number of beneficial bacteria and an increase in number of pathogenic bacteria are usually observed. These changes in biofilm interfere with normal tissue function and host response (**dysbiosis**).[43] In individuals who are not susceptible, the balance between bacterial biofilm and inflammatory response remains stable. In this case, the host response is proportionate and the inflammation is resolved. Therefore, the tissue response is limited to within the gingiva and does not extend deeper in the connective tissue.
3. In susceptible individuals, dysbiotic biofilm activates the host response to produce excessive amounts of cytokines (IL-1, IL-8, IL-6), reactive oxygen species, and MMPs. This overwhelming tissue response leads to collagen breakdown, bone resorption, and periodontal tissue damage. At this stage, inflammation becomes unresolving and chronic. The removal of bacterial biofilm can help reestablish health-promoting biofilm, reducing inflammation, and promoting a balanced biofilm–host response.[44]
4. Environmental, acquired, and genetic risk factors, such as smoking, obesity, poor oral hygiene, and diabetes, are associated with dysbiotic dental biofilm communities. The same disease-modifying risk factors can drastically alter the host immunoinflammatory response to biofilm and impact an individual's susceptibility to periodontal disease.[43]

Section 2

Histologic Stages in the Development of Periodontal Disease

In 1976, Page and Schroeder published the classic, landmark research paper outlining the pathogenesis of gingivitis and periodontitis.[4,45,46] They described four distinct histologic stages in the development of periodontal disease: (1) initial lesion, (2) early lesion, (3) established lesion, and (4) advanced lesion. For clarity, these terms appear in the discussion below, but more is understood today about the host immune response related to these stages than when they initially published their descriptions (**Boxes 13-1** to **13-4** and Figures 13-3 to 13-6).

Box 13-1 Bacterial Accumulation (Initial Lesion)

1. **Bacterial Features.** Bacteria colonize the tooth surface near the gingival margin (**Figure 13-3**).
2. **Cellular Features.** The presence of Gram-negative bacteria and their metabolic products initiates the host immune response.
    a. In response to the bacterial pathogens, the junctional epithelial cells release various biochemical mediators, including cytokines, PGE_2, MMPs, and TNF-α.
    b. These mediators stimulate the immune response, recruiting polymorphonuclear leukocytes (PMNs) to the site.
    c. **Transendothelial Migration of PMNs**
        1. The PMNs need to reach the sulcus in order to fight the bacterial infection. To do this, PMNs must travel through the gingival connective tissue and the junctional epithelium to exit out into gingival sulcus.
        2. As PMNs pass into the gingival connective tissue they release cytokines. Cytokines released by PMNs destroy healthy gingival connective tissue, creating a pathway that allows the PMNs to move quickly through the tissue.
        3. The goal of the PMNs is to reach the bacteria in the sulcus and destroy them. The damage to the healthy connective tissue is not normally a concern. In a healthy body, tissue will be repaired after the bacterial infection is brought under control.
    d. PMNs migrate into sulcus and phagocytize bacteria.
    e. The presence of Gram-negative bacteria activates the complement system.
3. **Tissue-Level Features**
    a. Initial location of the plaque biofilm is supragingival.
    b. Vascular dilatation occurs in the arterioles, capillaries, and venules in the dentogingival complex.
    c. Gingival crevicular fluid increases in volume.
4. **Clinical Features**
    a. At this stage, the gingiva looks healthy clinically.
    b. This initial lesion phase develops 2 to 4 days following plaque biofilm accumulation.
5. **Outcome of Host Response**
    a. The host response is successful if most of the bacteria are destroyed.
    b. If the bacterial infection is brought under control—through the efforts of the immune system and effective plaque biofilm control—the body can repair the destruction caused by the immune response.
    c. If the bacterial pathogens are not controlled, however, early gingivitis develops.

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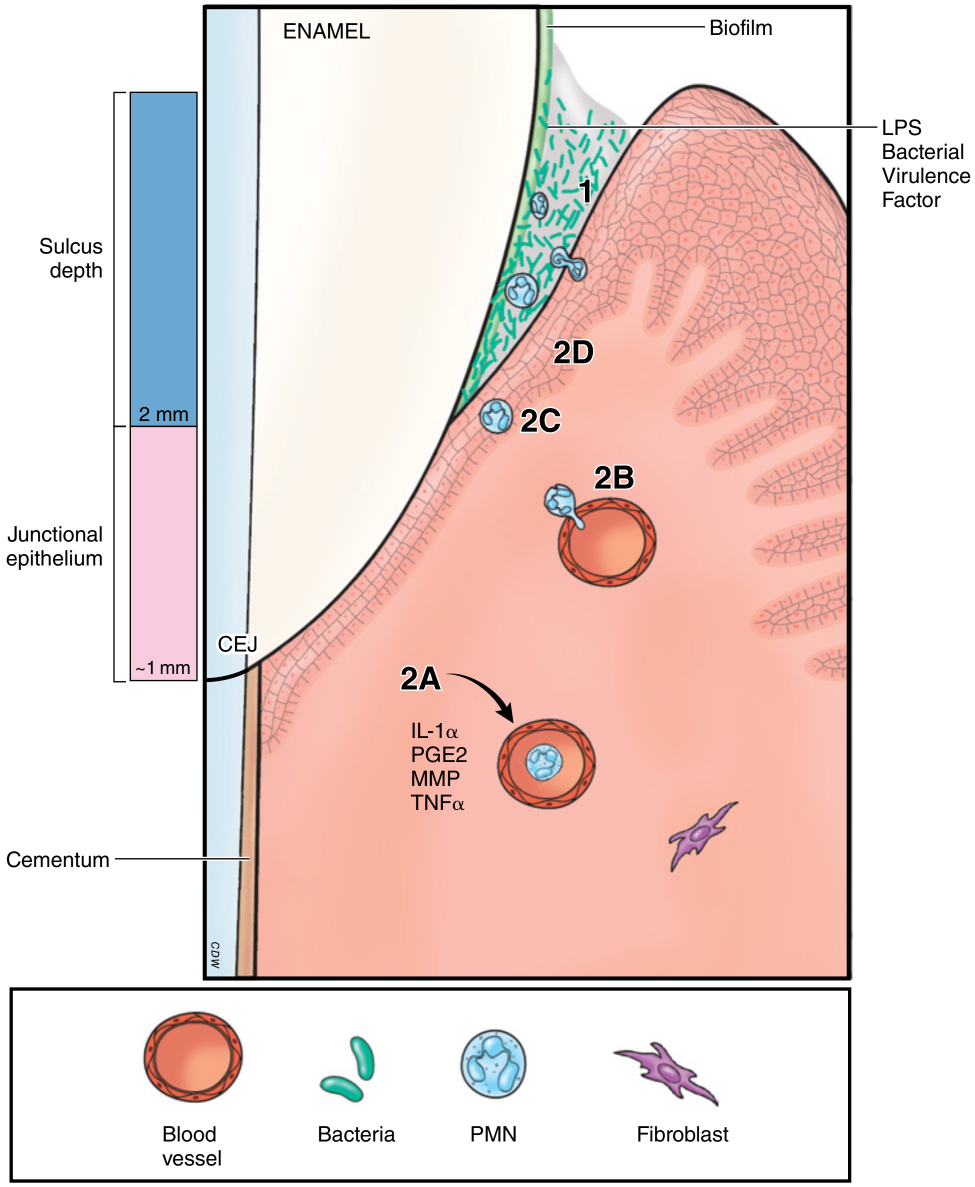
ENAMEL
Biofilm
LPS
Bacterial
Virulence
Factor
Sulcus
depth
1
2D
2 mm
2C
2B
Junctional
epithelium
CEJ
~1 mm
2A
IL-1α
PGE2
MMP
TNFα
Cementum
CDW
Blood
vessel
Bacteria
PMN
Fibroblast

Figure 13-3 Bacterial Accumulation (Initial Lesion). This first phase is characterized by bacterial colonization near the gingival margin, increased vascular dilatation, and PMN migration into the gingival sulcus. Although the host immune and inflammatory responses are activated, the gingival tissue looks clinically healthy in this phase.

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Box 13-2 Early Gingivitis (Early Lesion)

1. **Bacterial Features.** Bacterial accumulation continues and biofilm maturation occurs. This results in the production of bacterial toxins and byproducts that penetrate the junctional epithelium (**Figure 13-4**).
2. **Cellular Features: Amplification of PMN Transendothelial Migration and Chemotaxis**
    a. Cytokines released by the junctional epithelial cells in response to the increased bacterial challenge attract additional cellular defenders to the site.
    b. Increased permeability of the blood vessels allows larger numbers of PMNs to move into the gingival connective tissue near the infection site. On their way to the infection site, PMNs destroy additional healthy gingival connective tissue as they rush toward the bacterial invaders in the sulcus.
    c. PMNs then migrate through the junctional epithelium to form a "wall of cells" between the biofilm and the sulcus wall. These PMNs comprise the most important component of the local defense against bacteria.
    d. Cytokines released by the PMNs cause localized destruction of the connective tissue. This tissue destruction allows more PMNs to migrate through the connective tissue toward the sulcus. In an effort to protect the host tissues, PMNs neutralize the invading microorganisms through the process of phagocytosis.
3. **Migration of Additional Cellular Defenders**
    a. Macrophages are recruited to the connective tissue. These cells release biochemical mediators, including cytokines, PGE_2, and MMPs, which recruit additional immune cells to the infection site.
    b. T-lymphocytes migrate to the connective tissue and produce cytokines and antibodies. The early lesion is predominantly a T-cell lesion.
4. **Tissue-Level Features**
    a. Sulcular epithelium and the adjacent connective tissue are affected the most: collagen loss of 60% to 70% is noted at this stage.
    b. Sulcular epithelium starts forming epithelial ridges (epithelial extensions that protrude into connective tissue) due to inflammatory changes.
    c. Junctional epithelial cells start to proliferate.
5. **Clinical Features**
    a. Inflammatory changes such as edema and redness of gingival marginal tissue can be observed clinically.
    b. Early lesion phase develops 4 to 7 days following plaque biofilm accumulation. Duration of early gingivitis can vary between individuals.
6. **Outcome of Host Response at the Stage**
    a. The large number of PMNs, macrophages, and T-lymphocytes may control the bacterial pathogens.
    b. Initiation of good patient self-care can disrupt the plaque biofilm and result in a return to health. If the bacterial infection is brought under control—through the efforts of the immune system and effective plaque biofilm control—the body can repair the destruction caused by the immune response.
    c. If the host immune response fails to "hold the line," the early lesion will progress to the "established gingivitis"—the next phase of disease progression.

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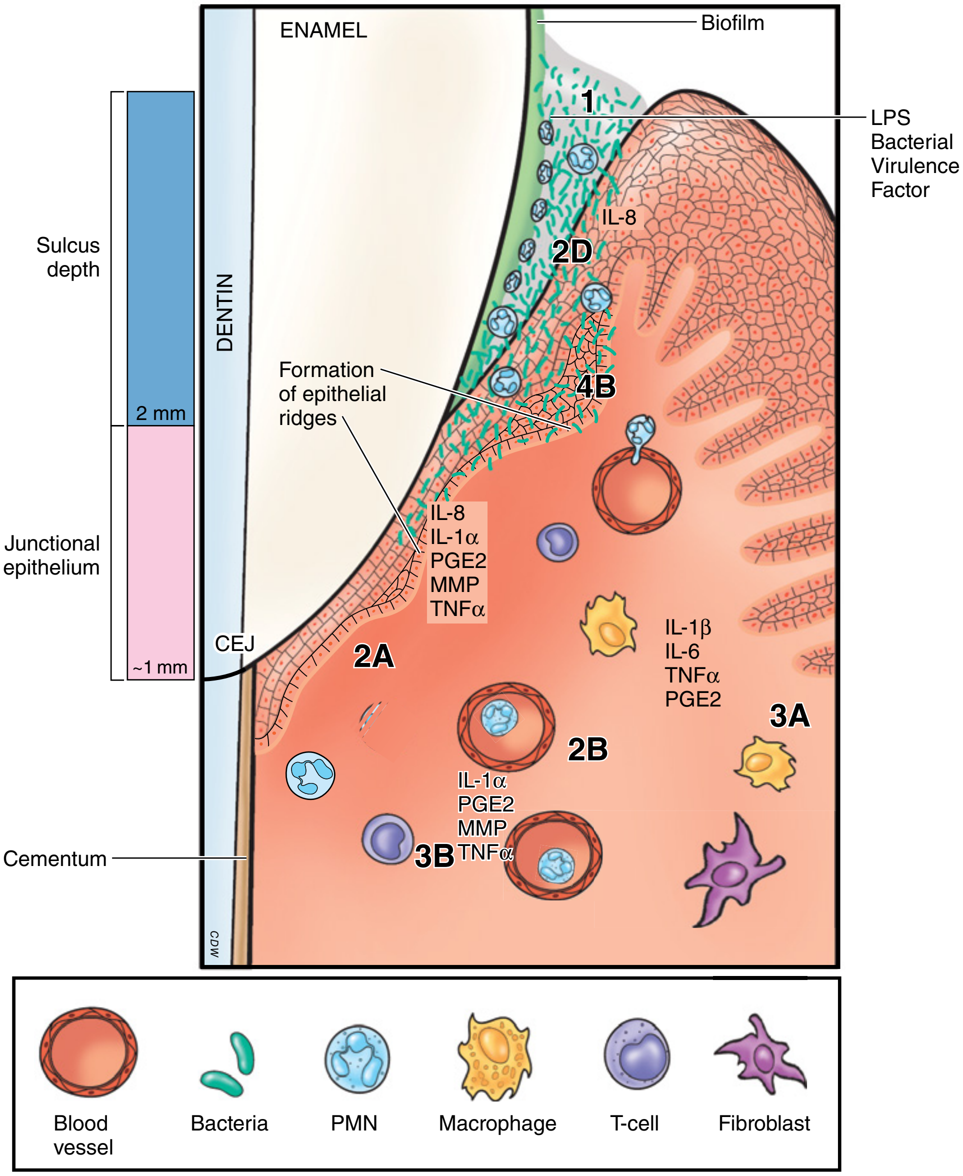
ENAMEL
Biofilm
1
LPS
Bacterial
Virulence
Factor
Sulcus
depth
DENTIN
IL-8
2D
Formation
of epithelial
ridges
4B
2 mm
IL-8
IL-1α
PGE2
MMP
TNFα
Junctional
epithelium
IL-1β
IL-6
TNFα
PGE2
CEJ
2A
~1 mm
3A
2B
IL-1α
PGE2
MMP
TNFα
3B
Cementum
CDW
Blood
vessel
Bacteria
PMN
Macrophage
T-cell
Fibroblast

Figure 13-4 Early Gingivitis (Early Lesion). The biofilm overgrowth phase is characterized by subgingival plaque biofilm formation and intensified immune and inflammatory response compared to plaque biofilm accumulation (initial lesion) phase. Early inflammatory changes are observed clinically as redness and swelling of the marginal gingiva.

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Box 13-3 Established Gingivitis (Established Lesion)

1. **Bacterial Features.** Plaque biofilm extends subgingivally into the gingival sulcus, disrupting the attachment of the coronal-most portion of the junctional epithelium from the tooth surface (**Figure 13-5**).
2. **Cellular Features: Migration of Additional Cellular Defenders to the Site**
   a. Large numbers of subgingival bacteria stimulate the epithelial cells to secrete more cytokines, resulting in greater recruitment of additional PMNs, macrophages, and lymphocytes.
   b. The established lesion is predominated by the presence of plasma cells (leukocytes that produce antibodies) in the affected connective tissue zone. Hence, the established lesion is considered to be a plasma cell lesion. PMNs, macrophages, and lymphocytes continue to assist in fighting the bacteria in the sulcus.
   c. Plasma cells produce large quantities of antibodies to assist in controlling the bacterial challenge.
   d. The immune system sends more immune cells to fight the bacteria. More toxic chemicals are released and additional healthy connective tissue is destroyed.
      1. Cytokines, PGE_2, and MMPs are produced by macrophages exposed to Gram-negative bacteria.
      2. Cytokines recruit additional macrophages and lymphocytes to the area.
      3. PGE_2 and the MMPs initiate collagen destruction.
      4. Gingival fibroblasts are stimulated to produce additional PGE_2 and MMPs.
3. **Tissue-Level Features**
   a. Epithelial ridges extend deeper in connective tissue to maintain epithelial integrity.
   b. Junctional epithelium loosens its attachment to the root surface and starts to transform into pocket epithelium. Pocket epithelium is thinner and more permeable compared to junctional epithelium.
   c. Continued collagen loss in the infiltrated connective tissue zone.
4. **Clinical Features**
   a. All the usual clinical features of gingivitis are evident in this phase, but are more accentuated compared to the initial stage and early stage.
   b. Established gingivitis is generally observed 21 days after plaque biofilm accumulation.
5. **Outcome of Host Response**
   a. In many individuals, the host response is adequate to contain the bacterial challenge during this phase.
   b. Periodontal instrumentation and patient education, at this point, can be helpful in controlling the bacterial challenge. The combination of professional treatment and good patient self-care can stop the bacterial challenge and return the periodontium to health.
   c. In certain susceptible individuals, if the bacterial infection is not controlled, established gingivitis progresses to periodontitis. Unfortunately, no one can predict when and if established gingivitis will ever progress to periodontitis. Current research is directed to trying to determine which individuals are at risk for developing periodontitis.

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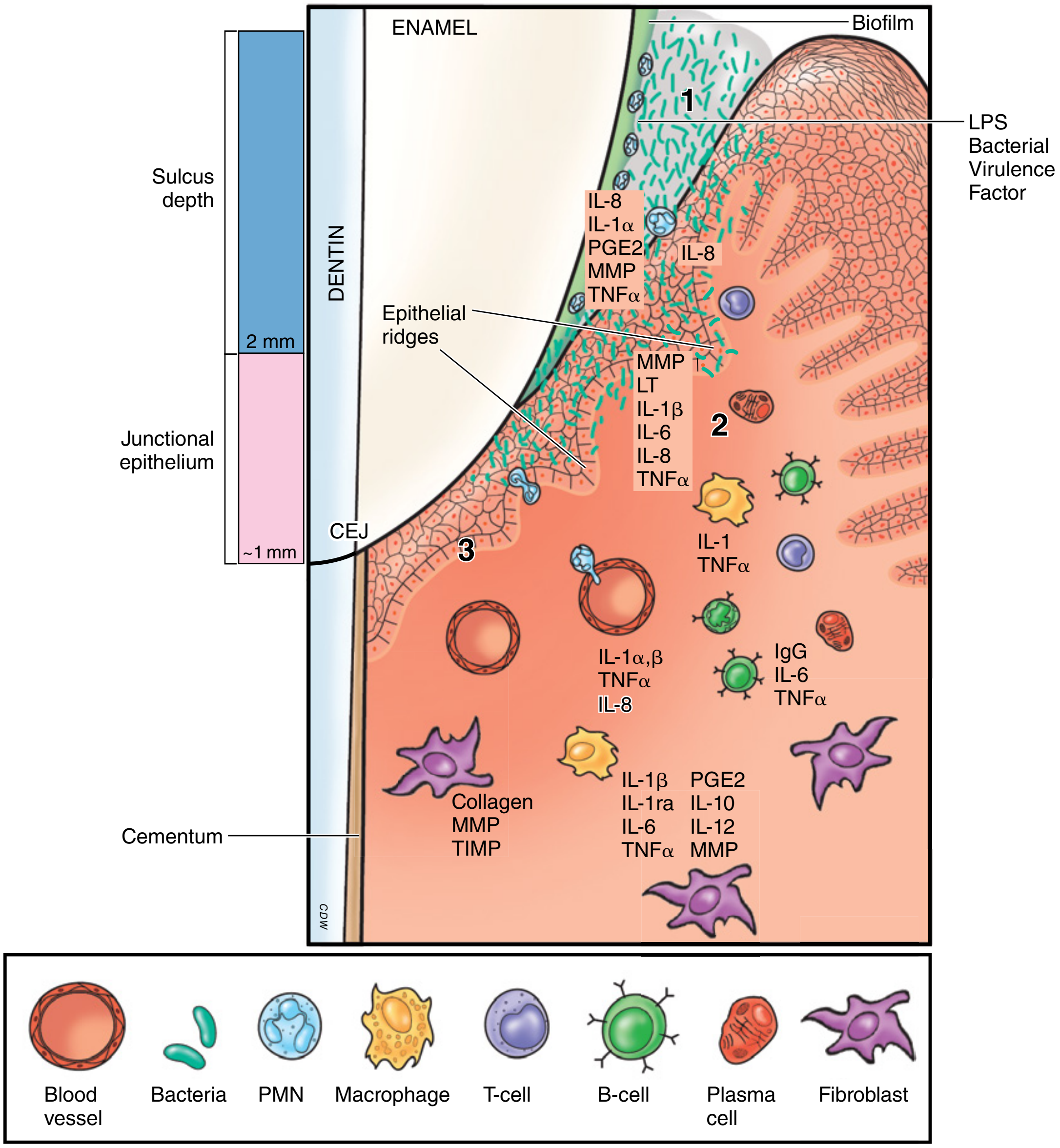
Sulcus depth
2 mm
Junctional epithelium
~1 mm
Cementum
DENTIN
ENAMEL
Biofilm
LPS
Bacterial
Virulence
Factor
1
IL-8
IL-1α
PGE2
MMP
TNFα
IL-8
Epithelial ridges
MMP
LT
IL-1β
IL-6
IL-8
TNFα
2
CEJ
3
IL-1
TNFα
IL-1α,β
TNFα
IL-8
IgG
IL-6
TNFα
Collagen
MMP
TIMP
IL-1β
IL-1ra
IL-6
TNFα
PGE2
IL-10
IL-12
MMP
CDW
Blood vessel
Bacteria
PMN
Macrophage
T-cell
B-cell
Plasma cell
Fibroblast

Figure 13-5 Established Gingivitis (Established Lesion): The Subgingival Plaque Phase. Subgingival plaque biofilm extends to junctional epithelium. Increased cellular infiltrate and collagen breakdown in connective tissue is observed. All clinical characteristics of gingivitis are evident in this phase.

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Box 13-4 Periodontitis (Advanced Lesion)

1. **Bacterial Features.** Plaque biofilm grows laterally and apically along the root surface. The periodontal pocket provides an ideal protected environment for continued growth of subgingival bacteria, presenting a chronic, repeated challenge to the host (**Figure 13-6**).
2. **Cellular Features: Host Response Intensifies**
    a. The bacterial infection becomes chronic, leading to chronic inflammation. The immune response becomes so intense that it begins to harm the periodontium.[27,40]
    b. Cellular defenders intensify their defense against the bacterial pathogens.
        1. PMNs, macrophages, and epithelial cells produce cytokines that cause destruction of the gingival connective tissue and periodontal ligament fibers.
        2. Macrophages produce high concentrations of cytokines, PGE_2, and MMPs that result in destruction of connective tissue and alveolar bone.[30,40]
        3. MMPs mediate destruction of the extracellular matrix of the gingiva, collagen fibers attached at the apical edge of the junctional epithelium, and the periodontal ligament.
        4. PGE_2 mediates bone destruction by stimulating large numbers of osteoclasts to resorb the crest of the alveolar bone. The gingival pocket progresses to become a periodontal pocket.
    c. The tissue destruction caused by the host immune response now overwhelms the host's ability to repair the damage.
3. **Tissue-Level Features (Destruction of Periodontal Tissues Ensues)**
    a. Cells of the junctional epithelium migrate apically on the root surface, resulting in the development of a periodontal pocket.
    b. Gingival fibroblasts shift to a state that favors the destruction of the gingival connective tissue and periodontal ligament fibers (Figure 13-6).
    c. Osteoclasts destroy the crest of the alveolar bone (Figure 13-6).
4. **Clinical Features**
    a. This advanced lesion phase is characterized by periodontal pocket formation, bleeding on probing, destruction of the periodontal ligament, alveolar bone loss, furcation involvement, and tooth mobility.
    b. These tissue changes—apical migration of junctional epithelium, connective tissue destruction, periodontal ligament destruction, and alveolar bone loss—are not reversible.
5. **Outcome of Host Response**
    a. Chronic infection by the periodontal pathogens induces a chronic inflammatory response. The chronic inflammation destroys periodontal tissues and causes more damage to the periodontium than the bacterial infection. Irreversible tissue damage is the hallmark of periodontitis.
    b. Factors influencing the host's failure to control the bacterial challenge may include:
        1. Abnormal PMN function.
        2. Persistence and virulence of bacteria in the biofilm.
        3. Acquired and environmental factors, such as smoking and stress.
        4. Systemic factors, such as uncontrolled diabetes mellitus or genetic factors.

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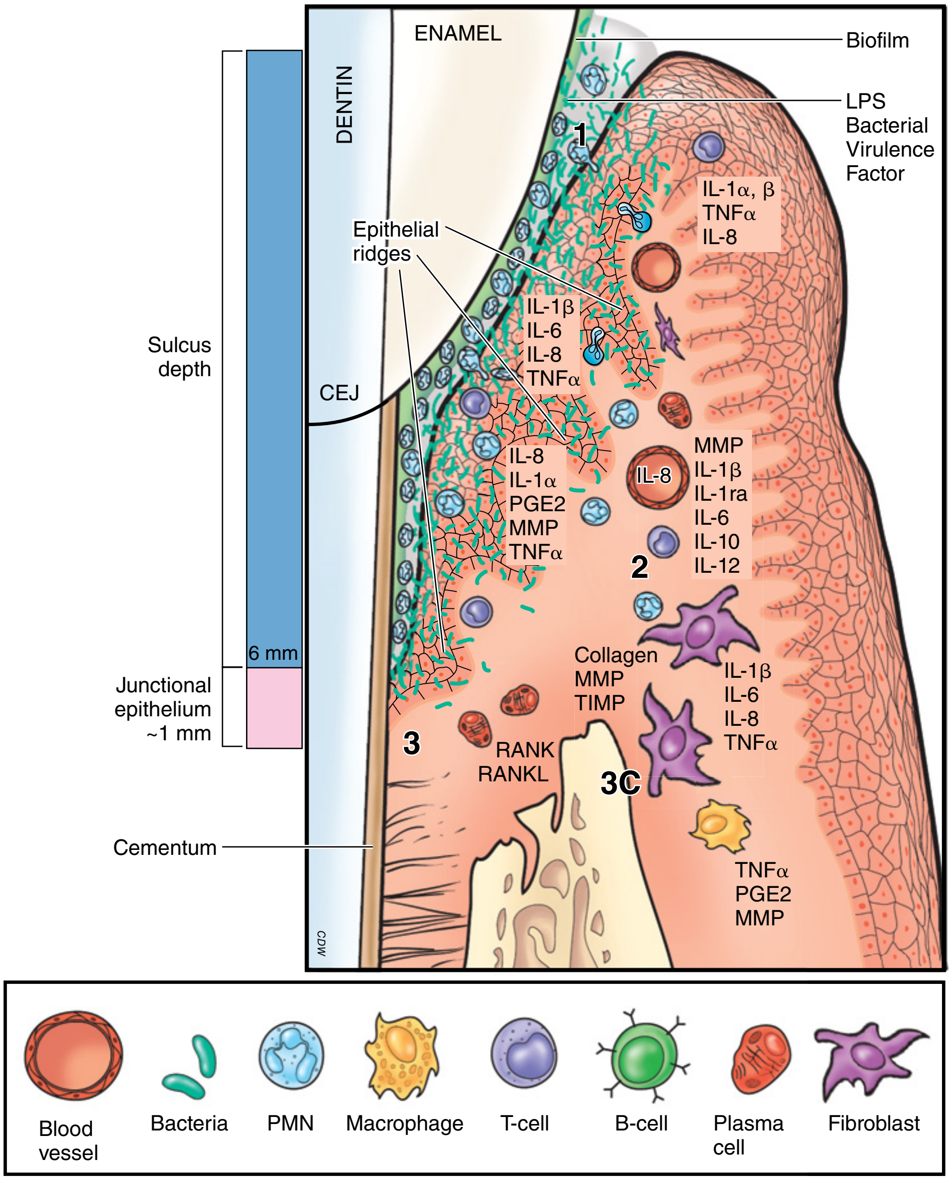
ENAMEL
DENTIN
Biofilm
LPS
Bacterial
Virulence
Factor
1
IL-1α, β
TNFα
IL-8
Epithelial
ridges
IL-1β
IL-6
IL-8
TNFα
Sulcus
depth
CEJ
IL-8
IL-1α
PGE2
MMP
TNFα
IL-8
MMP
IL-1β
IL-1ra
IL-6
IL-10
IL-12
2
6 mm
Collagen
MMP
TIMP
IL-1β
IL-6
IL-8
TNFα
Junctional
epithelium
~1 mm
3
RANK
RANKL
3C
Cementum
TNFα
PGE2
MMP
CDW
Blood
vessel
Bacteria
PMN
Macrophage
T-cell
B-cell
Plasma
cell
Fibroblast

Figure 13-6 **Periodontitis (Advanced Lesion).** This phase is characterized by periodontal pocket formation, bleeding on probing, alveolar bone loss, furcation involvement, and tooth mobility.

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Section 3

The Impact of the Host Immunoinflammatory Response on Bone Homeostasis

Despite its solid appearance, bone is in a constant state of remodeling. **Bone remodeling** is the breakdown of old bone and the subsequent creation (deposition) of new bone. Remodeling occurs constantly; for example, (1) when bones grow, remodeling maintains the shape and structure of the bone, and (2) in response to new stresses applied to a bone, remodeling increases bone strength by adding new bone tissue where appropriate. The remodeling cycle is repeated throughout the course of an individual's life. In the periodontium, remodeling means that at all times some parts of the mandible and maxilla are being resorbed, while other parts are growing by formation of new bone.

1. **The Bone Remodeling Cycle**
    a. The bone remodeling cycle is tightly coordinated by the concerted interactions of two principal cells: the **osteoblasts** (bone-building cells) and the **osteoclasts** (bone-resorbing cells).
        1. Osteoclasts are specialized multinucleated cells.
            a. Embryologically, osteoclasts are derived from hematopoietic progenitors in the bone marrow.
            b. Notably, the differentiation and activation of osteoclasts is mediated by a cell-surface receptor found on hematopoietic precursor cells, known as receptor activator of nuclear factor kappa-β (RANK). When a protein catalyst known as RANKL (discussed below) binds to RANK, these precursor cells differentiate into mature osteoclasts.
            c. Functionally, an osteoclast breaks down (resorbs) existing bone matrix. This function is critical in the maintenance, repair, and remodeling of bone.
        2. Osteoblasts are specialized bone-forming cells that synthesize collagen and other bone proteins and are involved in mineralization of the bone matrix.
    b. The normal bone remodeling cycle is an ongoing process that results from the continuous resorption of bone followed by the subsequent deposition of new bone. **Figure 13-7** depicts the sequential phases of the bone remodeling cycle.
        1. **Resorption phase:** Remodeling is initiated by the attraction of osteoclasts to the bone surface. The osteoclasts break down the bone mineral matrix, creating erosion cavities in the bone.
        2. **Reversal phase:** In this phase, the osteoclasts cease the process of bone resorption and detach from the erosion cavity. Mononuclear cells then adhere to the erosion cavity and release signals that attract osteoblasts to the eroded area.
        3. **Formation phase:** Osteoblasts line the erosion cavity and form a matrix to replace resorbed bone with new bone. Under normal conditions, the amount of new mineralized bone is equal to the amount of bone resorbed by the osteoclasts.
        4. **Resting phase:** A lengthy resting period follows the formation phase. The resting phase is maintained until the next bone remodeling cycle.
2. **Regulation of the Bone Remodeling Cycle.** Control of the normal bone remodeling cycle is governed by an intricate signaling mechanism that occurs between the immature osteoclasts and osteoblasts.
    a. **The "Main Players" in Bone Metabolism**
        1. **Receptor activator of nuclear factor kappa-β (κB) ligand (RANKL)** is a protein catalyst expressed (produced and released) on the membrane of an osteoblast. RANKL is known as an osteoclastic differentiation factor because it plays a role in the differentiation of osteoclastic precursors into mature activated osteoclasts.
            a. Bone resorption occurs when RANKL binds to RANK expressed on the cell surfaces of osteoclastic precursor cells.
            b. This, in turn, stimulates the precursor cells to differentiate into osteoclasts, thus playing a critical role in osteoclastogenesis. **Figure 13-8** illustrates the normal physiologic outcome of the RANK–RANKL interaction.

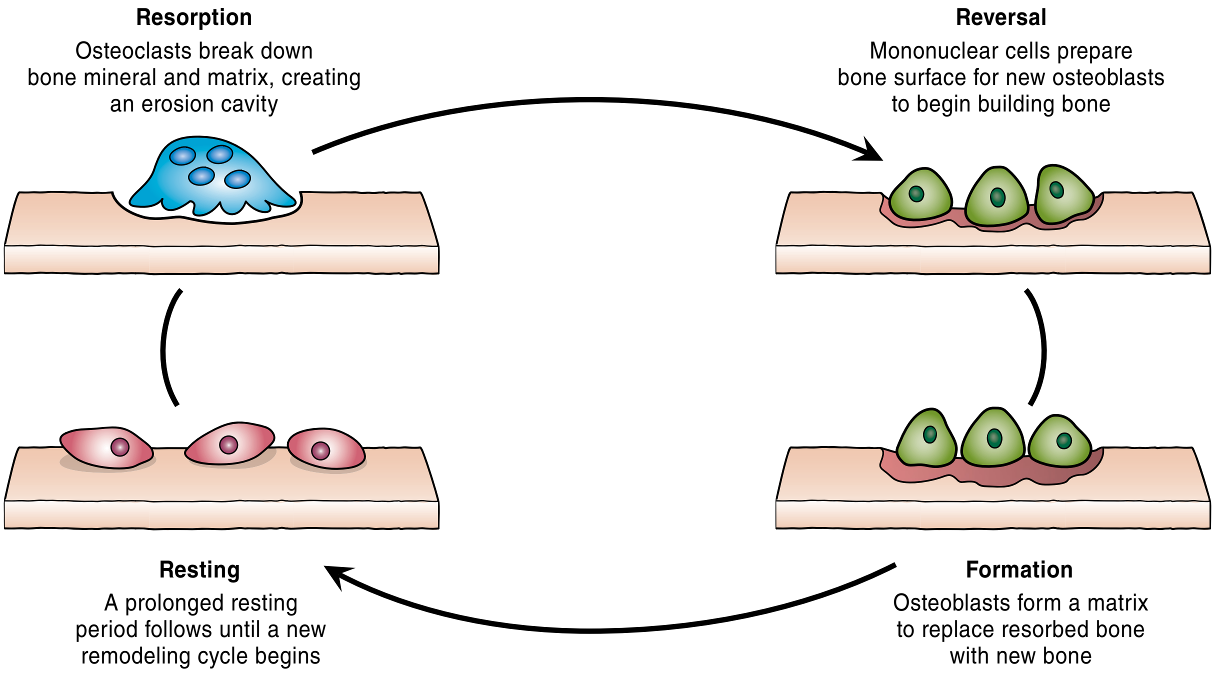
Resorption
Osteoclasts break down bone mineral and matrix, creating an erosion cavity
Reversal
Mononuclear cells prepare bone surface for new osteoblasts to begin building bone
Resting
A prolonged resting period follows until a new remodeling cycle begins
Formation
Osteoblasts form a matrix to replace resorbed bone with new bone

Figure 13-7 The Bone Remodeling Cycle. Normal bone remodeling is an ongoing process that couples continuous resorption of bone with subsequent deposition of new bone. The four phases of this cycle are the resorption phase, reversal phase, formation phase, and the resting phase.

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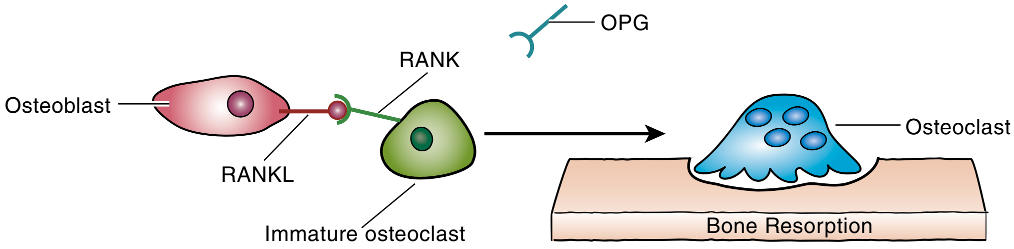
OPG
RANK
Osteoblast
Osteoclast
RANKL
Immature osteoclast
Bone Resorption

Figure 13-8 The Biological Mechanisms Underlying the Normal Bone Remodeling Process. RANKL, RANK, and OPG are the key molecular players in the bone remodeling process. When RANKL binds to RANK, osteoclastic precursors—immature osteoclasts—will differentiate into mature, activated osteoclasts, leading to bone resorption. (The RANK–RANKL interaction leads to bone resorption.)

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2. **Osteoprotegerin (OPG)** is another protein signaling molecule produced by osteoblasts. However, unlike the RANKL-RANK interaction OPG protects bone from excessive resorption by binding to RANKL. By competing with RANK OPG blocks the binding of RANKL to RANK, which subsequently suppresses the differentiation of osteoclastic precursors into mature osteoclasts. This, in turn, preserves bone, thus stabilizing the bone remodeling process. **Figure 13-9** illustrates the important role OPG plays in tightly regulating osteoclastic differentiation, thus maintaining the homeostatic balance between bone resorption by osteoclasts and bone formation by osteoblasts.
3. The OPG–RANKL–RANK signaling pathway thus regulates osteoclastic formation and activation. Additionally, this signaling pathway highlights the close interplay of osteoblasts and osteoclasts working in tandem to direct the normal bone remodeling process.

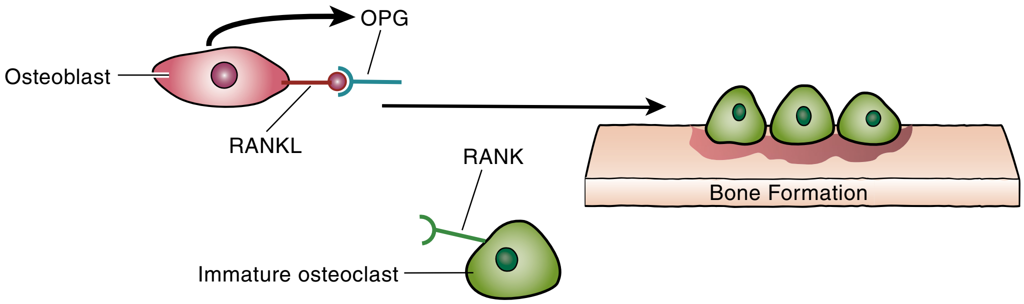

Figure 13-9 Suppression of Osteoclastic Formation by OPG. Like RANKL, OPG is produced by the osteoblast. However, OPG's primary function is to act as a decoy receptor that binds to RANKL expressed on the surface membrane of the osteoblast. When this interaction occurs, it inhibits the binding of RANK to RANKL, thereby suppressing the differentiation and activation of osteoclasts. You might envision this pathway as OPG blocking RANK from binding to RANKL, thus preventing the differentiation of osteoclastic precursor cells into osteoclasts. (The OPG–RANKL interaction leads to bone formation.)

b. **Mechanism of Bone Metabolism.** Bone metabolism is a dynamic process that balances bone formation and bone resorption. The OPG–RANKL–RANK balance is an important factor in regulating alveolar bone resorption.
   1. A **homeostatic condition** occurs when the body is able to maintain stable levels of alveolar bone.
      a. Homeostatic conditions are thought to exist when the levels of RANKL and OPG are in balance in the periodontal tissues.
      b. When the RANKL–OPG levels are in balance, OPG blocks RANKL, thus inhibiting the activation of osteoclasts. Bone resorption is inhibited, and thus alveolar bone levels remain stable.
   2. **Bone resorption** occurs when osteoclasts are stimulated by RANKL to resorb alveolar bone.
      a. When the periodontium is inflamed, levels of OPG decrease.
      b. When the OPG-to-RANKL levels are out of balance in the periodontal tissues, osteoclast activity stimulates resorption of alveolar bone.

c. **Biochemical Mediators.** Various molecular factors can either activate or inhibit the RANKL–RANK–OPG pathway. **Table 13-2** summarizes the stimulatory mediators and inhibitory mediators that govern this pathway.

Table 13-2 Bone Metabolism: A Dynamic Process

Bone Resorption	Bone Formation
■ OPG-to-RANKL levels are out of balance. ■ RANKL stimulates the differentiation of osteoclastic precursors to mature activated osteoclasts, which leads to bone resorption.	■ OPG-to-RANKL levels are in balance. ■ OPG blocks RANKL, thus inhibiting differentiation of osteoclastic precursors into mature activated osteoclasts.

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   1. Examples of molecules that stimulate bone resorption (Figure 13-9):
      a. RANKL
      b. Stimulatory cytokines—pro-inflammatory mediators such as IL-1β, TNF-α, IL-6, IL-11, and IL-17.
      c. Prostaglandin E2 (PGE_2)—a hormone-like substance.
   2. Examples of molecules that inhibit bone resorption:
      a. OPG—a receptor produced by osteoblasts that inhibits osteoclastic differentiation.
      b. Inhibitory cytokines—anti-inflammatory mediators such as IL-4, IL-10, IL-12, IL-13, IFN-γ.

3. **The Link Between Periodontitis and RANKL-Mediated Bone Resorption.** As discussed, the normal bone remodeling process depends on the delicate balance between the activities of the osteoclast and the osteoblast.
   a. In periodontal disease, however, the invading pathogens trigger the release of proinflammatory cytokines, such as IL-1, IL-6, TNF-α, PGE_2, IL-11, that have significant effects on the bone remodeling process. **Figure 13-10** illustrates the role proinflammatory mediators play in inducing osteoclastic formation.

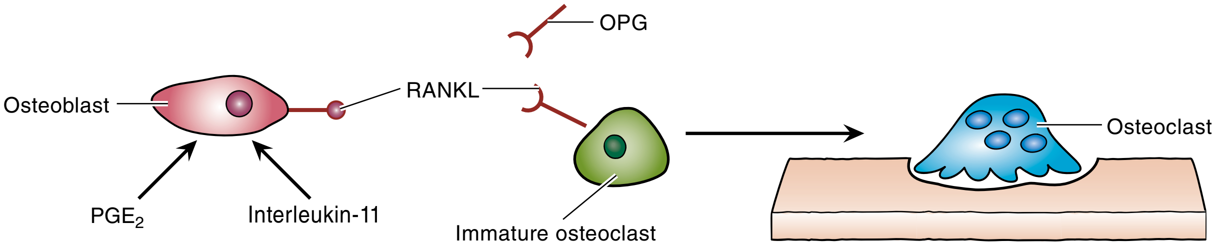

Figure 13-10 Role of Proinflammatory Mediators. In inflammatory disorders (such as periodontitis), elevated levels of proinflammatory cytokines disrupt normal bone remodeling/homeostasis by potently stimulating osteoblasts to produce disproportionate levels of RANKL which leads to excessive RANKL-induced osteoclastic formation. This, in turn, disrupts the homeostatic balance. The result is pathologic bone destruction.

      1. In response to microbial invasion, the body deploys a wide array of different types of immune cells to participate in the counterattack.
      2. These immune cells include PMNs, macrophages, dendritic cells, B-lymphocytes, T-lymphocytes, and monocytes/macrophages.
      3. When reaching the infection site, the immune cells release their full arsenal of proinflammatory mediators, such as IL-1β, TNF-α, IL-6, IL-11, IL-17, and PGE_2, to combat the microbial invasion.
   b. Although these same proinflammatory mediators protect the host from the microbial attack, they also stimulate osteoblasts, fibroblasts, T-lymphocytes, and B-lymphocytes to produce RANKL, which, in turn, activates osteoclasts. If left unabated, the host inflammatory response leads to irreversible destruction of alveolar bone.[45]

Chapter Summary Statement

The importance of the host immunoinflammatory process involved in the pathogenesis of periodontal disease cannot be overstated, especially given that our current understanding of disease pathogenesis is based on the central roles played by various cellular components of the host immune system and biochemical inflammatory mediators in initiating and modulating the host response to microbial biofilm challenge. In many cases, multiple players of the host immune system—neutrophils, macrophages, and lymphocytes—are deployed to the infection site to contain the bacterial challenge with little to no permanent tissue damage. However, in some cases the microbial biofilm, along with any acquired, genetic, or environmental risk factors, can trigger an overwhelming, disproportionate host immunoinflammatory response that is mediated by excessive levels of cytokines, RANKL, MMPs and PGE_2. In turn, the cascade of events leads to (1) the destruction of gingival connective tissue fibers and periodontal ligament fibers and (2) resorption of alveolar bone. ***Therefore, although the host immunoinflammatory response has an important protective role, it can also play a destructive role in certain individuals who are susceptible to periodontitis.***

Section 4

Focus on Patients

Clinical Patient Care

CASE 1

Mrs. Smith is a new patient in the dental office. Mrs. Smith is 45 years of age and has not received regular dental care in the past. Her new employer provides dental insurance, and she comes to your office for oral health care. Mrs. Smith has periodontitis. How will you explain inflammatory periodontal disease to Mrs. Smith?

CASE 2

Two unrelated individuals who have the same level of daily self-care and the same amount of plaque biofilm accumulation do not necessarily develop the same severity of periodontal disease. How do you explain this fact?

CASE 3

Mr. Parker recently moved to your area and is a new patient in the office. He is 28 years old and in good general health. Based on your periodontal and radiographic examination, you detect clinical attachment loss and alveolar bone loss on incisor and molar teeth. How will you explain the role of risk factors in periodontal disease to Mr. Parker?

Evidence in Action

Case 1: In reading a dental journal, you find an article that describes a new medication that is reported to stop collagen destruction by one of the matrix metalloproteinases (MMPs). If this new medication indeed blocks such collagen destruction, what effect might this medication have on a patient with periodontitis? Can you think of another biochemical mediator target that could be inhibited that would benefit a patient with periodontitis?

Ethical Dilemma

The next two patients, Mary and Katherine, are identical twin sisters who are new to your practice. They request that you review their health histories together. You learn that they are 68 years old, both widowed, and live together. They have received routine dental care from their longtime friend and dentist, Dr. Lipmann, who just retired at 80. So, they have decided to become patients in your dental office. They inform you that their mother is still alive at 90, but is edentulous, as she lost all her teeth due to "gum disease." They both state that they are in good health. However, Mary was diagnosed with type II diabetes a few years back. She further states that she hasn't paid much attention to adhering to her physician's instructions to managing her diabetic condition.

Your clinical and radiographic examination of Katherine reveal that she has generally good oral health, with slight gingival inflammation localized to the mandibular anterior region. Her self-care appears to be more than adequate, and you compliment her on it.

Mary's examination is quite the opposite. She presents with generalized periodontitis, and vertical bone loss on her mandibular posterior teeth. She exhibits attachment loss in the 4 to 5 mm range, with localized posterior readings of 6 to 7 mm. Some of her mandibular posterior teeth are mobile. When you present your findings to her, she becomes very agitated. She states that she and Katherine "always" went to Dr. Lipmann together and received the same treatments. She questions why her outcome is so different than that of her sister and asks if you think Dr. Lipmann provided substandard dental care.

1. What factors may have contributed to the different disease presentation seen in these two sisters?
2. How will you discuss with Mary the host immune response regarding her periodontal disease presentation?
3. What potential ethical dilemmas are involved in this situation?

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CHAPTER 14

Impact of Systemic Conditions on Periodontal Health

- Section 1 The Impact of Systemic Disorders on the Oral Cavity
- Section 2 Systemic Disorders/Conditions as Risk Factors for Periodontitis
  - Diabetes Mellitus
  - Stress
  - Hormonal Fluctuations During Puberty, Pregnancy, and Menopause
  - Metabolic Syndrome
  - HIV/AIDS
  - Neutropenia
  - Down syndrome
  - Leukemia
- Section 3 Systemic Medications with Periodontal Side Effects
- Section 4 Focus on Patients
  - Clinical Patient Care
  - Evidence in Action
  - Ethical Dilemma

CLINICAL APPLICATION

The oral cavity is a clear and reliable window to our body's overall health through which the *first* perceptible signs and symptoms of an underlying and undiagnosed systemic disorder/condition can be identified. To put it in another way, a disorder within the oral cavity can be indicative of an underlying systemic condition(s). Diabetes, leukemia, HIV, hormonal fluctuations, and neutropenia are some systemic disorders that are known to have oral manifestations. A meticulous dental examination performed by a trained dental practitioner can therefore offer the earliest glimpse of an underlying, undiagnosed systemic condition, such as diabetes. Indeed, periodontitis may be one of the *first* perceptible signs and symptoms of an underlying, undiagnosed systemic condition. This chapter reviews how common modifying systemic factors—such as diabetes, metabolic syndrome, hormonal changes, stress, and HIV/AIDS—impact oral health. It is worth emphasizing that this chapter solely focuses on one arm of the bidirectional periodontal–systemic link; that is, the effect of systemic disorders/conditions on oral health. Chapter 15 provides an in-depth discussion of the opposite arm of the periodontal–systemic relationship, namely the effect of oral diseases on systemic health.

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LEARNING OBJECTIVES

- Name several systemic diseases/conditions that may modify the host response to periodontal pathogens.
- Engage other health professionals—appropriate to the specific care situation—in shared patient-centered problem-solving.
- Place the interests of patients at the center of interprofessional health care delivery.
- Recognize the importance of educating patients about the relationship between oral health and systemic diseases, states, or conditions (such as the link between diabetes mellitus and periodontitis).
- Discuss the potential implications of these systemic conditions on the periodontium: uncontrolled diabetes, leukemia, and acquired immunodeficiency syndrome (AIDS).
- Describe the significance of the AGE–RAGE interactions and its role in amplifying periodontal inflammation.
- Discuss how hormone alterations may affect the periodontium.
- Define the term *osteoporosis* and discuss the link between skeletal osteoporosis and alveolar bone loss in the jaw.
- Discuss the implications of Down syndrome on the periodontium.
- Name three medications that can cause gingival enlargement.
- For a patient in your care with periodontal disease that is amplified by a systemic condition, explain to your clinical instructor the risk factors that may have contributed to the severity of your patient's periodontal disease.

KEY TERMS

Systemic risk factors
Diabetes mellitus
Well-controlled (diabetes)
Interprofessional collaborative practice
Pregnancy gingivitis
Pregnancy-associated pyogenic granuloma
Menopausal gingivostomatitis
Metabolic syndrome
Acquired immunodeficiency syndrome (AIDS)
Human immunodeficiency virus (HIV)
Linear gingival erythema (LGE)
Osteoporosis
Drug-induced gingival enlargement
Medication-related osteonecrosis of the jaw (MRONJ)
Neutropenia
Down syndrome
Leukemia
Oral mucositis
Phenytoin
Cyclosporine
Nifedipine

Section 1

The Burden of Systemic Disorders on the Oral Cavity

According to data extrapolated from the 2018 U.S National Health Interview Survey, 51.8% of American adults suffer from one of the following debilitating systemic conditions: arthritis, cancer, chronic obstructive pulmonary disease (COPD), coronary heart disease, asthma, diabetes, hepatitis, hypertension, stroke, and weak or failing kidneys, while nearly 30% suffer from two or more of these systemic conditions.[1] Although this trend is anticipated to increase over the next few decades, fatality rates attributed to the above systemic conditions have declined considerably in recent years, reflecting a substantial increase in survival rate for these conditions.

At the same time, as more and more individuals with one or more systemic disorders seek dental care, it is important to keep in mind that these same systemic conditions may inevitably increase an individual's susceptibility to oral diseases, such as periodontitis. This was highlighted in a 2019 systematic review by Seitz et al. which concluded that periodontitis was the dental condition *most* frequently correlated with chronic systemic diseases.[2] Conversely, type 2 diabetes was the chronic systemic disease that had the most frequently observed correlations with dental conditions. In anticipation of a growing number of medically complex patients with either uncontrolled or controlled systemic conditions seeking dental care, the investigators underscore the importance of taking into consideration the ramifications of treating individuals with multiple systemic conditions and chronic dental diseases. They also suggest that heightened awareness is needed to correlate dental conditions with chronic systemic diseases. This, in turn, can open potential opportunities to widen the integration of medicine and dentistry in health care delivery.

In 2021, the U.S. National Institutes of Health published a wide-ranging report titled *Oral Health in America: Advances and Challenges* that outlined several key findings and a call to action to improve the nation's overall health.[3] Along with many other important recommendations, the report highlights that oral and medical conditions share common risk factors, etiologies, and pathogenesis. So, just as medical conditions can influence oral health, so can oral conditions affect systemic health. For over 50 years, this concept has shaped our understanding of the inextricable periodontal–systemic link and continues to be expanded upon with newer insights into the mechanisms of the pathogenesis of systemic conditions and periodontal disease.

Taking all of this into consideration, the need for competent, well-trained dental practitioners knowledgeable in the link between oral health and systemic health will become even more urgent as we move forward to tackling these challenges.

Section 2

Systemic Disorders as Risk Factors for Periodontitis

A number of systemic disorders, diseases, or conditions are associated with an increased risk of developing periodontitis. **Systemic risk factors** are disorders, diseases, or conditions that increase an individual's susceptibility to periodontal infection by modifying or amplifying the host response to microbial infection. Systemic risk factors can be modifiable, such as smoking, or nonmodifiable, such as genetic factors, age, or gender.

Many systemic conditions predispose patients to developing more severe and progressive forms of periodontal disease. Some of the more common systemic conditions that are risk factors for periodontitis include diabetes mellitus; osteoporosis; hormone alterations, which fluctuate as individuals age; certain medications; tobacco use; and genetic influences. Although all systemic risk factors are equally important, cigarette smoking is the most well-known and well-studied of all risk factors, and will be discussed in Chapter 17.

The relationship between periodontal disease and systemic disease is closely intertwined and bidirectional—like a two-way street. On the one side of this bidirectional relationship, certain systemic diseases/conditions may increase the susceptibility of an individual to severe oral diseases, such as periodontitis. Going in the opposite direction, oral diseases may contribute to the development and progression of systemic conditions and diseases in certain susceptible individuals—this part of the relationship will be discussed in detail in Chapter 15.

Diabetes Mellitus

Despite advances in the prevention and medical management, diabetes mellitus continues to be an alarming public health burden in the United States. A 2020 analysis on the prevalence and incidence of diabetes and prediabetes found that while the *overall* number of new diabetes cases (incidence) has decreased in the United States over the last decade, *new* cases have significantly sprung up among individuals younger than 20 years of age, non-Hispanic Blacks, and individuals of Hispanic origin.[4] In the same report, diabetes ranks as the seventh leading cause of death in the United States.[4]

Using figures obtained from the National Health and Nutrition Examination Survey (NHANES), the same report found an alarmingly high prevalence of the disease—approximately 7.3 million adults in the United States have undiagnosed diabetes mellitus, while 27 million American adults have diagnosed diabetes mellitus.[4] The negative impact of diabetes and its complications are not only confined to the United States. The World Health Organization (WHO) estimates that the global prevalence of diabetes has nearly quadrupled from 1980 to 2014 (in 1980, it was estimated that the worldwide number of diabetic individuals was 108 million; in comparison, the number was estimated to be 422 million in 2014), and has risen faster in low-and middle-income countries than in high-income countries.[5]

Among the various risk factors for periodontitis, diabetes mellitus has been confirmed as a major systemic risk factor.[6,7] Diabetic individuals tend to show a greater prevalence and greater severity of periodontitis compared to nondiabetics. Furthermore, the prevalence of periodontitis is higher and its symptoms more severe in individuals with diabetes mellitus compared with nondiabetics.[6,8,9] The link between diabetes and periodontal diseases has been the subject of study for many years. Findings from many studies corroborate that diabetes mellitus contributes to a hyperinflammatory response to oral microbial biofilms and impairs resolution of inflammation and repair, which leads to accelerated periodontal destruction.

1. **Characteristics of Diabetes Mellitus.** **Diabetes mellitus** is a chronic, lifelong metabolic disorder in which the body either fails to produce sufficient amounts of insulin or properly use insulin at the end-target organ level. Insulin is a hormone that regulates the body's metabolism of sugar by converting sugar, starches, and other carbohydrates into energy that the body uses to sustain life.

2. **Diabetes as a Risk Factor for Periodontitis**
   a. **Well-Controlled Diabetes and Periodontal Disease**
      1. Patients with **well-controlled diabetes** are not any more susceptible to periodontal disease compared to nondiabetic patients. Diabetes can be well controlled if the blood glucose levels are stabilized within the recommended range.
      2. The response of individuals with controlled diabetes to nonsurgical periodontal therapy is like that of nondiabetic persons, with similar trends in improved probing depth and attachment gain.[10]
   b. **Undiagnosed or Poorly Controlled Diabetes and Periodontal Disease**
      1. Diabetes mellitus is currently considered as an established risk factor for periodontitis and contributes to increased prevalence, severity, and progression of periodontitis.[6,9,11]
      2. Periodontitis is considered a complication of uncontrolled diabetes. Many epidemiologic studies conclude that periodontitis is more prevalent and severe in individuals with uncontrolled diabetes mellitus versus well-controlled or nondiabetic individuals (**Figures 14-1** to **14-3**). Patients with uncontrolled or undiagnosed diabetes are approximately three times more likely to develop periodontitis.[10–17]

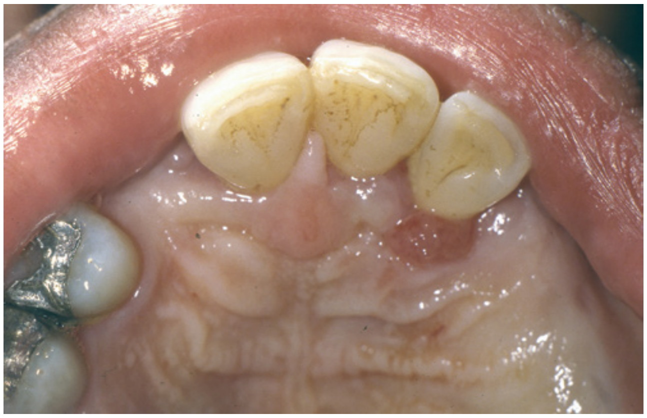

Figure 14-1 Inflammatory Reaction. Note the localized inflammatory swelling of the gingiva on the palatal surface of the maxillary lateral incisor. The patient has uncontrolled diabetes mellitus. This intense inflammatory reaction is typical for individuals with uncontrolled diabetes.

Courtesy of Dr. Ralph Arnold, San Antonio, TX.

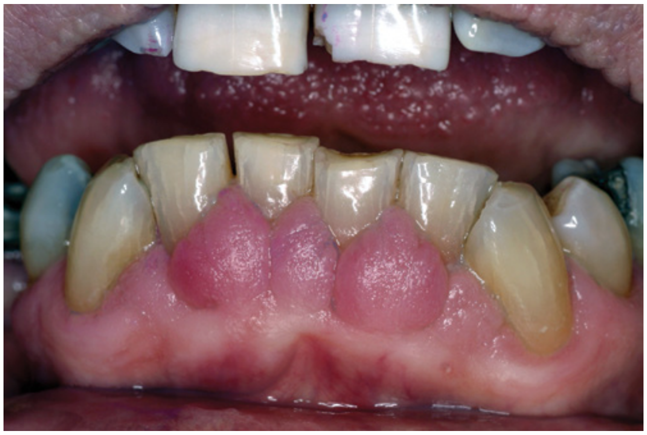

Figure 14-2 Periodontitis Associated with Poorly Controlled Diabetes. Marked tissue changes are evident in this individual with poorly controlled diabetes.

Courtesy of Dr. Richard J. Foster, Educational Solutions and Design, Greensboro, NC.

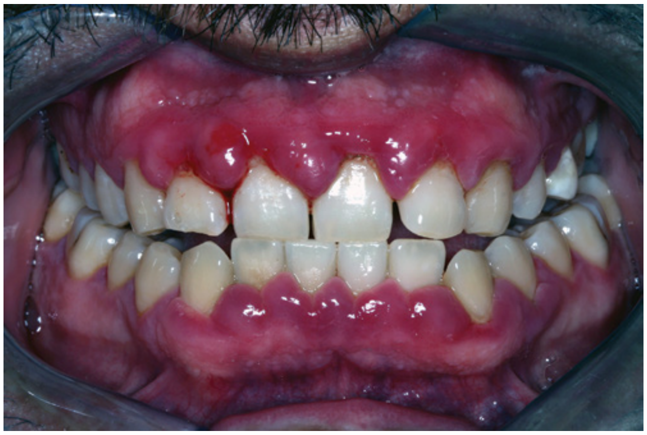

Figure 14-3 Periodontitis Associated with Uncontrolled Diabetes. Examination of this patient revealed pronounced tissue inflammation. The patient had been previously diagnosed with diabetes, but has not been compliant with following through with the physician's and dentist's recommendations to manage the condition.

Courtesy of Dr. Richard J. Foster, Educational Solutions and Design, Greensboro, NC.

a. Individuals with undiagnosed or poorly controlled diabetes have high blood glucose levels. There is a clear relationship between the degree of high blood sugar and the severity of periodontitis.[13,14] As glucose levels increase, individuals with undiagnosed or poorly controlled diabetes experience a dramatic decline in periodontal health.[14,18–20]
b. Periodontal attachment loss (connective tissue destruction and bone loss) occurs more frequently in individuals with poorly controlled diabetes than in individuals with well-controlled diabetes.[18,20–23]
c. An individual with diabetes who smokes and who is age 45 or older, is 20 times more likely than a nondiabetic, nonsmoking individual to experience severe periodontitis.
d. The composition of subgingival periodontal microbiota in a diabetic patient is no different to that found in a nondiabetic patient.[14] In a study comparing diabetic children and their nondiabetic siblings, no differences in the subgingival microflora were found.[24] It can be concluded, then, that diabetes neither favors nor influences the growth of a specific type of periodontal pathogen. Instead, the periodontal destruction seen in diabetic patients may be attributed to the host immunoinflammatory response to nonspecific periodontal pathogens.
e. Wound healing is adversely affected by diabetes, especially if poorly controlled. An unfavorable treatment outcome may occur in long-term maintenance therapy of individuals with poorly controlled diabetes.[25] Individuals with poorly controlled diabetes have a poorer response to nonsurgical and surgical periodontal therapy, more rapid recurrence of deep pockets, and a less favorable long-term response to treatment.

c. **Other Oral Complications of Poorly Controlled Diabetes Mellitus**
  1. Reduced salivary flow, multiple episodes of abscess formation, cheilosis (painful inflammation at the corners of the lip), and burning mouth or tongue are other common complaints of patients with uncontrolled diabetes.[24,26,27] Dental health care professionals should suspect underlying undiagnosed diabetes in a patient who presents with these symptoms. Referral of the patient to a physician or nurse practitioner for evaluation is strongly recommended.
  2. Reduced salivary flow and xerostomia can encourage the overgrowth of *Candida albicans*, which leads to oral candidiasis.[28] It can also create an environment that is conducive to cariogenic bacterial growth, thereby predisposing the individual to a higher risk of caries.
  3. Individuals with undiagnosed or poorly controlled diabetes frequently present with multiple periodontal abscesses, leading to rapid destruction of periodontal bone support.
  4. Although these complications are seen in poorly controlled diabetic patients, well-controlled diabetic patients do not typically present with these conditions because they still have an intact immune defense system that preserves the health of the periodontium and lowers the risk of disease.

3. **Effects of Uncontrolled/Poorly Controlled Diabetes on the Periodontium.** An association between diabetes mellitus and destructive periodontal diseases has been reported in the literature since the 1960s. **Box 14-1** summarizes key points of the association of diabetes mellitus and periodontal disease. Several mechanisms have been proposed to explain the link between diabetes mellitus and periodontitis that focus on the altered hyperinflammatory response to infection, the uncoupling of bone destruction and repair, and the role of the advanced glycation end products in inflammation.[29]

Box 14-1 Key Points: The Diabetes Mellitus–Periodontal Disease Association

- Diabetes mellitus is an established risk factor for periodontitis.
- Diabetes mellitus contributes to a hyperinflammatory response to the microbial challenge in periodontitis and impaired wound healing; these effects are at least partly mediated by the signaling mechanisms of the AGE–RAGE interaction.

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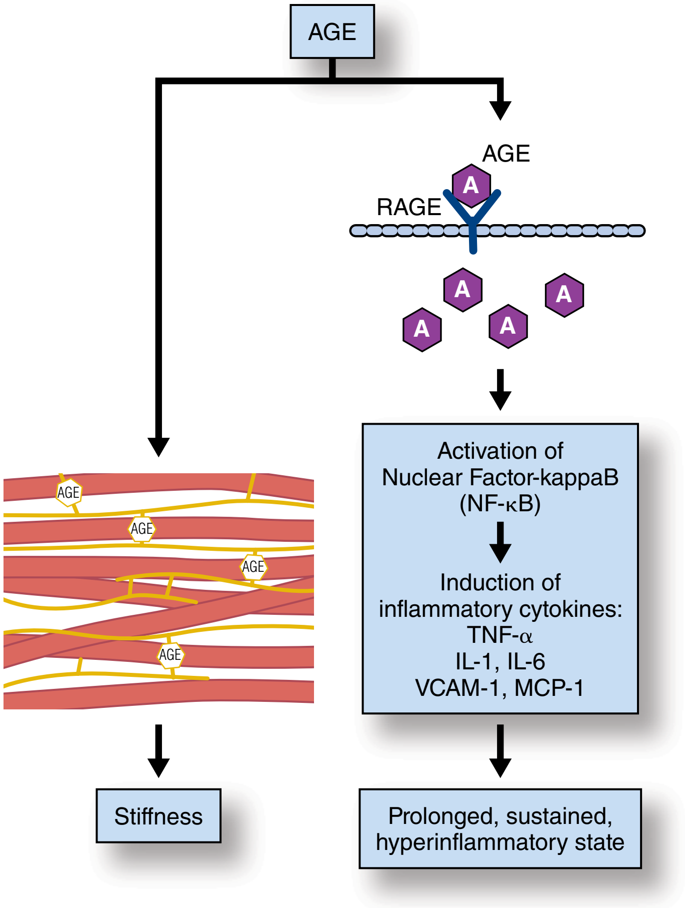
a. **Altered Hyperinflammatory Response to Infection.** As mentioned previously, it was hypothesized that individuals with diabetes mellitus might have different oral microbial profiles than individuals without diabetes mellitus.
   1. Researchers comparing the subgingival bacterial profiles of diabetic individuals with periodontitis versus nondiabetic individuals with periodontitis, however, found that both groups have similar bacterial profiles.[30]
   2. Individuals with diabetes mellitus developed accelerated and exaggerated gingival inflammation compared with those without the disorder, despite a similar bacterial challenge.[31] Thus, the current evidence suggests that it is the host immune response to the oral microbial challenge (rather than the oral microbial challenge alone) that primarily drives the intensity of periodontal destruction seen in patients with diabetes mellitus.
      a. Defective neutrophilic chemotaxis and phagocytosis observed in the diabetic patient impedes the normal initial immune response to infection. Hence, the diabetic patient is more susceptible to infection due to delayed and defective neutrophilic function.
      b. Hyperresponsive monocytes/macrophages in diabetic patients result in the overproduction of proinflammatory cytokines in response to pathogenic invasion. In the presence of periodontal pathogens, the oral cavity is flooded with elevated levels of proinflammatory mediators. Proinflammatory cytokines, such as tumor necrosis factor (TNF)-α, may initiate or worsen the diabetic-related complication or spread to other organ systems and contribute to the worsening of other types of chronic inflammatory conditions, such as cardiovascular disease.
   3. Diabetes mellitus may increase the inflammatory response to bacteria both locally (in the oral cavity) and systemically.

b. **Imbalanced Bone Destruction and Repair.** Research suggests several mechanisms to explain the enhanced alveolar bone destruction seen in individuals with diabetes mellitus.
   1. Alveolar bone is a dynamic, living tissue that is continuously remodeling itself. Bone remodeling maintains the integrity of the bony architecture to meet changing mechanical needs and helps to repair microdamages in the bone matrix. For example, the mandibular condyle is continuously undergoing bone remodeling to respond to everyday mechanical stress that is being applied to it.
      a. The process of bone remodeling is a lifelong process that occurs through the coordinated actions of osteoclasts and osteoblasts.[32]
      b. Osteoclasts are cells that remove old bone (bone resorption). Osteoblasts are cells that then refill the bone cavities with new bone and aid in its mineralization.
      c. In the bone remodeling process, bone formation is tightly coordinated with bone resorption. This coordinated, balanced activity between osteoblasts and osteoclasts is referred to as coupling. Figure 13-7 illustrates the coupling process involved in bone remodeling.
   2. While some of the tissue damage associated with periodontitis likely results from the direct actions of bacteria and infiltrating leukocytes, the rapid alveolar bone loss seen in individuals with diabetes mellitus may result from an uncoupling of the activities of osteoblasts and osteoclasts. Two recent studies indicate that diabetes mellitus could contribute to a net loss of alveolar bone in periodontitis via prolonged osteoclastic formation and activity and increased programmed cell death (apoptosis) of osteoblasts, which impairs bone formation following bone resorption.[33,34]

c. **The Role of Advanced Glycation End Products in Inflammation**
   1. **AGE (Advanced Glycation End Products)**
      a. Glycation is a natural metabolic process in which glucose in the bloodstream (from carbohydrates and sugars) irreversibly binds to biomolecules—such as proteins, lipids, and even nucleic acids—thus transforming the normal function of these constituent biomolecules into harmful, tissue destructive molecules known as advanced glycation end products (AGEs).

        1. In a normal physiologic, healthy state, AGE-modified biomolecules are present but are at such low levels that they do not have any demonstrable pathologic effect.
        2. However, under certain pathologic conditions, such as hyperglycemia in patients with diabetes, AGE levels are increased beyond normal physiologic levels.
    b. AGE-modified proteins have been found in the gingival tissues of diabetic patients with periodontitis and in the saliva of diabetic patients.[35,36]
    c. Excessive accumulation of AGE-modified proteins has been implicated in impairing the physiologic properties and normal function of collagen, which is the major protein found in the human body.

2. AGEs impart their deleterious effects via two mechanisms: (a) cross-linking of AGEs and (b) interactions with RAGEs (receptor for advanced glycation end products).
    a. **Cross-Linking of AGEs**
        1. Formation of cross-linked AGEs on extracellular matrix molecules inextricably links collagen fibers together, thereby rendering the AGE-mediated cross-linked fibers incapable of undergoing the normal tissue remodeling process. This alters the constitution of the extracellular matrix and increases tissue stiffness.
        2. Within the walls of blood vessels, collagen fibers can become glycated and transform into AGE-modified collagen.
            a. This results in increased vascular stiffness and loss of vascular elasticity (due to the inability of the collagen fibers within the blood vessel wall to undergo the normal repair and remodeling mechanisms), thereby leading to impaired blood flow through the vessels, with possible cardiovascular dysfunction.
            b. In turn, the increase in vascular stiffness interferes with numerous physiologic cellular functions that are vital for survival, such as the transfer of oxygen and vital nutrients from blood to cells and the movement of cells (such as leukocytic transendothelial migration) from the bloodstream to the surrounding tissue, and vice versa.
        3. AGE modification of the proteins and lipids is of particular concern for diabetics, who already suffer from the effects of poor glucose control. In fact, elevated levels of AGEs have been implicated in a number of diabetes-related complications, such as neuropathy (nerve damage), retinopathy (eye damage), or nephropathy (kidney disease).
    b. Direct interaction of AGE with a cell surface receptor known as RAGE triggers pathologic tissue destruction. Cell surface receptors are important proteins on the membranes of cells that mediate communication between the cell and the outside world. They act by binding to extracellular molecules.
        1. A receptor known as RAGE (receptor for advanced glycation end products) is expressed on the cell membrane surface of many cells, such as endothelial cells, neurons, mononuclear cells (monocytes/macrophages), and even cells of the periodontium, such as fibroblasts and keratinocytes.
        2. The binding of AGE to RAGE stimulates the above cells to produce and release proinflammatory cytokines, such as TNF-α and interleukin (IL)-1β. Over time, if diabetes is left uncontrolled, the sustained interaction between AGE–RAGE leads to an overproduction of proinflammatory mediators which can have deleterious effects on host tissues.
        3. **Figure 14-4** outlines the effects of AGE on extracellular tissue functions along with the inflammatory effects induced by the AGE–RAGE interaction.
        4. Intraorally, elevated RAGE expression is reported in gingival tissues of individuals with diabetes mellitus and periodontitis.[37–39] Hence, the AGE–RAGE interaction is one of the major factors that contributes to the exaggerated periodontal and systemic inflammation, insulin resistance, and impaired tissue repair seen in diabetic patients with periodontitis.


AGE
AGE
A
RAGE
A
A
A
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AGE
AGE
AGE
AGE
Activation of Nuclear Factor-kappaB (NF-κB)
Induction of inflammatory cytokines:
TNF-α
IL-1, IL-6
VCAM-1, MCP-1
Stiffness
Prolonged, sustained, hyperinflammatory state

Figure 14-4 Systemic Effects of AGE at the Cellular Level. Accumulation of AGE-modified biomolecules interferes with normal cellular processes by (1) forming rigid, stiff cross-links between molecules in the basement membrane of the extracellular matrix and in the wall of blood vessels and (2) engaging the RAGE receptors expressed on the cell membranes of mononuclear cells, endothelial cells, fibroblasts, keratinocytes, and neurons, which perpetuates the prolonged, sustained hyperinflammatory state.

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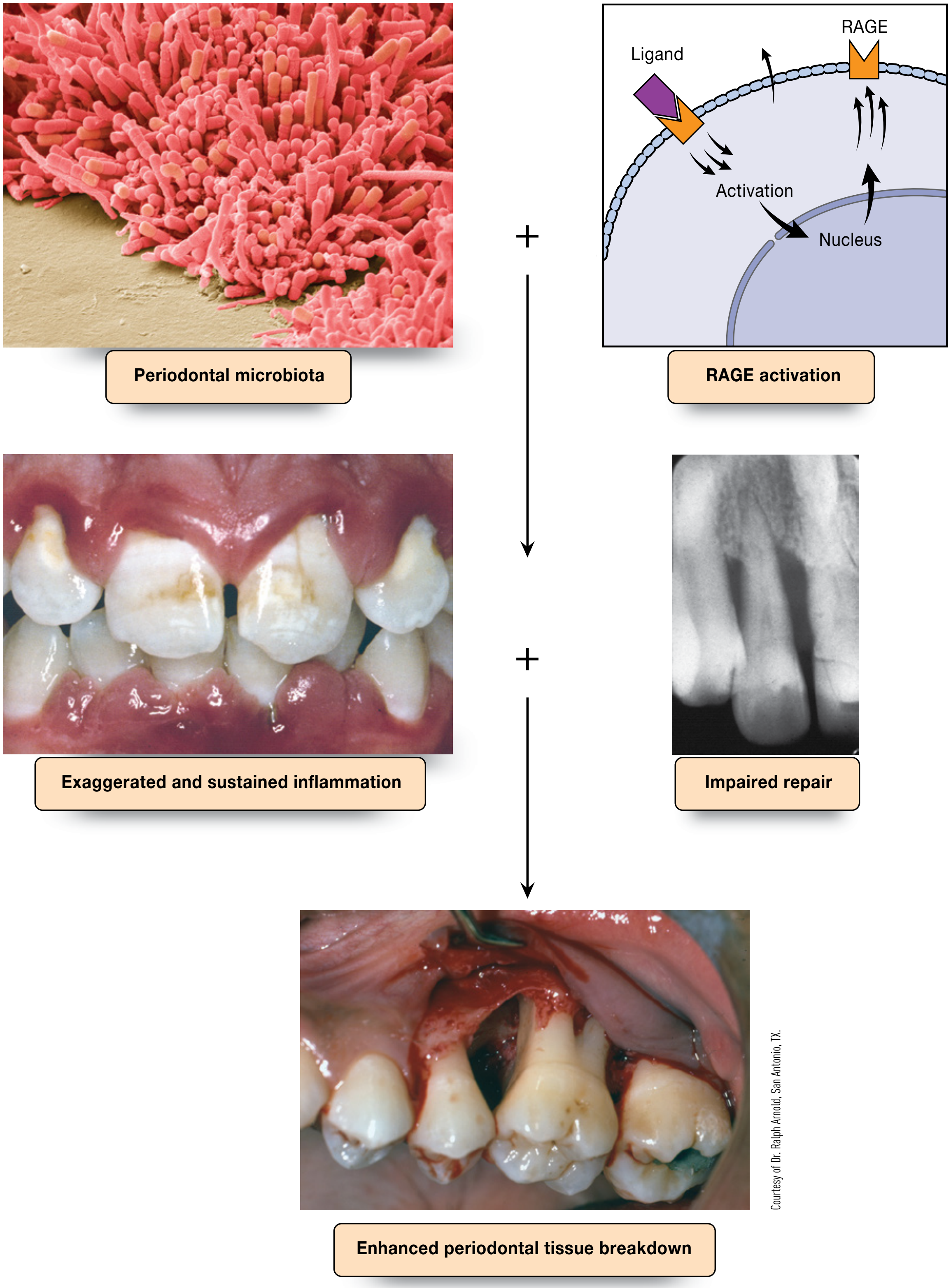
5. Thus, it is believed that the bacterial challenge in an environment of elevated RAGE expression in the periodontium of an individual with diabetes mellitus leads to exaggerated inflammation and impaired repair, which then results in accelerated and severe periodontal destruction.[29] **Figure 14-5** illustrates the postulated effects of uncontrolled diabetes mellitus on the periodontium.

4. **Diabetes Mellitus: Implications for the Dental Hygienist.** **Interprofessional collaborative practice** is a process whereby health care providers from different professional backgrounds work together to deliver the highest quality of patient-centered care. Knowledge about the association between diabetes and periodontal disease should be increased among dental and medical practitioners to prevent, manage, and control both periodontal disease and diabetes effectively.[35]
   a. **Screening for Undiagnosed and Poorly Controlled Diabetes.** Dental professionals are important participants in an interprofessional team approach for diabetes management. The dental team can act as an important point of contact for early screening of undiagnosed diabetes.


Ligand
RAGE
Activation
Nucleus
Periodontal microbiota
+
RAGE activation
Exaggerated and sustained inflammation
+
Impaired repair
Enhanced periodontal tissue breakdown

© Science Photo Library - STEVE GSCHMEISSNER/Brand X Pictures/Getty Images

Courtesy of Dr. Ralph Arnold, San Antonio, TX.

Figure 14-5 **Postulated Effects of Uncontrolled Diabetes Mellitus on the Periodontium.** A model for the role of diabetes mellitus in the accelerated periodontal destruction in individuals with uncontrolled or poorly controlled diabetes mellitus. The microbial challenge combined with an environment of enhanced RAGE expression leads to exaggerated inflammation and impaired repair, which, in turn, causes accelerated and severe periodontal destruction. (RAGE, receptor for advanced glycation end products.)

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    1. Ineffective screening for diabetes is cited as the major contributing factor for the large numbers of persons with undiagnosed diabetes mellitus.[36,40]
    2. Closer collaboration between medical and dental clinical teams is necessary for the joint management of people with diabetes and periodontitis.[15,41] Collaboration of dental professionals with certified diabetes educators (CDEs) will improve the ability of CDEs to educate their clients about oral health topics.[42]
    3. Since 70% of Americans visit their dentist regularly often more frequently than they visit their physician, members of the dental team are well placed to screen patients for diabetes.[43,44] The level of diabetic control and the health status should be carefully monitored for all individuals with diabetes.
  b. **Educating Patients About Oral Health and Diabetes.** People with diabetes mellitus often are not aware of the significance of the relationship between diabetes and oral health.
    1. A study of dental patients with diabetes mellitus noted that 85% had never received information about the relation between diabetes and oral health, and 83% were unaware of the link.
      a. Forty-eight percent of patients with diabetes mellitus believed that the dentist/dental hygienist did not know of their having diabetes.[45]
      b. These results indicate that challenges exist in improving educational programs in dental offices to raise awareness and understanding of the relationship between oral health and overall health.
    2. Educating patients with diabetes about the importance of good oral self-care needs to become a priority for dental hygienists.[46]
      a. Dental hygienists' role in periodontal care and regular, ongoing involvement with these individuals places hygienists in an optimal position to provide this education.[47]
      b. Dental hygienists are very adept and experienced in motivating behavioral changes in their patients and represent an untapped resource for medical colleagues in this role.
    3. Researchers at Columbia University reported on one of the largest studies composed of 6- to 18-year-old individuals with diabetes and 350 individuals in the same age group without diabetes.
      a. The children and adolescents with diabetes showed significantly more periodontal disease with greater attachment loss than those without diabetes.
      b. Based on these findings, children with diabetes should be examined for signs of periodontal disease and receive early intervention and prevention.[10,48]
    4. Dr. Angel Siu Ying Nip presented a paper at the American Diabetes Association 2017 Scientific Sessions entitled "Disordered Eating Behaviors in Youth and Young Adults with Type 1 and Type 2 Diabetes: The Search for Diabetes in Youth Study."[49]
      a. Dr. Nip and colleagues find that more than half of young patients with type 2 diabetes report eating disorders, such as bingeing and vomiting.
      b. It is postulated that the "food-focused" nature of diabetes management lends itself to issues around controlling food and insulin intake.
      c. Given the association between diabetes and disordered eating, early identification is key so patients can receive support.

Stress

1. **Characteristics of Stress**
  a. Stress elicits a physiologic and psychological response, which is one of the body's survival mechanisms. Broadly, there are two types of stress: acute and chronic.
    1. Acute stress (short-term stress) is beneficial because it physiologically prepares the cardiovascular, musculoskeletal, and neuroendocrine systems to respond to a fight-or-flight situation and is considered an essential adaptive instinct to have for survival.
    2. Chronic stress (long-term stress), however, is maladaptive, because it takes a prolonged toll on the body by impeding the normal physiological regulatory mechanisms, which governs homeostasis. This regulatory mechanism is mediated by cortisol, a steroid hormone produced in the adrenal cortex, which is subsequently released to mediate an individual's response to stress. At physiologic levels,

cortisol is an essential hormone needed for maintenance of normal blood pressure and heart rate and regulating inflammation. However, excessive levels of cortisol will disrupt the homeostatic balance and lead to elevated blood pressure and excessively rapid heart rate (tachycardia), which could predispose an individual to cardiovascular complications. Further, with respect to the host immune response, elevated levels of cortisol will intensify the natural immunosuppressive effect of this hormone and increase an individual's susceptibility to infection. **Figure 14-6** depicts the relationship of chronic stress to systemic conditions and periodontal disease.

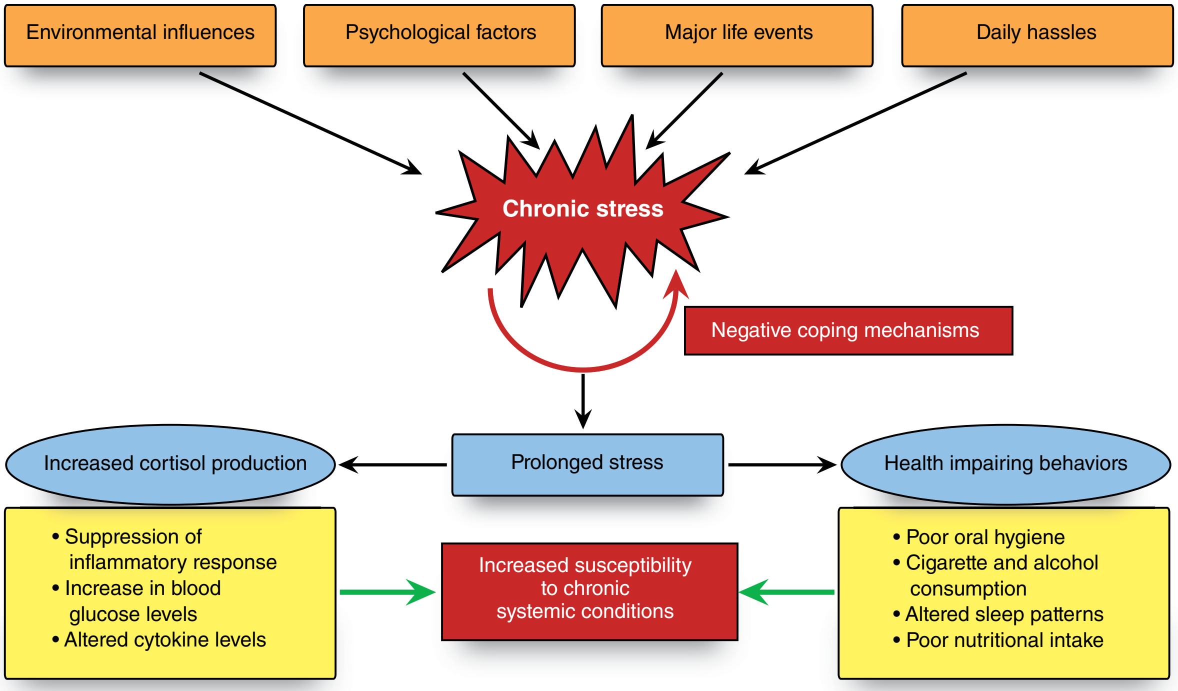
Environmental influences
Psychological factors
Major life events
Daily hassles
Chronic stress
Negative coping mechanisms
Prolonged stress
Increased cortisol production
Health impairing behaviors
• Suppression of inflammatory response
• Increase in blood glucose levels
• Altered cytokine levels
Increased susceptibility to chronic systemic conditions
• Poor oral hygiene
• Cigarette and alcohol consumption
• Altered sleep patterns
• Poor nutritional intake

Figure 14-6 Relationship of Chronic Stress to Systemic Conditions and Periodontal Disease.

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b. The potential effects of chronic stress can manifest as anxiety, depression, impaired cognition, or altered self-esteem.

2. **Stress as a Risk Factor for Periodontitis**
   a. Numerous clinical studies have investigated the correlation between chronic periodontal disease and stress.[50–53]
      1. A majority of studies included in a recent systematic review showed a positive relationship between stress and periodontal disease.[54]
      2. High financial stress and depression are significant risk factors for periodontal disease after adjusting for age, gender, smoking, diabetes, and periodontal microorganisms.[53,55]
   b. Stress-induced behavioral changes may explain the detrimental effect of stress on periodontal health. These behavioral changes include poor self-care,[56] unhealthy dietary habits,[57] increased smoking,[52] increased alcohol consumption,[58] and nonadherence to periodontal maintenance regimens.[59]
      1. The relationship between stress and neglected self-care was observed in a group of medical students preparing for exams. Exam students reported a reduction in thoroughness of oral hygiene behavior

and had higher plaque scores than the control group of students not taking exams.[60] The authors of this study conclude that stress may cause individuals to be neglectful in performing their daily self-care routines, thereby leading to increased biofilm retention.

  2. Stress-induced eating behaviors—such as excessive consumption of refined carbohydrates—may increase accumulation of plaque biofilm.[61]

c. **Implications for the Dental Hygienist**
  1. Prolonged or intense periods of stress may suppress the immune system, which might tip the host–microbial interaction in favor of bacteria causing increased attachment loss. Stress also effects how individuals look after themselves and might lead to less effective daily plaque biofilm removal, increased tobacco use, and poor nutrition.
  2. Managing periodontal health in individuals undergoing significant stress requires recognition of stress as a risk factor for periodontal disease.
  3. Informing patients of the potential adverse effects of stress on their general and oral health is the first step to guiding individuals to think about strategies for stress management. In this context, the dental team may need to closely collaborate with the patient's physician and a mental health care expert to achieve optimal treatment outcomes.
  4. It may be necessary to recommend shorter intervals between professional care appointments to monitor changes to the patient's compliance and behavior.
  5. The 2021 *Oral Health in America: Advances and Challenges* identifies that one of the challenges to health care delivery in the 21st century is unraveling the complex interrelationship between mental health (such as individuals suffering from chronic stress) and oral health.[3]
  6. Patients with mental health issues often have difficulty maintaining a regular oral hygiene regimen or a relationship with an oral health provider. Dental or orofacial pain can worsen depression.
  7. As such, the report concludes that one way to overcome this challenge is by encouraging the dental, medical, and other health professionals to work collectively with the patient to integrate oral, medical, behavioral health care in medical settings, dental clinics, community health centers, and nursing homes and in medical, dental, nursing, and dental hygiene programs.

Hormonal Fluctuations During Puberty, Pregnancy, and Menopause

Fluctuations in the level of sex hormones throughout puberty, pregnancy, menopause, and postmenopause can influence the health of periodontal tissues.

1. **Puberty**
   a. **Impact of Puberty on the Periodontium**
      1. During puberty, elevated levels of estradiol in females and testosterone in males accumulates within the gingival tissues, which amplifies the host inflammatory response to plaque biofilms.
      2. The period around puberty is associated with increased gingival inflammation in both sexes, despite constant plaque biofilm levels.[62–65]
         a. Changes in the composition of the subgingival microbiota, rather than in the amount of plaque biofilm, are the most likely contributing factors.[66]
         b. The increased secretion of steroid hormones during puberty may have a direct association with the prevalence of certain periodontal pathogens.
      3. Pubertal gingivitis occurs equally in girls and boys. The tendency for biofilm-induced gingivitis usually decreases as the young person progresses through puberty.
      4. Clinical features of pubertal gingivitis include:
         a. An accumulation of high levels of plaque biofilm.
         b. Erythematous and edematous gingival tissue that bleeds easily upon probing.
         c. Inflammation that is reversible with meticulous daily self-care; reversible following puberty.
   b. **Implications for the Dental Hygienist**
      1. Dental hygienists should stress that daily attention to biofilm control (self-care) and frequent professional care are very important for gingival health during puberty.[67]

    2. Because early diagnosis ensures the greatest chance for successful treatment, it is important that children and adolescents receive a periodontal examination as part of their routine dental visits.
    3. Depending on the patient's level of self-care, the frequency of professional care may need to be increased.

2. **Pregnancy**
    a. **Impact of Hormonal Fluctuations in Pregnancy on the Periodontium**
        1. Studies show that no pregnancy-related changes occur if the gingiva is clinically healthy.[68,69] However, under the influence of increased sex hormones, the severity and extent of existing gingival inflammation is dramatically exaggerated during pregnancy.[70–74]
        2. Increased levels of estrogen have been shown to alter the microbial profile of subgingival plaque.
            a. In a classic study conducted by Kornman and Loesche examining the subgingival microbial profile in pregnancy, it was found that the second trimester was associated with increased gingival inflammation and increased levels of *Prevotella intermedia*.[75]
            b. It was hypothesized that the increased growth of *P. intermedia* was due to the microorganism's ability to exploit estrogen as a substitute for its natural growth factor, menadione. This finding would suggest that elevated levels of *P. intermedia* is correlated to the gingival inflammation seen in pregnant women.
        3. Gingival inflammation can occur in pregnant women even in the presence of minute amounts of plaque biofilm. Pregnant women tend to exhibit more gingival inflammation than do women 6 months postpartum, despite similar plaque scores and compliance levels.
            a. The likelihood of gingival inflammation increases in the second and third trimesters when elevated estrogen levels in the blood exaggerate the host response to plaque biofilm and other local irritants.[76] Pregnant women, near or at term, produce higher levels of estradiol, estriol, and progesterone compared to nonpregnant women.
            b. Elevated progesterone levels in pregnancy increase capillary permeability and dilation, resulting in increased gingival exudate and edema.[77]
            c. During pregnancy, profound changes in immune response impact the periodontal tissues.[78] High levels of progesterone and estrogen associated with pregnancy have been shown to suppress the immune response to dental plaque biofilm. Polymorphonuclear neutrophil (PMN) chemotaxis and phagocytosis have been reported to be depressed in response to high levels of gestational hormones.[79]
        4. Gingival inflammation initiated by plaque biofilms and exacerbated by hormonal changes in the second and third trimesters of pregnancy is referred to as **pregnancy gingivitis**.
            a. The gingival tissue may be edematous and dark red, with bulbous interdental papillae (**Figures 14-7** and **14-8**).

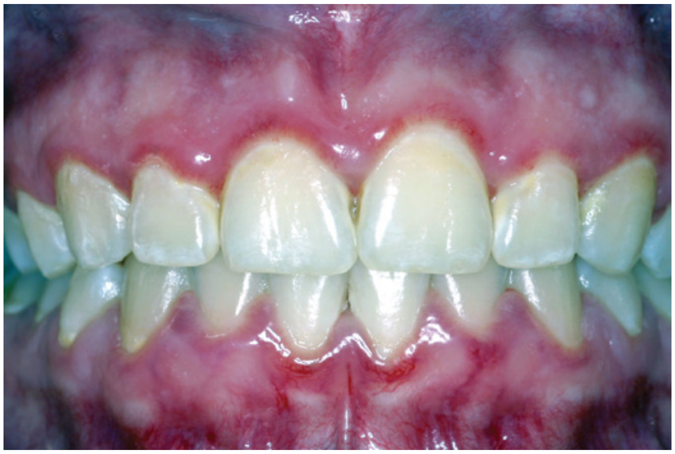

Figure 14-7 Pregnancy Gingivitis. Clinical appearance of reddened, swollen tissues of pregnancy gingivitis.

Courtesy of Dr. Richard J. Foster, Educational Solutions and Design, Greensboro, NC.

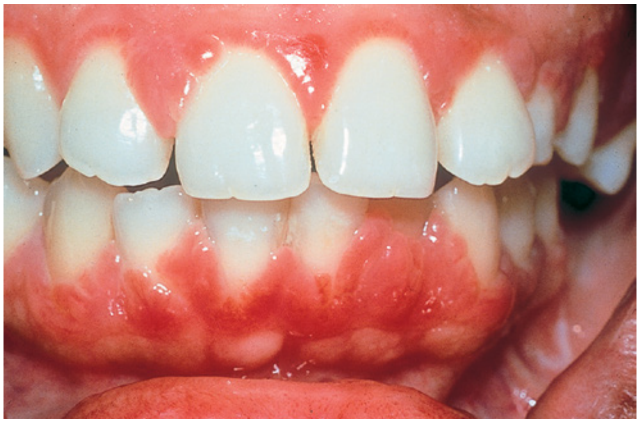

Figure 14-8 Hormonal Gingivitis. Hormonal gingivitis is evident 3 weeks postpartum (after giving birth) in this patient.

Used with permission from Langlais RP. *Color Atlas of Common Oral Diseases*. Wolters Kluwer; 2003. © Wolters Kluwer.

b. In some cases, a gingival papilla can react so strongly to plaque biofilm that a large, localized overgrowth of gingival tissue, called a **pregnancy-associated pyogenic granuloma** (pregnancy tumor), may form on the interdental gingiva or on the gingival margin (**Figure 14-9**).

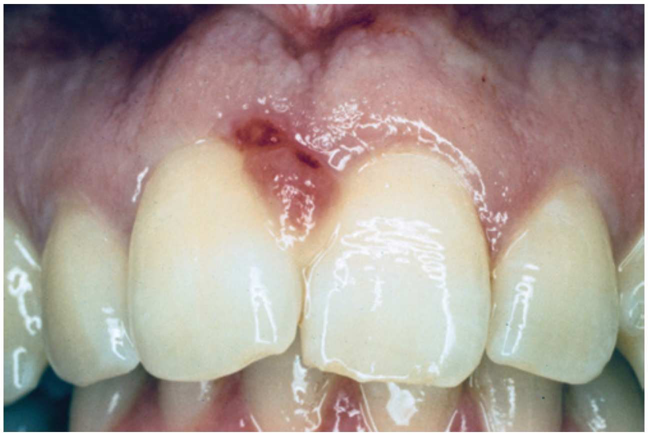

Figure 14-9 **Pregnancy-Associated Pyogenic Granuloma.** The clinical appearance of a pyogenic granuloma (pregnancy tumor).

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1. These growths are benign and are generally not painful.
2. If the growth persists after delivery, it can be surgically removed.

c. Probing depths, bleeding on probing, and crevicular fluid flow are increased in pregnancy gingivitis.

b. **Expectant Patient: Implications for the Dental Hygienist**

1. Dental professionals and gynecologists should educate pregnant patients about the importance of oral health. The perinatal period is an ideal time to educate and perform dental treatment on expectant mothers.[75,80] Pregnancy provides an opportunity to educate women regarding oral health self-care and future child-care.[81]
    a. The dental hygienist should ask the expectant woman if she has any concerns about receiving dental care while pregnant and be ready to address any of her concerns.
    b. Expectant women should be advised that prevention, diagnosis, and treatment of oral diseases—including needed dental x-ray and use of local anesthesia—is safe, beneficial, and can be undertaken any time during pregnancy.[82,83] Also, acute/emergency care may be provided at any time during pregnancy.
    c. Hygienists should encourage behaviors that support good oral health:
        1. Meticulous daily self-care for biofilm control.
        2. Prenatal vitamins, including folic acid to reduce the risk of birth defects such as cleft lip and palate.
        3. Chewing sugarless gums (gum free of sucrose) or candies that are sweetened by noncavity sweeteners (such as aspartame, xylitol, sorbitol, or mannitol) can stimulate salivary flow, dimmish caries risk, and promote remineralization of incipient carious lesions.
        4. Expectant women experiencing frequent nausea and vomiting should be advised that erosion of tooth surfaces might be reduced by:
            a. Eating more frequent, smaller meals consisting of nutritious foods.
            b. A rinse of a teaspoon of baking soda in a cup of water should be used to rinse and spit out after vomiting. Caution the patient to avoid tooth brushing immediately after vomiting as the effect of erosion can be exacerbated because the teeth are still coated by gastric acids.
2. A comprehensive periodontal treatment plan should be developed that includes preventive treatment and maintenance care throughout pregnancy. In addition, the importance of regular dental care and check-ups during the postpartum period and thereafter should be emphasized.

        3. Dental care should be coordinated with the expectant woman's medical care professional to ascertain whether other risk factors—such as gestational diabetes—are present and to advise the medical professional of the periodontal status of the patient and any proposed treatment.[83]

3. **Menopause**
    a. **Impact of Menopause on the Periodontium**
        1. Menopause typically occurs in the fourth-to-sixth decade of a woman's life.
            a. Menopause is a natural condition that all women experience as they age.
                1. Unlike the pubertal stage and the pregnancy stage, menopause is characterized by fluctuating levels of estrogen production from the ovaries; by the time a woman reaches the latter stages of menopause and/or the postmenopausal stage (beyond the sixth decade), estrogen levels have dramatically decreased (**Figure 14-10**).

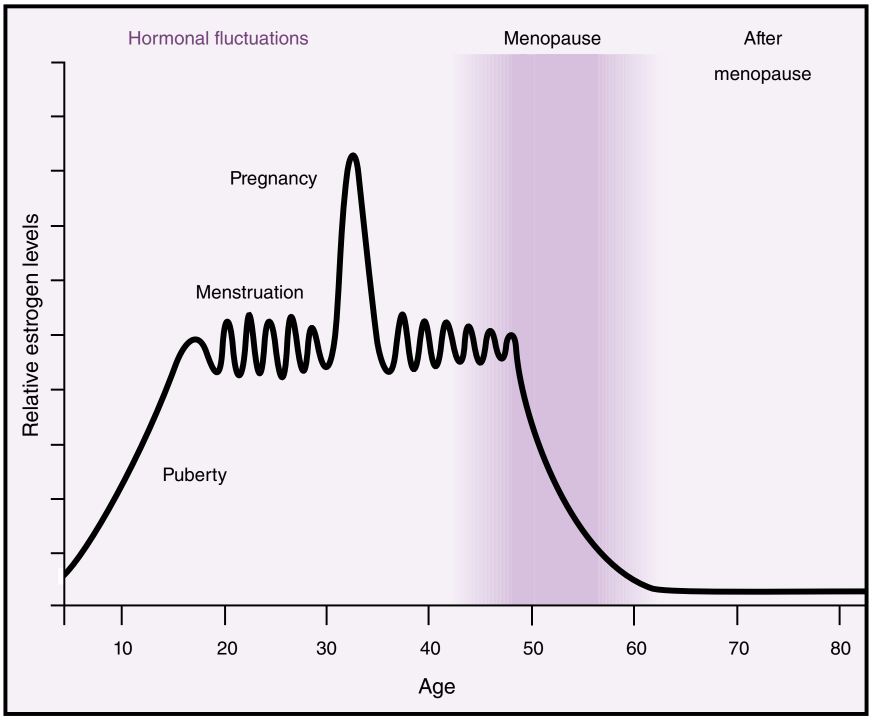

Figure 14-10 **Fluctuation in Estrogen Levels Throughout a Woman's Life.**

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                2. Fluctuating, unstable estrogen levels have widespread systemic effects because estrogen is a key hormone that regulates the function of many parts of the body, including the brain, heart, blood vessels, skin, reproductive organs, and bones.
            b. In menopause, many drastic physical and behavioral changes occur. Several of these changes may adversely affect the oral cavity and make some women more susceptible to periodontal disease.
                1. Dental health care providers may be the first health care professionals to notice the oral and systemic changes that occur during menopause. The periodontium is extremely sensitive to hormonal changes that take place just before and during menopause.
                2. A literature review by Dutt and colleagues compiled data on the major orodental complications observed during menopause. This review observed that the health of the periodontium is most severely affected, followed by dry mouth and burning mouth, which, in turn, may increase the occurrence of oral mucosal and dental diseases, such as candidiasis.[84]

3. When menopause triggers gingival inflammation, it is called **menopausal gingivostomatitis**. Menopausal gingivostomatitis is characterized by gingiva that bleed readily, with an abnormally pale, dry, shiny erythematous appearance.[85]
4. During menopause, sex hormone levels dramatically fluctuate, particularly with respect to estrogen.

c. Altered levels of estrogen during and after menopause may contribute to the loss of bone density. The rapid decline in estrogen can lead to systemic bone loss. **Osteoporosis** is a reduction in bone mass that causes an increased susceptibility to fractures (**Figure 14-11**). Osteopenia is a condition in which there is a lower-than-average bone density but not necessarily an increase in the risk of fracture.

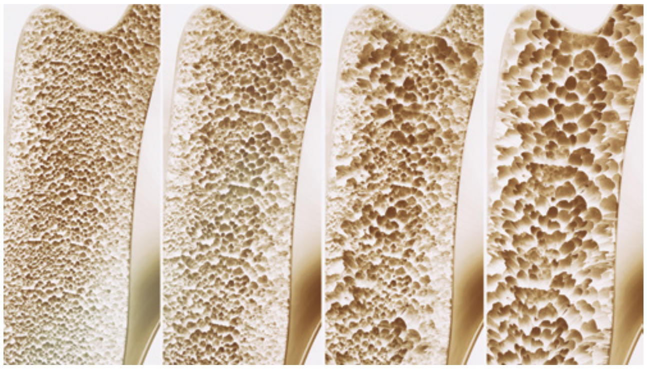

Figure 14-11 The far left illustration depicts the structure of healthy bone. The middle two illustrations show progressive reduction in bone mass. The illustration on the far right depicts the significant reduction in bone mass commonly seen in osteoporosis.

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d. Decreased estrogen levels in women with osteopenia were found to be associated with loss of crestal density of alveolar bone.[86]

2. Bisphosphonates are the most prescribed medications to inhibit the bone resorption of systemic osteoporosis. In dentistry, bisphosphonates are associated with an increased risk of **medication-related osteonecrosis of the jaw (MRONJ)**.[87] MRONJ is a rare disorder characterized by painful areas of exposed bone in the mouth that fail to heal after an extraction or oral surgery procedure. Bisphosphonates and MRONJ are discussed in Chapter 29, Host Modulation Therapy.
3. The same processes that lead to loss of bone in the spine and hips can also lead to loss of alveolar bone.
   a. There may be a link between skeletal osteoporosis, alveolar bone loss in the jaw, and tooth loss.[88–92] Preliminary studies report significant correlations between mandibular bone mineral density and hipbone mineral bone density.[90,91]
   b. A vast number of studies show a positive association between osteoporosis and periodontal destruction.[91,93–97]
   c. However, there are contradictory studies which show no association between osteoporosis and periodontal disease.[98,99] Hence, the relationship between alveolar bone loss and systemic bone loss is not yet fully understood.[100] Thus, more research needs to be conducted to better understand this relationship.
   d. Estrogen replacement therapy improves bone density in postmenopausal women. In a 3-year study, hormone/estrogen replacement therapy significantly increased alveolar bone alveolar bone mass compared with placebo and tended to improve alveolar crest height.[101]

4. Genco and Grossi have proposed a model for estrogen deficiency as a risk factor for periodontal disease (**Figure 14-12**).[94]

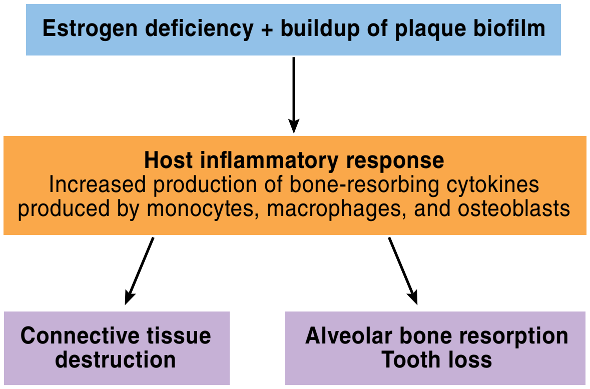
Estrogen deficiency + buildup of plaque biofilm
Host inflammatory response
Increased production of bone-resorbing cytokines produced by monocytes, macrophages, and osteoblasts
Connective tissue destruction
Alveolar bone resorption
Tooth loss

Figure 14-12 Postulated Effects of Estrogen Deficiency on the Periodontium. Estrogen deficiency leads to a decrease in bone mineral density. Together with the oral biofilm infection and the host inflammatory response, the affected site will exhibit accelerated bone loss and more exaggerated connective tissue destruction. Postmenopausal female smokers were more likely to lose alveolar bone height and density than nonsmokers with a similar periodontitis, plaque, and gingival bleeding experience. In addition, both smoking and osteoporosis/osteopenia had a negative impact on alveolar bone.[95]

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b. **Menopause: Implications for the Dental Hygienist**
   1. Dental hygienists and gynecologists should educate postmenopausal women about osteoporosis and interventions such as calcium supplements and weight-bearing exercise.
   2. Meticulous daily self-care, combined with regular professional care, decreases the likelihood of periodontal problems during menopause.
   3. Dental hygienists need to advise patients of the common risk factors, such as smoking, for both osteoporosis and periodontal disease.[95]
   4. Dental hygienists should be aware of the possible oral side effects associated with bisphosphonates, antiresorptive agents, and antiangiogenic medications and should be able recognize the signs and symptoms of MRONJ.
   5. As newer classes of osteoporotic medications are being approved and introduced to the public, dental hygienists should be aware of how they work and what possible oral side-effects these agents may possess.
      a. For example, the U.S. Food and Drug Administration has approved several new classes of antiresorptive agents to prevent and manage osteoporosis in postmenopausal women—namely, selective estrogen receptor modulators (SERMs, e.g., Evista®), parathyroid analogs (e.g., Forteo®), and monoclonal antibodies (e.g., Prolia®).
      b. According to the 2022 American Association of Oral and Maxillofacial Surgeons' Position Paper on MRONJ, SERMs and monoclonal antibodies carry the risk of MRONJ. In contrast, preliminary

evidence suggests that parathyroid analogs may prevent MRONJ and potentially improve wound healing due to a different mechanism of action.

c. Hence, as newer medications are introduced, dental practitioners must constantly stay abreast with all potential oral side-effects associated with these medications.

Metabolic Syndrome

1. **Characteristics of Metabolic Syndrome**
    a. **Metabolic syndrome** refers to a cluster of closely related metabolic disturbances—(1) increased blood pressure, (2) high blood sugar, (3) excess body fat around the waist (abdominal obesity), (4) low levels of HDL (high-density lipoproteins) cholesterol (the “good” cholesterol); and (5) high levels of triglyceride—that tend to occur together and initiate a proinflammatory state that increases the risk for heart disease, stroke, and diabetes.[96]
    b. Early recognition of metabolic syndrome is crucial in identifying patients who are at high risk for developing type 2 diabetes mellitus and cardiovascular disease–related conditions.[97]
        1. A diagnosis of metabolic syndrome is made based on a patient having abdominal obesity with any two of the four following factors: hypertension, hyperglycemia, low HDL cholesterol levels, and elevated triglyceride levels.[102]
        2. Metabolic syndrome has a greater prevalence reported among older populations.[98] However, as the obesity rate increases among the pediatric population, recent reports indicate that metabolic syndrome affects a significant number of children and adolescents across most ethnic groups.[99,103,104] In the United States alone, metabolic syndrome is far more common among children and adolescents than previously reported, and there is a direct correlation between metabolic syndrome and obesity. Among the adult population in the United States, 1 in 3 American adults were estimated to suffer from metabolic syndrome.[105]
2. **Metabolic Syndrome and Periodontitis**
    a. **Impact of Metabolic Syndrome on the Periodontium**
        1. Metabolic syndrome and abdominal obesity are considered to be risk factors for periodontitis.[7,106–113] Having three or more components of metabolic syndrome is significantly associated with a higher risk of periodontitis.[114]
        2. The apparent association between metabolic syndrome and periodontitis may be explained by shared common risk factors, such as obesity, diet, and inadequate self-care.[111] Obesity is associated with increased levels and proportions of periodontal pathogens, especially in patients with periodontitis.[115]
        3. Metabolic syndrome may influence the progression of periodontitis by contributing to chronic low-grade inflammation of prolonged duration.[116,117]
            a. Low-grade chronic inflammation is postulated to be due to the sustained actions of the adipocytes (fat cells). Previously, adipocytes were thought to be dormant, inactive cells.
            b. However, recent research has shed light that adipocytes are very active cells that produce various types of adipokines (cytokines produced by adipocytes).
        4. In obese patients, the increased numbers of adipocytes lead to greater release of adipokines, which causes a sustained inflammatory state, insulin resistance, and increased susceptibility to periodontal disease[118] (**Figure 14-13**).
    b. **Metabolic Syndrome: Implications for the Dental Hygienist**
        1. Results from a recent randomized controlled trial indicate that periodontal therapy in individuals with metabolic syndrome leads to a significant improvement in periodontal health.[118]
        2. Management of metabolic syndrome is based on lifestyle modifications, including increased physical activity and dietary modifications. Dental hygienists are well positioned to provide nutritional counseling for individuals exhibiting signs of metabolic syndrome.

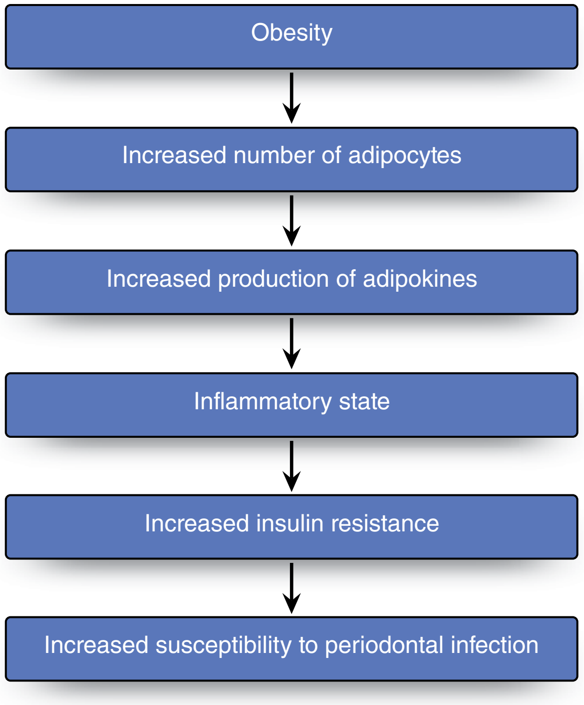
Obesity
Increased number of adipocytes
Increased production of adipokines
Inflammatory state
Increased insulin resistance
Increased susceptibility to periodontal infection

Figure 14-13 Relationship of Metabolic Syndrome and Periodontal Disease. In individuals who are obese, the increased numbers of adipocytes lead to greater release of adipokines, which causes a sustained inflammatory state, insulin resistance, and increased susceptibility to periodontal disease. Hypertension associated with metabolic syndrome may exacerbate periodontitis due to impaired blood flow to the periodontium.[118]

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HIV/AIDS

1. **Characteristics of HIV Infection**
   a. **Acquired immunodeficiency syndrome (AIDS)** is a communicable disease caused by the **human immunodeficiency virus (HIV)**. HIV-infected individuals are at an increased risk for developing certain cancers and for infections that usually occur only in individuals with a compromised immune system.
      1. In most developed countries, the death rate from AIDS among adults has declined largely because of newer antiretroviral therapies and improved access to these therapies.[119,120]
      2. Nevertheless, dental and medical practitioners will need to maintain strict adherence to infection control guidelines while treating oral and periodontal conditions in HIV-positive adults and children.[121]

   b. HIV infection has a profound effect on cellular immunity (T-lymphocytes, B-cells, monocyte/macrophage function). Decreased monocyte chemotaxis and phagocytosis, together with increased production of TNF-α and IL-1, and diminished capacity to present antigens to T-cells explain the increased periodontal attachment loss found in HIV-positive individuals.[122–125]

   c. Studies of healing following tooth extraction in HIV-positive patients indicate that there is a delayed healing response in both the hard and soft tissues.[126]

2. **Periodontal and Oral Manifestations of HIV Infection**
   a. HIV-related oral manifestations include hairy leukoplakia, candidiasis, herpes labialis, recurrent intraoral herpes simplex, herpes zoster, recurrent aphthous ulcers, and Kaposi sarcoma.

b. Studies suggest that gingival recession and alveolar bone loss are more frequent in HIV-positive patients.[127–130]
   1. The effect of HIV infection on the progression of periodontitis is not clear. A recent 7-year study found no difference in the progressions of periodontitis between HIV-positive and HIV-negative women.[131]
   2. Periodontal diseases strongly associated with HIV-infection include linear gingival erythema, necrotizing periodontal diseases, and periodontitis.[119,132]

c. Necrotizing periodontal diseases are discussed in Chapter 9.

d. **Linear Gingival Erythema**
   1. Gingival manifestations of HIV infection were formerly known as HIV-associated gingivitis but currently are designated as **linear gingival erythema (LGE)**. LGE is characterized by a 2- to 3-mm marginal band of intense erythema (redness) in the free gingiva (**Figure 14-14**). LGE is not associated with pocketing and does not affect clinical attachment levels or alveolar bone levels.

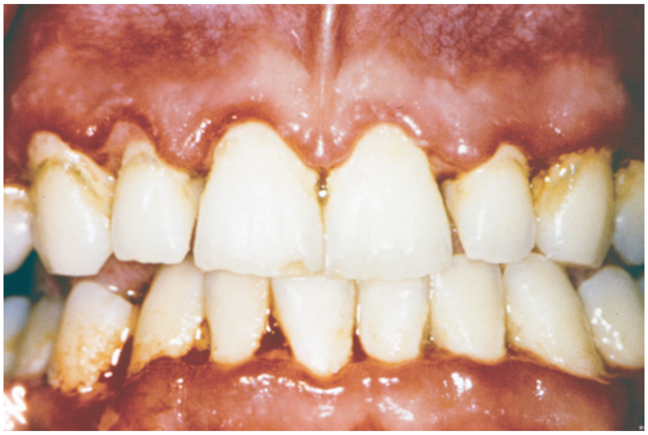

Figure 14-14 Linear Gingival Erythema. Gingival changes associated with linear gingival erythema. Note the marginal band of intense erythema in the free gingiva.

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   2. The band of gingival erythema may extend into the attached gingiva and/or extend beyond the mucogingival line into the alveolar mucosa.[134] LGE may be localized to one or two teeth but is more commonly a generalized gingival condition.
   3. The intensity of LGE inflammation is exaggerated in relation to the amount of plaque biofilm present. A lack of response of LGE to conventional self-care and periodontal therapy is an important criterion in its diagnosis.[134]
   4. The etiology of LGE is not well understood. Research suggests that organisms not generally associated with gingivitis, such as *Candida* species, are associated with linear gingival erythema.[133,135]
   5. With the advent of antiretroviral therapy for HIV-positive patients, the prevalence of HIV-specific lesions has been dramatically reduced.[13,109,136,137]

3. **HIV-Infected Individuals: Implications for the Dental Hygienis**
   a. Evidence suggests that it is safe to perform periodontal therapy in HIV-infected patients who have an undetectable viral level and high CD4+ count (> 500 cells/mm^3). Even surgical therapy is permitted as long as the individual's immune system is competent.[138]
      1. The initial treatment for LGE should be standard periodontal therapy plus the use of 0.12% chlorhexidine gluconate as a mouth rinse. Consideration may be given to the use of antibiotics.[134]
      2. HIV-infected individuals should be monitored to prevent irreversible periodontal damage.
      3. The need for early periodontal therapy and more frequent maintenance visits should be stressed. Continuity of dental care remains important for HIV+ patients even when they are being treated with antiretroviral therapies.[137,139,140]

        4. Dental health care providers should emphasize the importance of careful self-care at home and frequent professional care visits.[139]
    b. Care should be coordinated with other health professionals. Interprofessional collaboration among professionals such as physicians, dental professionals, social workers, dieticians, and case managers is an essential component of patient-centered care.[141,142] To assess for a patient's HIV status, consultation with the patient's physician (or nurse practitioner) requesting the most up-to-date CD4+ count (the number of functioning T-helper cells) and viral level count should be performed.
    c. Despite the beneficial effects of antiretroviral therapies, drug interactions with other medications have been observed. For instance, fluconazole, ketoconazole, itraconazole, metronidazole, ciprofloxacin, midazolam, and triazolam can interact with some antiretroviral medications, such as zidovudine, nevirapine, and ritonavir. Dental professionals need an understanding of possible drug interactions in HIV-infected individuals that can occur during the course and duration of dental treatment.[119]
    d. Strict adherence to infection control is strongly recommended by the American Dental Association (ADA) and the Centers for Disease Control and Prevention (CDC). This will minimize the risk to both dental staff and immunocompromised patients who are not only at risk of transmitting the disease, but also at risk of acquiring an infection from a health care setting.

Neutropenia

1. **Characteristics of Neutropenia.** Polymorphonuclear neutrophils (PMNs) are a type of white blood cell important to fighting off infections—particularly those caused by bacteria. **Neutropenia** is a disorder characterized by the presence of abnormally few numbers of neutrophils in the blood, leading to increased susceptibility to infection. A diagnosis of neutropenia is made when the absolute neutrophil count falls below 1,500 cells/μL of blood.
    a. Neutropenias may be the result of bone marrow failure and/or accelerated destruction of PMNs.
    b. Neutropenias may be congenital—such as cyclic neutropenia—associated with leukemia, drug-induced, radiation-induced, or idiopathic (of unknown cause).
2. **Neutropenia and Periodontitis**
    a. Because neutrophils are the first responders the immune system deploys to fight off microbial invasion, individuals with neutropenia are more susceptible to periodontal diseases compared to non-neutropenic, healthy individuals.[143]
    b. Higher plaque scores, gingival bleeding, gingival inflammation, and alveolar bone loss are reported in individuals with neutropenia.[143–146] **Figure 14-15** shows a patient with cyclic neutropenia who exhibits pronounced gingival inflammation.

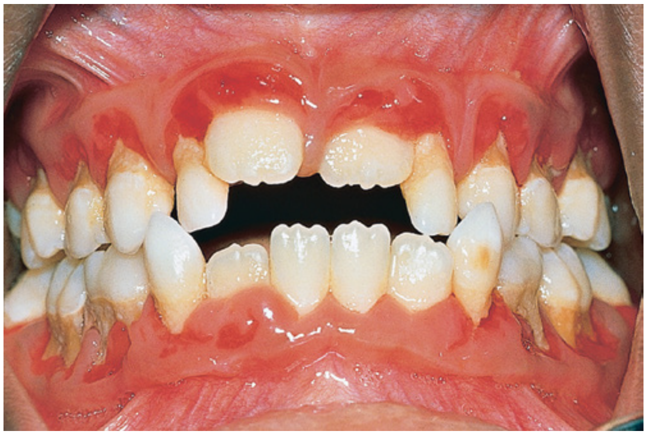

Figure 14-15 Cyclic Neutropenia. This individual with cyclic neutropenia exhibits pronounced gingival erythema.

Used with permission from Langlais RP. *Color Atlas of Common Oral Diseases*. Wolters Kluwer; 2003. © Wolters Kluwer.

3. **Neutropenia: Implications for the Dental Hygienist**
    a. Prevention of oral infections is important in reducing the risk of systemic bacteremia and septicemia, as well as maintaining the patient's dentition for quality of life.[144,147,148]
        1. Periodontal infections may induce fever and systemic dissemination of microorganisms. Hygienists should educate patients with neutropenia about the importance of meticulous self-care and frequent professional care for the maintenance of systemic and oral health.
        2. Chlorhexidine gluconate mouthwash (0.12%) has been shown to reduce the severity of oral mucosal inflammation and decrease plaque levels.[149–152]
    b. Acute periodontal exacerbations should be treated by removal of local causative factors by gentle periodontal instrumentation, chlorhexidine mouthwash rinse, and/or irrigation.[153]
    c. Patients with neutropenia are considered to be immunocompromised. Therefore, strict adherence to infection control is strongly recommended to reduce the risk of neutropenic patients acquiring a disease from a health care setting.

Down Syndrome

1. **Characteristics of Down Syndrome.** **Down syndrome** is a genetic disorder caused by a gene problem that happens before birth. Down syndrome is a lifelong condition, which varies in severity, causing intellectual disability and developmental delays, and in some people, health problems.
    a. **Genetic Changes in Down Syndrome**
        1. Normally, the nucleus of each cell contains 46 chromosomes. In Down syndrome, however, the nucleus contains 47 chromosomes. Most cases of Down syndrome occur because there are three copies of chromosome 21. For this reason, Down syndrome is also referred to as trisomy 21.
        2. Due to advances in medical treatment, individuals with Down syndrome are living longer. As the mortality rate associated with Down syndrome decreases, the prevalence of adults with Down syndrome in our society will increase. More and more dental health care providers will interact with individuals with this condition, increasing the need for education and acceptance.
    b. **Orofacial Features Characteristic of People with Down Syndrome.** Among the most common orofacial traits of individuals with Down syndrome are:
        1. An underdeveloped midfacial region, affecting the appearance of the lips, tongue, and palate.
            a. The maxilla, the bridge of the nose, and the bones of the midface region are smaller than in the general population, creating a prognathic occlusal relationship. Mouth breathing may occur because of smaller nasal passages, and the tongue may protrude because of a smaller midface region. People with Down syndrome often have a strong gag reflex due to placement of the tongue, as well as anxiety associated with any oral stimulation.
            b. The palate, although normal sized, may appear highly vaulted and narrow. This deceiving appearance is due to the unusual thickness of the sides of the hard palate. This thickness restricts the amount of space the tongue can occupy in the mouth and affects the ability to speak and chew.
            c. The lips may grow large and thick. Fissured lips may result from chronic mouth breathing. Additionally, decreased muscle tone may cause the mouth to droop and the lower lip to protrude. Increased drooling, compounded by a chronically open mouth, contributes to angular cheilitis.
            d. The tongue also develops cracks and fissures with age; this condition can contribute to halitosis.
        2. Malocclusion is found in most people with Down syndrome because of the delayed eruption of permanent teeth and underdevelopment of the maxilla. A smaller maxilla contributes to an open bite, leading to poor positioning of teeth and increasing the likelihood of periodontal disease and dental caries.
    c. **Medical and Developmental Problems of Patients with Down Syndrome**
        1. Children are at increased risk for congenital heart defects, susceptibility to infection, respiratory problems, gastrointestinal abnormalities, and childhood leukemia.
        2. Abnormal PMN function is seen in about half of all patients with Down syndrome.
        3. Most individuals with Down syndrome have IQs in the mild to moderate range of mental retardation. Those who receive good medical care and experience a supportive social environment can attend school, hold jobs, and participate in decisions that affect them (**Figure 14-16**).

Figure 14-16 Individuals with Down Syndrome in the Workforce. With appropriate training and support, people with Down syndrome can and do make a huge contribution to their workplace.

© Valeria Blanc/E+/Getty Images

2. **Down Syndrome and Periodontitis.** Down syndrome is one of the most common birth defects. Though rare genetic syndromes are unlikely to be encountered by the dental hygienist outside a hospital setting, individuals with Down syndrome are frequently treated by all members of the dental team in general and periodontal dental offices.
    a. It is widely known that individuals with Down syndrome often exhibit severe and rapid periodontal breakdown.
        1. The prevalence of periodontal disease ranges from 58% to 96% of young adults younger than 35 years with Down syndrome.[154]
        2. Substantial plaque biofilm formation, deep periodontal pockets, and extensive gingival inflammation characterize periodontal disease in Down syndrome (**Figures 14-17** and **14-18**).[155]

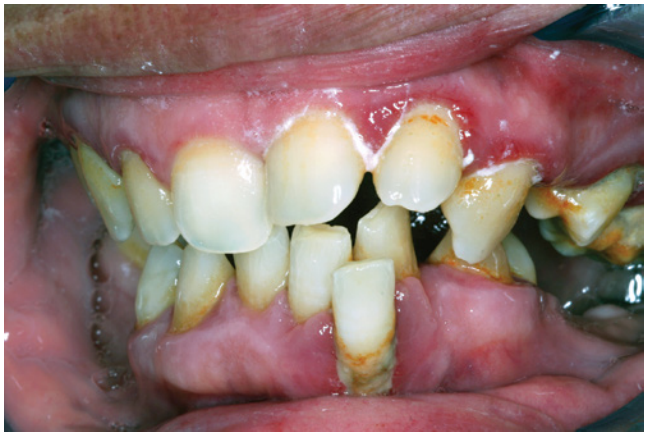

Figure 14-17 Periodontitis and Down Syndrome. A 25-year-old patient with Down syndrome exhibits severe periodontal destruction.

Courtesy of Dr. Richard J. Foster, Educational Solutions and Design, Greensboro, NC.

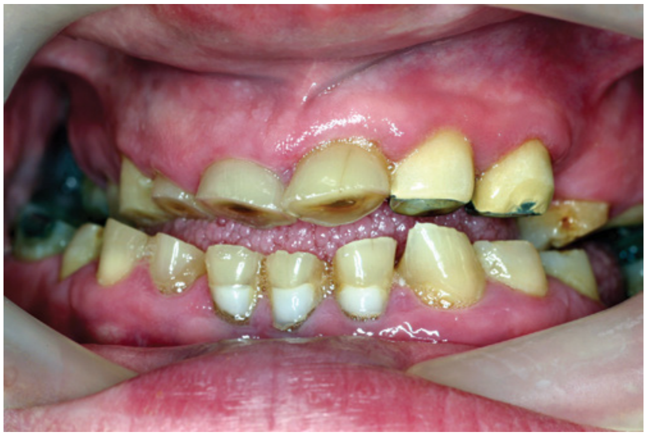

Figure 14-18 Periodontitis and Down Syndrome. This patient with Down syndrome exhibits pronounced attrition and localized loss of attachment.

Courtesy of Dr. Richard J. Foster, Educational Solutions and Design, Greensboro, NC.

    b. Children with Down syndrome experience rapid, destructive periodontal disease. Consequently, large numbers of them lose their permanent anterior teeth in their early teens.
        1. Some children with Down syndrome are congenitally missing at least one salivary gland.[156]
        2. Studies indicate that various periodontal pathogens colonize the gingival tissues in the very early childhood years of children with Down syndrome.[157]

c. The etiology of periodontal disease in persons with Down syndrome is complex. The prevalence of periodontal disease cannot simply be attributed to poor daily self-care. In recent years, much focus has been placed on the altered immune response resulting from the underlying genetic disorder.[154,158]
   1. Impaired PMN chemotaxis and phagocytosis most likely explain the high prevalence and increased severity of periodontitis associated with Down syndrome.
   2. Impaired cellular motility of gingival fibroblasts that prevents wound healing and regeneration of periodontal tissues may be involved in the etiology of Down syndrome periodontitis.[159]

3. **Down Syndrome: Implications for the Dental Hygienist**
   a. Early and frequent professional treatment and meticulous daily care at home can mitigate the severity of periodontal disease in individuals with Down syndrome.
      1. Some people with Down syndrome can brush and floss independently, but many need help from caregivers.
      2. Encourage independence in daily self-care in those individuals who are capable on their own. Involve patients in hands-on demonstrations of brushing and interdental cleaning aids.
   b. Hygienists should educate caregivers about daily self-care. The dental health care provider should demonstrate proper home care techniques to both the patient and the caregiver. A power toothbrush and power water flossing device can simplify oral care.
      1. The hygienist can demonstrate techniques to caregivers on techniques to access the oral cavity, such as having the person close slightly for improved access to the posterior teeth and where to sit or stand to gain easier access to different areas of the dentition.
      2. The hygienist should emphasize to the caregiver the importance of establishing a daily routine for oral care.

Leukemia

1. **Characteristics of Leukemia**
   a. **Leukemia** is a rapid cancer of the blood cells that usually begins in the bone marrow.
      1. Most often, it is a cancer of white blood cells, but it can be a cancer of other types of cells, such as red blood cells and platelets.
      2. In people with leukemia, the bone marrow rapidly produces many abnormally functioning cells.
         a. At first, leukemia cells function almost normally. In time, however, they may spread throughout the body (such as the lymph nodes, liver, and spleen) and crowd out normal-functioning white blood cells, red blood cells, and platelets.
         b. The increase in number of leukemic cells in the blood circulation leads to several serious systemic disorders: leukopenia (a decrease in the number of normal functioning white blood cells), thrombocytopenia (a decrease in the number of normal-functioning platelets), and anemia (a decrease in the number of normal-functioning red blood cells).
      3. Leukemia is a disease of both children and adults and is more common in men and boys than girls and women.
   b. **Types of Leukemia**
      1. Leukemia is classified on the duration (acute or chronic) and the type of cell involved (myeloid or lymphoid).
         a. Leukemia is either chronic (gets worse slowly) or acute (gets worse quickly and is rapidly fatal).
         b. Leukemia that affects the lymphocytes is called lymphocytic while leukemia that affects the myeloid cells (such as red blood cells, platelets, and neutrophils) is called myelogenous leukemia.
      2. Leukemia is the most common cancer in children younger than 15 years of age.[160]
   c. **Medical Treatment of Leukemia**
      1. Treatment for leukemia is complex and is not the same for all patients. Treatment varies with the type of leukemia, extent of the disease, and the patient's age, symptoms, and general health. The physician tailors the treatment to fit each individual patient's needs.
      2. Most patients with leukemia are treated with chemotherapy. Some also may have radiation therapy and/or bone marrow transplantation or biological therapy.

3. Chemotherapy causes patients to suffer from severe suppression of the immune system. Chemotherapy functions by suppressing the growth and spread of malignant cells; unfortunately, normal cells are also adversely affected. Normal cells with the highest rate of cell turnover (proliferation)—such as those of the periodontium—are affected because chemotherapy interferes with cell production, maturation, and replacement.
4. Dental care is a vital component of treatment. Anticancer treatments for leukemia can make the mouth sensitive, easily infected, and likely to bleed.[148,152,160–163]

2. **Oral Complications of Leukemia**
   a. **Leukemia-Associated Gingivitis**
      1. **Inflammation of the Gingiva.** Signs of gingival inflammation in the leukemic patient include swollen, glazed, and spongy tissues that are red to deep purple in appearance (**Figure 14-19**) and bleed with the slightest provocation or even spontaneously. Leukemic patients exhibit profuse gingival bleeding because of a reduction in the number of normal functioning platelets (thrombocytopenia).

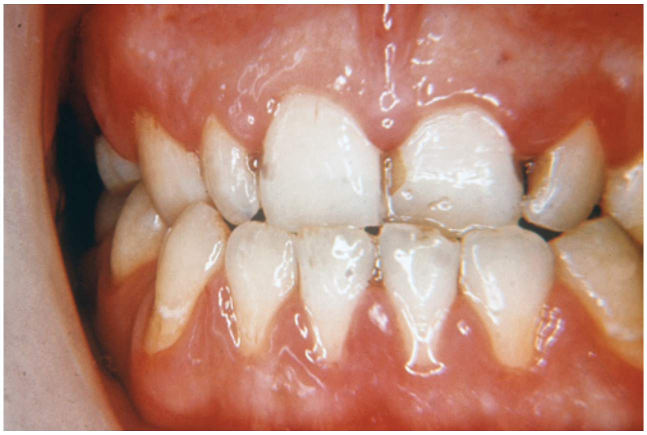

Figure 14-19 **Leukemia-Associated Gingivitis.** Note the swollen, red gingival tissues in this patient with leukemia.

Courtesy of Dr. Ralph Arnold, San Antonio, TX.

         a. In a study of 1,093 adult in-patients undergoing chemotherapy treatment for leukemia, 14.9% of patients manifested gross bleeding from the mouth during the course of chemotherapy.[164,165] The most common oral bleeding sites were the lips, tongue, and gingiva.
         b. In children, the prevalence of gingival inflammation is highest in the maintenance phase of chemotherapy followed by the induction phase with radiotherapy.[161]
      2. **Gingival Enlargement.** Gingival enlargement is a common characteristic, initially beginning at the interdental papilla followed by marginal and attached gingiva. This enlargement is due to increased infiltration of immature leukemic cells in the gingiva. Additionally, as the gingiva enlarges, a periodontal pocket is created that will harbor more pathogenic microorganisms and worsen the inflammation.
      3. **Oral Infection.** Compared to healthy, nonleukemic individuals, leukemic patients are more susceptible to oral infection due to a reduction in the number of normal functioning white blood cells (leukopenia). As a result, leukemic patients have an impaired immune system.
   b. **Oral Mucositis as an Oral Complications of Leukemia Therapy**
      1. **Oral mucositis** is an inflammation of the oral mucous membranes caused when chemotherapy attacks and kills the rapidly dividing cells of the mucous membranes. Cells of the oral mucosa have a lifespan of only 10 to 14 days. Therefore, during chemotherapy, mucosal cells are dying at a faster rate than new cells can be produced.
      2. Sloughing of mucosal surfaces can be localized or generalized involving the buccal mucosa, palate, floor of the mouth, gingiva, lips, and/or tongue (**Figure 14-20**). Ulcerations of the oral cavity also are a common complication of chemotherapy.

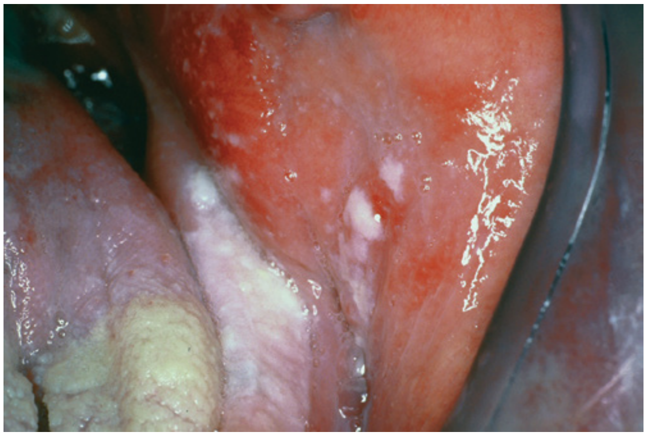

Figure 14-20 Mucositis Associated With Chemotherapy. Oral mucositis directly attributable to anti-leukemia chemotherapy occurs in almost 20% of adults undergoing such treatment. The associated extreme discomfort may produce physical and psychological obstructions to continued anticancer treatment.

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3. Although oral mucositis is an undesired side effect of leukemic therapy, this is not a reason for a leukemic patient to avoid chemotherapy. Instead, while on chemotherapy, the oral condition of the leukemic patient should be closely monitored by a dental professional for signs of oral mucositis. Early detection and management of oral mucositis in a leukemic patient is critical in reducing the severity of the tissue sloughing and relieving the pain associated with this complication.

c. **Xerostomia as an Oral Complications of Leukemia Therapy**
   1. Xerostomia may occur due to damage of the salivary glands during radiation therapy. A reduction in salivary flow alters the self-cleaning mechanisms of the oral cavity, resulting in rapid biofilm accumulation and the development of dental caries.
   2. Lack of saliva also can result in diminished taste perception and/or difficulty in swallowing and talking.
   3. Reduced salivary flow and xerostomia can encourage the growth of *C. albicans* and the development of oral candidiasis.[166]
   4. Although xerostomia is an undesired side effect of leukemic therapy, this should not be a reason for a leukemic patient to avoid therapy. Instead, while on radiation therapy, the oral condition of the leukemic patient should be closely monitored by a dental professional for signs of xerostomia. Early detection and management of xerostomia in a leukemic patient is critical in reducing the severity of this complication.

3. **Leukemia: Implications for the Dental Hygienist**
   a. The dental team can act as an important point of contact for early screening and diagnosis of undiagnosed leukemia. In some individuals, the first signs of leukemia show up in the oral cavity.[160,167]
      1. When a dental patient has spontaneous gingival bleeding and/or gingival enlargement for no apparent reason—combined with symptoms such as facial swelling, tiredness, poor appetite, lethargy, musculoskeletal pain—the individual should be referred to an oncologist.[160,167]
      2. The fact that leukemia frequently presents with early oral manifestations emphasizes the need for dental professionals to be aware of the early oral signs of leukemia and can provide a timely referral. Timely referral to an oncologist is critical.
   b. Frequent unfavorable oral conditions of individuals undergoing anticancer therapy for leukemia highlights the responsibility of the otolaryngologist and oncologist to refer these patients to the dental office.[161,168,169] The planning of anticancer therapy for leukemia should include dental professionals in the multidisciplinary oncology team.
      1. Immune suppression during chemotherapy may cause serious oral infections.
      2. Adequate oral care before, during, and after chemotherapy is necessary to prevent oral diseases and systemic complications of oral origin. Frequent dental care is essential for improvement in oral conditions

that may diminish patient suffering and prevent the spread of serious infections from the oral cavity to other parts of the body.[161]

3. Chemotherapy can cause a sore and sensitive mouth that bleeds easily. Soft toothbrushes with gentle brushing should be recommended. If the mouth is too sensitive to tolerate tooth brushing, soft dental sponges (available from a pharmacy) can be recommended.

c. Pain from oral mucositis afflicts from 40% to 70% of patients receiving chemotherapy or radiation therapy.
  1. Current methods of clinical pain management (e.g., topical anesthetics, systemic analgesics) have limited success.[170] Oral mucositis is common in children undergoing chemotherapy.
  2. In 2011, a study by Soares et al. recommends that the prophylactic use of 0.12% chlorhexidine gluconate reduces the frequency of oral mucositis and oral pathogens in children with leukemia.[152]

d. Care should be coordinated with other health professionals. Interprofessional collaboration among professionals such as physicians, dental professionals, dieticians, and case managers is an essential component of patient-centered care.[141]

Section 3

Systemic Medications with Periodontal Side Effects

Many medications used to treat systemic diseases can cause oral complications. Effects of medications can modify oral hygiene habits, plaque biofilm composition, size of gingival tissues, level of bone, and salivary flow. Educating patients about potential oral side effects is critical to reducing the medication-related risks of periodontal disease. Commonly prescribed medications that can affect the periodontium are summarized in **Table 14-1**.

Table 14-1 Harmful Effects of Commonly Prescribed Medications on Periodontium

Medication Class	Generic Name (Brand Name)	Effect on Periodontium
Anticonvulsant	Phenytoin (Dilantin®)	Gingival overgrowth
Antianxiety agents	Alprazolam (Xanax®)	Increased biofilm formation
Antihypertensive	Enalapril (Vasotec®)	Increased gingival inflammation
Calcium blocker	Nifedipine (Procardia®)	Gingival overgrowth
Immunosuppressive	Cyclosporine (Sandimmune®)	Gingival overgrowth

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1. **Medications That Alter Plaque Biofilm Composition, pH, or Salivary Flow**
  a. **Plaque Biofilm Composition or pH**
    1. Many oral medications alter plaque biofilm composition and pH in ways that are harmful to the periodontium.
    2. Sugar is a major component of some cough drops, liquid medications, cough syrups, tonics, chewable vitamins, antacid tablets, and other medications. Medications that contain sugar add significantly to the alteration of pH and composition of the biofilm.
    3. Sugar is metabolized by bacteria to form acid, causing enamel to demineralize. The demineralized areas are rough and act as attachment sites for bacteria, keeping bacterial plaque biofilm against tissues and eventually resulting in inflammation of the gingiva.
    4. Some over-the-counter (OTC) preparations contain sugar and vitamin C (ascorbic acid). This combination delivers sugar and may lower the pH.
    5. Products that alter the plaque biofilm pH significantly can cause root-surface caries in older adults and influence the metabolism of periodontal pathogens.[171,172]

b. **Salivary Flow and pH**
   1. Adequate saliva flow is necessary for the maintenance of healthy oral tissues. The ability of saliva to limit the growth of pathogens is a major determinant of systemic and oral health.
      a. The physical flow of the saliva helps to dislodge microbes from the teeth and mucosa surfaces. Saliva can also cause bacteria to clump together so that they can be swallowed before they become firmly attached.
      b. Saliva is rich in antimicrobial components. Certain molecules in saliva can directly kill or inhibit a variety of microbes.
   2. Patients with xerostomia suffer from an increase in the incidence of oral candidiasis, coronal and root-surface caries, as well as excess plaque biofilm formation.
   3. More than 400 OTC and prescription drugs have xerostomia as a possible side effect.[173]
   4. Some of the more common groups of medications that cause xerostomia are cardiovascular medications (blood pressure, diuretics, calcium channel blockers), antidepressants, sedatives; antiparkinsonism medications, allergy medications, and antacids.[174]

2. **Drug-Induced Gingival Enlargement**
   a. **Introduction**
      1. **Drug-induced gingival enlargement** is an esthetically disfiguring overgrowth of the gingiva that is a side effect associated with certain medications.
      2. Drugs associated with gingival enlargement can be broadly divided into three categories: anticonvulsants, calcium channel blockers, and immunosuppressants. These three classes of medications influence gingival fibroblasts to overproduce collagen matrix when stimulated by plaque-induced inflammation.[175]
         a. More than 20 medications have been shown to have the potential to induce gingival enlargement.
         b. The anticonvulsant phenytoin accounts for the highest prevalence rate (> 50%) of drug-associated gingival enlargement. Calcium channel blockers account for 6% to 15% of drug-associated gingival enlargements; immunosuppressants account for 25% to 30% of drug-associated gingival enlargements seen in adults.[176]
      3. The clinical characteristics of drug-induced gingival enlargement include painless enlargement of the keratinized gingiva with a tendency to occur more often in the anterior gingiva, a prevalence in younger age groups, an increased tendency to bleed, and an onset within 3 months of use.[177] Drug-induced gingival enlargement rarely affects the mucosal tissue and never appears in edentulous areas.
      4. Drug-induced gingival enlargement may make effective self-care more difficult due to the size and mass of enlarged gingival tissue. As a result, this may contribute to the retention of pathogenic microorganisms around the enlarged gingiva. Thus, reinforcement of good oral hygiene is critical in controlling plaque biofilm levels and reducing inflammation. However, good oral hygiene will not do anything to eliminate or lessen the gingival overgrowth that is induced by the medication.
   b. **Anticonvulsants**
      1. **Phenytoin** (FEN-i-toyn) is one of the most prescribed anticonvulsant medications used to control convulsions or seizures in the treatment of epilepsy. Phenytoin is marketed worldwide under various brand names, including Dilantin®, Phenytek®, Cerebyx®, and Phenytoin Sodium®. Phenytoin is among the 20 most-prescribed drugs in the world.
      2. Overgrowth of the gingiva is one of the most common side effects of phenytoin. It has been estimated that 40% to 50% of the millions of individuals who take phenytoin will develop gingival overgrowth to some extent.[176] Overgrowths appear to be more common in children and young adults.
      3. Gingival overgrowth begins with enlargement of the interdental papillae.
         a. The interdental papillae overgrow, forming firm triangular tissue masses that protrude from the interdental area.
         b. Gradually, the enlarged papilla from one interdental area may unite with the adjacent enlarged papilla to partially cover the anatomic crown with marginal gingiva (**Figure 14-21**).

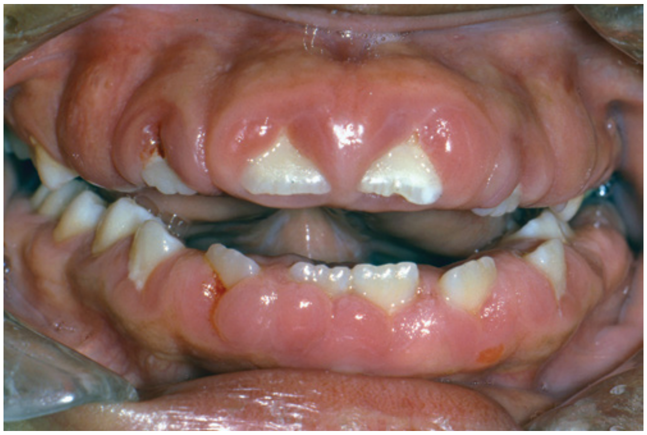

Figure 14-21 Phenytoin-Influenced Gingival Overgrowth. Severe enlargement of the gingiva associated with phenytoin (Dilantin) medication in an individual with epilepsy.

Courtesy of Dr. Ralph Arnold, San Antonio, TX.

c. Overgrowths most commonly affect the facial gingiva of the maxillary and mandibular anterior teeth. The gingival overgrowth may serve as plaque-retentive areas because of the difficulty in keeping the areas free of plaque.

d. In the presence of good biofilm control, the enlarged tissue is pink in color and firm and rubbery in consistency. In the presence of poor biofilm control, the tissue appears red, edematous, and spongy.

c. **Immunosuppressants**

1. **Cyclosporine** (SIGH-kloe-spor-een) belongs to the group of medicines known as immunosuppressive agents used for prevention of transplant rejection as well as for management of several autoimmune conditions such as rheumatoid arthritis.
2. The incidence of cyclosporine-associated gingival overgrowth affects approximately 25% to 30% of adult patients taking the medication. However, in children, the prevalence of cyclosporine-associated drug enlargement can be as much as 70%.
3. The clinical appearance of cyclosporine-associated gingival overgrowth resembles that of phenytoin-associated gingival enlargement (**Figure 14-22**).

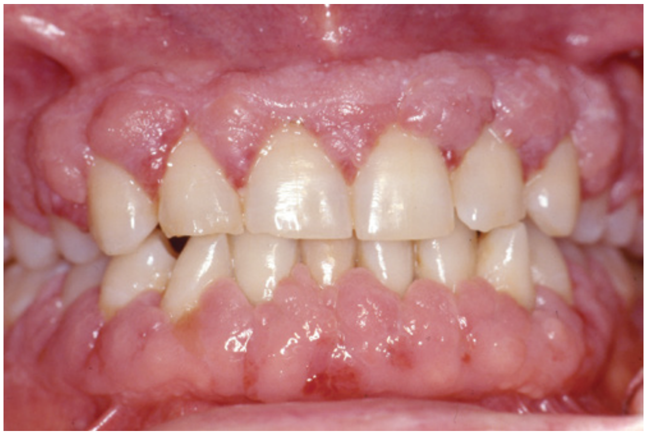

Figure 14-22 Cyclosporine-Influenced Gingival Overgrowth. The clinical appearance of cyclosporine-associated gingival overgrowth resembles that of phenytoin-associated gingival enlargement.

Courtesy of Dr. Ralph Arnold, San Antonio, TX.

d. **Calcium Channel Blockers**
   1. Antihypertensive drugs in the calcium channel blocker group are used extensively in elderly patients who have angina or peripheral vascular disease.
   2. The use of calcium channel blockers is associated with an increased risk of gingival hyperplasia.[178]
      a. **Nifedipine** (nye-FED-I-peen), one type of calcium channel blocker, is used as a coronary vasodilator in the treatment of hypertension, angina, and cardiac arrhythmias. Calcium channel blockers are a class of drugs that block the influx of calcium ions through cardiac and vascular smooth muscle cell membranes. This results in the dilation of the main coronary and systemic arteries.
      b. Various other calcium channel–blocking medications, such as diltiazem, felodipine, nitrendipine, and verapamil, also may induce gingival enlargement.
   3. The clinical appearance of gingival overgrowth associated with calcium channel blockers resembles that of phenytoin-associated gingival enlargement (**Figures 14-23** and **14-24**).

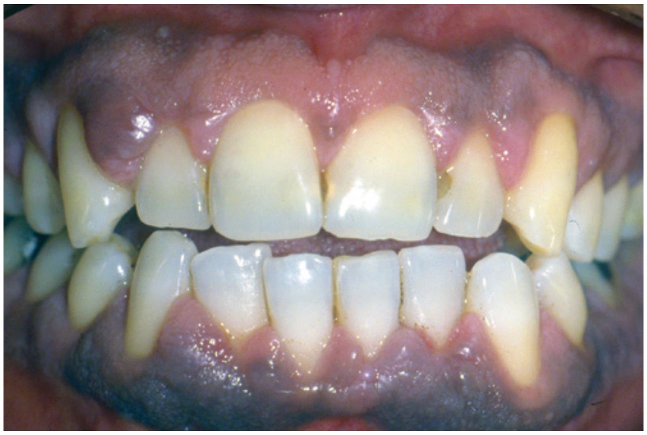

Figure 14-23 Gingival Overgrowth Associated with Nifedipine. Gingival overgrowth in a patient who takes Nifedipine for the treatment of cardiac arrhythmia.

Courtesy of Dr. Ralph Arnold, San Antonio, TX.

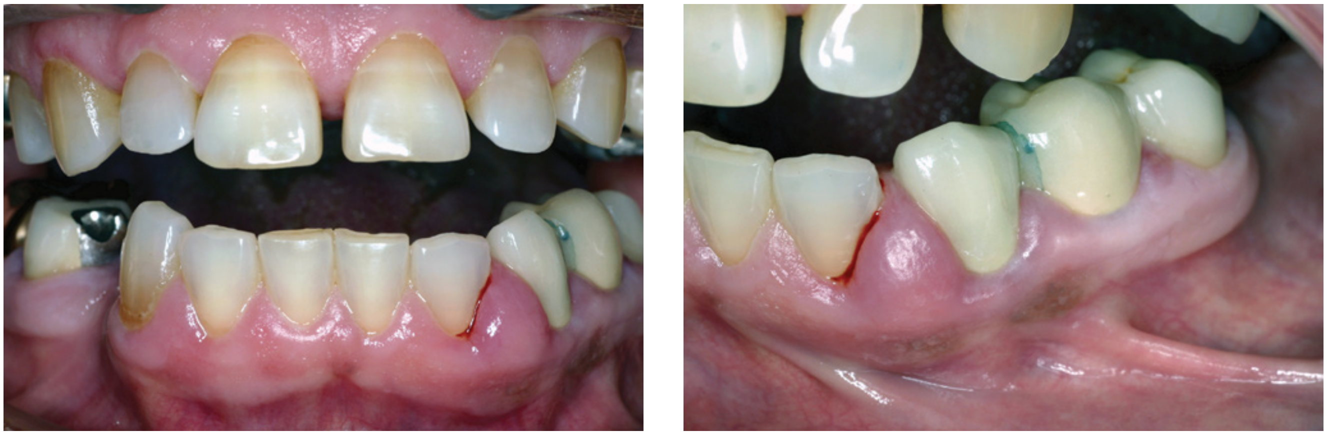

Figure 14-24 Gingival Enlargement Associated with Calcium Channel Blocking Drugs. Gingival enlargement of the papilla between the lateral incisor and canine induced by the calcium channel blocking medication Norvasc.

Courtesy of Dr. Richard J. Foster, Educational Solutions and Design, Greensboro, NC.

e. **Systemic Medications: Implications for the Dental Hygienist**
   1. Dental hygienists should be alert for patient medications that can alter biofilm composition, pH, or salivary flow.
      a. Sugar-containing liquid or chewable medications are sometimes used in the treatment of children with chronic medical problems. Parents should be made aware of the oral health consequences of such medications. Giving the medications at mealtimes instead of between meals is helpful.
      b. Some OTC preparations contain sugar and vitamin C. This combination delivers sugar and the vitamins cause an acid pH. Examples of products containing sugar and vitamin C include chewable vitamin C tablets, certain cough drops, and certain liquid cough preparations.
   2. Closer collaboration between medical and dental clinical teams is necessary for the joint management of individuals being treated with anticonvulsants, calcium channel blockers, or immunosuppressants.[141]
   3. Patients receiving cyclosporine are usually medically compromised, requiring close consultation with the patient's physician to ensure safe management of the patient's periodontal condition. In addition to professional and at-home plaque biofilm control, it has been shown that azithromycin induced a striking reduction in cyclosporine-induced gingival hyperplasia.[179]
   4. Treatment of gingival enlargement should include consultation with the physician, substitution of the current medication for another whenever possible, nonsurgical periodontal therapy, frequent periodontal maintenance, and surgical therapy, if needed.[178]
      a. The hygienist should emphasize the importance of meticulous daily self-care and involve patients in hands-on demonstrations of brushing and interdental cleaning aids.
      b. Surgical elimination of the tissue overgrowth is often required. If plaque biofilm control is inadequate, regrowth will occur rapidly. The patient should be advised of the likelihood of the recurrence of the gingival overgrowth following surgery.

Chapter Summary Statement

All the systemic conditions discussed in this chapter share the commonality of being inflammation-related disorders that dysregulate the host immune response and ultimately increase the susceptibility of certain individuals to oral diseases, such as periodontitis. In many individuals, the presence of multiple, overlapping, simultaneous-occurring factors can exacerbate inflammation, which can lead to a more accentuated and more widespread systemic effect. Often overlooked are the underappreciated connections between many of these systemic conditions and periodontitis, which exacerbate one another when occurring simultaneously. When put together, a perfect storm that combines an underlying debilitating systemic condition(s) and an existing oral infection (such as periodontitis) can blow systemic health and oral health way off-course, thereby creating the conditions ripe for the hyperinflammatory state.

Several systemic conditions can impact periodontal health, and this knowledge is of utmost importance for all members of the dental team. Some of these systemic contributing factors that influence the periodontium include diabetes mellitus, leukemia, HIV/AIDS, hormonal fluctuations, genetic risk factors, and systemic medications. All these conditions are highly prevalent, and their periodontal manifestations can lead to early recognition and treatment of the underlying systemic condition. Thus, dental professionals should provide health promotion education that contributes to overall systemic and periodontal health and collaborate with medical team members for joint patient management.

Worldwide, there are numerous therapeutic medications that have the potential to cause different oral side-effects, such as oral mucositis, gingival inflammation, and decreased salivary flow. With newer medications being approved worldwide by drug regulatory agencies, including the U.S. Food and Drug Administration and the Canadian Therapeutic Products Directorate, keeping up to date with the latest in pharmacology will be a necessary requirement for all members of the dental team.

Section 4

Focus on Patients

Clinical Patient Care

CASE 1

A patient, who has been previously treated for periodontitis and has been followed by your dental team for several years, calls your dental office with a concern. This individual is scheduled to undergo a liver transplant and has been warned by her physician that the medications she will need will make her more susceptible to infections. She asks if these medications might modify her continuing treatment for periodontitis. How might you respond to her concern?

CASE 2

The parents of a young patient currently being treated by your dental team inform you that, following a lengthy illness, their daughter has recently been diagnosed by her physician with a neutrophil defect. Neutrophils are also known as polymorphonuclear leukocytes. They inquire about any dental implications of this diagnosis. How might you respond to this inquiry?

Evidence in Action

Case 1: A new patient in your office reports that she has recently been diagnosed with diabetes mellitus and that her physician suggested that she should have a dental checkup. The patient confides in you that she feels like this disease is really changing her lifestyle. She laughingly says, "I have always had such good reports from my previous dentist, and I just don't really see why I need to be worried about my teeth now." Based on what is known about the relationship between diabetes and periodontal disease, how might you explain the need for the recommended dental exam to the patient?

Case 2: James is a 10-year-old patient who is new to your dental office. James's family just relocated to your city for his father's new job. James' health history indicates a history of epilepsy. He takes phenytoin (Dilantin®) for seizure control. You note gingival enlargement and bleeding on probing during the intraoral examination. Based on what you know about the relationship between phenytoin and gingival enlargement, what information would you provide to James and his parent? Would a consultation with James' physician be indicated?

Ethical Dilemma

You have just recently married and moved across the country to the city where your husband was raised. His parents helped you secure a full-time dental hygiene position with their family dentist, Dr. Ramos. You are quite happy working with the office staff, as well as meeting and getting to know many of your in-law's family and friends, who are patients of the practice as well.

Today your mother-in-law, June, is scheduled for her 6-month recall appointment. This is the first time you will be treating her. You review her medical history, and she states that lately she's been feeling tired, lethargic, and has a poor appetite. She assumed she was just overtired from all the wedding planning and festivities. She hasn't been able to work out regularly, as she has musculoskeletal aches and pains.

Your clinical examination reveals gingival enlargement and spontaneous gingival bleeding, despite excellent self-care. Her tissues are swollen, glazed, and spongy, and red to deep purple in appearance, and ooze blood intermittently. She also appears to have facial swelling. You review the notes from her last appointment, which was 6 months ago, and see that her tissues were classified as normal and healthy.

You become very concerned that June may have a serious medical condition, and you are not sure what to do. You feel that you need to discuss this with your husband before saying anything to June.

1. What systemic condition do you think could be causing June's signs/symptoms?
2. What ethical principles are in conflict in this dilemma?
3. What is the best way for you to handle this ethical dilemma?

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CHAPTER 15

Impact of Periodontal Inflammation on Systemic Health

- Section 1 Linking Periodontitis with Systemic Disease
- Section 2 The Role of Periodontitis on Systemic Diseases
  - Atherosclerotic Cardiovascular Disease
  - Adverse Pregnancy Outcomes
  - Diabetes Mellitus
  - Potential Associations with Other Systemic Conditions
- Section 3 Focus on Patients
  - Clinical Patient Care
  - Evidence in Action

CLINICAL APPLICATION

Over the last several decades, an important bidirectional relationship between periodontal disease and certain systemic diseases has been uncovered, bridging the once-wide gap between medicine and dentistry. On the one side of this relationship (as reviewed in Chapter 14), certain known systemic factors can potentially intensify the progression and severity of periodontal disease. On the other side of this relationship, periodontitis can be a potential contributing factor to the pathogenesis of certain systemic diseases. The possibility of an association between periodontal disease and systemic disease suggests that periodontal therapy may play an important role in decreasing the incidence and severity of certain systemic diseases. This possible association will be the focus of this chapter. Interprofessional relationships between dental team members and other health care providers should be established early to provide the highest standard of care for patients with systemic disease.

LEARNING OBJECTIVES

- Contrast the terms *association* and *causation* between a given factor (A) and a systemic disease (B).
- Educate patients at risk for cardiovascular diseases about the possible impact of periodontal infection on cardiovascular health and be able to motivate the patient to seek oral disease prevention/counseling service.
- Educate pregnant women and those planning pregnancies about the possible impact of periodontal infection on pregnancy outcomes and be able to motivate the patient to seek oral disease prevention/counseling service.
- Educate patients with diabetes about the probable bidirectional association between periodontal disease and diabetes and be able to motivate the patient to seek oral disease prevention/counseling services

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- Educate family members, caregivers, and affected patients about the association between periodontal disease and pneumonia in health-compromised individuals in hospitals and long-term care facilities.
- Establish collaborative relationships with other health care providers to ensure the highest standard of care for periodontal patients with systemic diseases and conditions.

KEY TERMS

Association
Causation
Metastatic infection
Atherosclerosis
C-reactive protein
Preeclampsia
Glycemic control
HbA1C level
Community-acquired pneumonia
Hospital-acquired pneumonia
Ventilator-associated pneumonia

Section 1

Linking Periodontitis with Systemic Disease

It is well known that oral health is closely linked to systemic health. Equally important is that the presence of certain oral infections, particularly periodontitis, have the potential to drastically alter the course and pathogenesis of a number of systemic diseases/conditions, such as cardiovascular disease, bacterial pneumonia, diabetes mellitus, and adverse pregnancy outcomes.

Although the association between oral health and systemic health has been well studied, no scientific evidence at this time suggests a causal relationship between the two. When reading and interpreting research, great care must be taken to understand exactly what the data and statistics are implying—and more important—what they are not implying. The terms *association* and *causality* are two important concepts relevant to medical and dental research. For example, when researchers find an association between a given factor (A) and a health effect (B), this relationship does not necessarily imply that factor A causes disease B. It is important for health care providers to understand the difference between association and causation and to explain to patients how evidence-based research applies to their health.

1. **Association Versus Causal Relationships.** Theoretically, the difference between an association and a causal relationship is easy to distinguish—variable A can cause variable B (e.g., smoking causes cancer), or variable A is associated with variable B (e.g., smoking is associated with alcoholism, but smoking does not cause alcoholism).
    a. **Association.** **Association** indicates that there is a relationship or a connection between two or more variables (e.g., the rate of alcoholism is higher in smokers). An association cannot explain why or how the variables are related and does not necessarily mean that one variable causes the other variable. More research is necessary before that conclusion can be reached.
    b. **Causation.** **Causation** means that variable A is certain to cause or lead to variable B (e.g., the *Mycobacterium tuberculosis* bacterium causes tuberculosis). Determining causation of a disease or condition is difficult because diseases may be caused by a multitude of factors which makes establishing causation even more challenging as this often involves joint actions of several complex disease mechanisms.
        1. For example, we used to believe that stomach ulcers were caused by stress and spicy foods. Yet, current research proves that ulcers are caused by a corkscrew-shaped bacterium, *Helicobacter pylori*. Natural stomach acids and spicy food can irritate the already damaged stomach lining (association) but not have caused the ulcers (causation).
        2. Based on available research, it is not possible to prove causality between periodontitis and systemic disease.[1] In 2013, Working Group 4 of the Joint European Federation of Periodontology (EFP) and American Academy of Periodontology (AAP) Workshop on Periodontitis and Systemic Diseases was unanimous in its opinion that the reported associations of periodontitis and systemic diseases do not imply causality.[2] The working group further concluded that gaps in knowledge are large. It is possible that the association is the result of common risk factors and not causality.
2. **Possible Mechanisms Linking Periodontitis to Systemic Disease.** The best evidence suggests that periodontitis is characterized by both infection and proinflammatory events and may contribute toward select

systemic diseases and disorders.[2] Three mechanisms have been postulated as to how periodontitis may modify some aspect of certain systemic diseases to make those diseases more severe: (1) metastatic infection, (2) inflammation, and (3) immune response.[3] All three of these mechanisms have the potential to impact the systemic inflammatory/immune response that, in turn, may mediate a range of systemic diseases.

a. **Metastatic Infection.** **Metastatic infection** is an infectious disease mediated by microorganisms that originate from a distant body site. For example, certain organisms found in the oral microbiome can travel to the lung and possibly cause a lung infection.[4,5]
    1. A periodontal infection is not limited to the periodontium or even the oral cavity.[5,6] The routine acts of chewing and tooth brushing can disseminate bacteria and their products to other nonadjacent distant organs or body parts.[7]
    2. Oral bacteria from periodontal lesions and the DNA of periodontal pathogens are present systemically and invade these extraoral sites, causing systemic disorders, such as endocarditis, lung infections, abscesses of the brain or liver, and fatty deposits in the carotid arteries.

b. **Inflammation**
    1. Infection of the periodontal pocket can stimulate the oral tissues to release proinflammatory mediators that then enter the bloodstream and initiate significant systemic inflammation. Leukocytes, hepatocytes, and endothelial cells respond to oral bacteria by releasing proinflammatory mediators (cytokines, chemokines, or C-reactive proteins) that amplify systemic inflammation.[8,9]
    2. Proinflammatory mediators—such as IL-1β, IL-6, TNF-α, and PGE$_2$—produced locally in the inflamed periodontal tissues disseminate into the bloodstream and have a systemic impact.[8,10]

c. **Immune Response**
    1. In periodontitis, bacterial antigens are processed and presented to the body's immune system and recognized by lymphocytes (T-lymphocytes and B-lymphocytes). In response to a microbial challenge, host immune cells release proinflammatory mediators. As discussed in Chapters 12 and 13, it is clear that the body's immune response plays a significant role in inflammation and tissue destruction.
    2. Systemic inflammation is defined by increased circulating proinflammatory mediators such as TNF-α. Increased levels of proinflammatory mediators is associated with obesity, diabetes, and periodontitis and has been proposed as an effect that bridges all these chronic inflammatory conditions.[11,12]

Section 2

The Role of Periodontitis on Systemic Diseases

Our understanding of the link between periodontal disease and systemic health is growing rapidly. Although periodontal diseases traditionally have been considered as inflammatory diseases of the periodontium, recent studies support an association between periodontal disease and various systemic diseases/conditions, such as cardiovascular diseases, diabetes mellitus, adverse pregnancy outcomes, and pulmonary diseases.[2,13–16] Before going further into discussion, the reader should once again appreciate that, unlike the preceding chapter, which focused on the impact of systemic diseases on periodontal health, the present chapter centers on the impact of periodontitis on systemic health.

Atherosclerotic Cardiovascular Disease

1. **Overview of Cardiovascular Diseases.** Atherosclerotic cardiovascular disease (ACVD) is a group of heart or vascular diseases including angina, myocardial infarction, stroke, transient ischemic attack (TIA), and peripheral artery disease. **Atherosclerosis**, a major component of cardiovascular disease, is a process characterized by a thickening of arterial walls. An atheroma is a fatty deposit in the inner lining of an artery; also called arterial plaque.
2. **Proposed Biologically Plausible Explanations. How Is Periodontitis Linked to Cardiovascular Disease?** The Consensus Report of the EFP/AAP Workshop on Periodontitis and Systemic Diseases concludes that the impact of periodontitis on ACVD is biologically plausible.[16] Four biologically plausible mechanisms have been proposed to explain the possible link between inflammation due to periodontitis and cardiovascular disease.[17] These proposed biologic mechanisms are outlined in **Figure 15-1**.

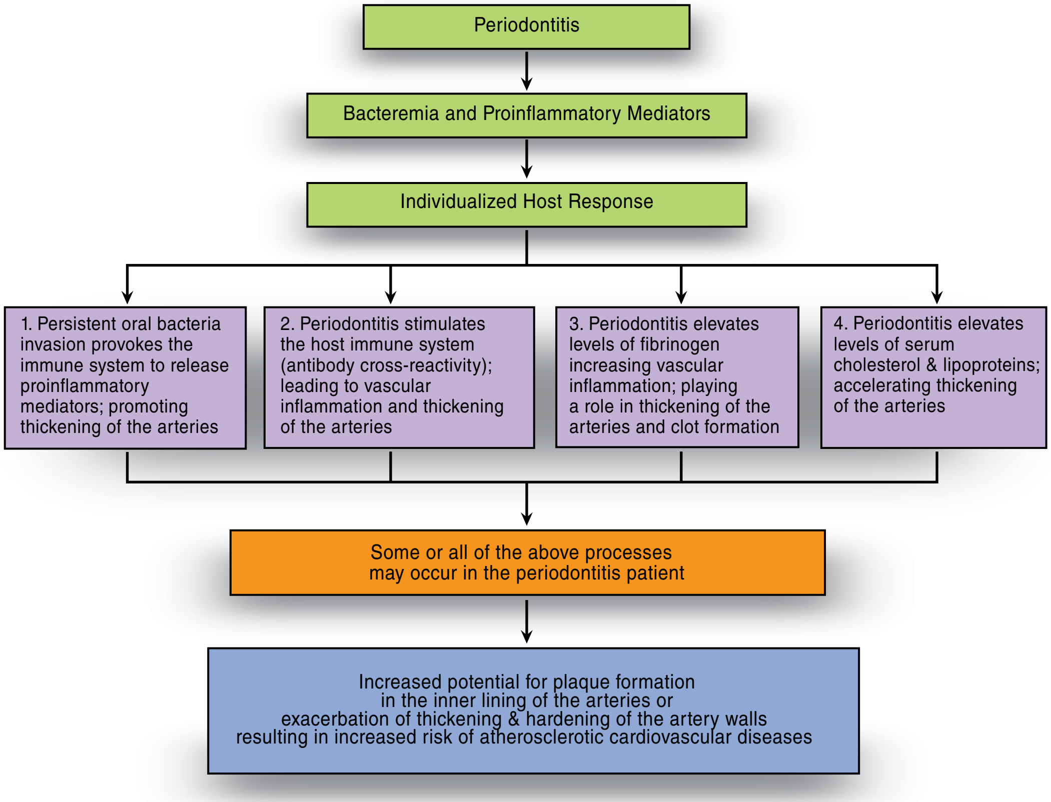
Periodontitis
Bacteremia and Proinflammatory Mediators
Individualized Host Response
1. Persistent oral bacteria invasion provokes the immune system to release proinflammatory mediators; promoting thickening of the arteries
2. Periodontitis stimulates the host immune system (antibody cross-reactivity); leading to vascular inflammation and thickening of the arteries
3. Periodontitis elevates levels of fibrinogen increasing vascular inflammation; playing a role in thickening of the arteries and clot formation
4. Periodontitis elevates levels of serum cholesterol & lipoproteins; accelerating thickening of the arteries
Some or all of the above processes may occur in the periodontitis patient
Increased potential for plaque formation in the inner lining of the arteries or exacerbation of thickening & hardening of the artery walls resulting in increased risk of atherosclerotic cardiovascular diseases

Figure 15-1 Plausible Biologic Pathways to Explain the Proposed Link Between Periodontitis and Cardiovascular Disease. A schematic representation showing the four biologic pathways that have been proposed to explain the possible link between periodontitis-induced inflammation and cardiovascular disease.

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a. Periodontal Lesions May Heighten Systemic Inflammation

1. In this first proposed pathway, periodontal infections may contribute to atherosclerosis by repeatedly challenging the arterial walls with proinflammatory mediators.
2. Subgingival plaque biofilm provides a large and persistent source of periodontal pathogens that enter the systemic bloodstream. These bacteria activate the host inflammatory response by multiple mechanisms.[18] The host immune response facilitates atheroma formation and exacerbation.[17,19]
3. Bacteria from periodontal lesions may stimulate inflammatory responses in tissues and organs distant from the oral cavity.[20–22] Thus, the evidence suggests an association between periodontal infections and a heightened state of systemic inflammation.[23]
   a. **C-reactive protein (CRP)** is a special type of plasma protein—produced primarily by the liver, but also by macrophages, endothelial cells, and smooth muscle—that is present during episodes of acute inflammation or infection.[3,24] CRP is an important cardiovascular risk predictor.[25–28] Elevations in serum CRP are well-accepted risk factors for cardiovascular disease.[27,28]
   b. One hypothesis is that periodontitis (which is the source of the inflammation) triggers the body to produce elevated levels of CRP.[28] Tellingly, *in vivo* studies have confirmed that serum CRP levels are elevated in individuals with periodontitis when compared to individuals without periodontitis.[29–31]
4. Evidence from clinical and epidemiological studies suggests that inflammation plays an important role in the onset and progression of atherosclerosis and cardiovascular disease. As such, since bacteria or their products provoke the immunoinflammatory response, a patient who suffers from any type of inflammatory disorder may be at risk of developing atheromatous lesions (fatty deposits in the inner lining of an artery).[23]

- b. **Periodontitis May Initiate the Host Immune Response: Antibody Cross-Reactivity**
  1. In this second proposed biologic pathway, periodontal infections may contribute to atherosclerosis by provoking a local immune response. Patients with periodontitis have elevated systemic antibody responses to a variety of periodontal microorganisms which are able to induce antibody cross-reactivity.[17]
  2. Cross-reactivity occurs when an antibody reacts with an antigen other than the one that initially induced its production. In this proposed pathway, the antibody reacts with the host's own endothelial cells in the blood vessel walls instead of the periodontal pathogens.[17]
  3. This pathway suggests that periodontal pathogens provoke the body's immune response to inadvertently target host cells in blood vessels, leading to vascular inflammation and atherosclerosis.
- c. **Periodontitis Elevates Levels of Fibrinogen: Increased Vascular Inflammation**
  1. In this third proposed biologic pathway, periodontal infections may contribute to atherosclerosis by triggering the host to produce fibrinogen, which plays a role in vascular inflammation.
  2. Under normal circumstances, blood coagulation (blood clotting) is a protective process that slows and stops blood loss. For example, Mary cuts her finger and starts to bleed. Immediately, the platelets in the bloodstream stimulate the production of thrombin. Thrombin, in turn, converts fibrinogen into fibrin, an insoluble protein that forms a network of thread-like structures and causes the blood plasma to gel. The blood cells and plasma enmesh in the network of fibrils to form a clot, thereby stemming blood loss at the site of vascular injury.
  3. A hypercoagulable state is a medical term for an abnormally increased tendency toward coagulation. In atherosclerotic cardiovascular disease, blood coagulation can have an adverse effect when a blood clot forms within the lumen of a coronary artery, thereby obstructing blood flow throughout the body.
  4. The coagulation and fibrinolytic systems play important roles in the thickening of the arteries and clot formation.[32,33] Elevated fibrinogen is a risk factor for atherosclerosis.
  5. The association of periodontitis with blood clotting factors has been reported by a number of investigators.
     - a. In an early study, Kweider and colleagues reported that patients with periodontitis have higher plasma fibrinogen levels than age-matched control subjects.[34]
     - b. Recent studies note increased fibrinogen levels in patients with periodontitis and an association between the number of periodontal pockets and fibrinogen levels.[35–39]
- d. **Periodontitis May Result in Dyslipidemia**
  1. In this fourth proposed pathway, periodontal infections may contribute to atherosclerosis by triggering the host to produce elevated levels of serum cholesterol, as well as low-density lipoproteins (LDLs), triglycerides, and very low-density lipoproteins (vLDL).[17]
  2. Dyslipidemia refers to abnormal amounts of lipids ("fats") and lipoproteins in the blood. A lipoprotein is a molecule that is a combination of lipid (fat) and protein. Lipoproteins are the form in which lipids are transported in the blood.
  3. Several studies indicate that blood serum concentrations of inflammatory lipids, including cholesterol, LDLs, triglycerides, and vLDL, are elevated in periodontitis patients. These inflammatory lipids may readily enter the blood vessel wall, and therefore may be incorporated into the atherosclerotic lesion (thickening of the vessel wall). This would accelerate development of the local lesions.[40–42]

3. **Clinical Relevance: ACVD and Periodontitis**
   - a. The Consensus Report of the EFP/AAP Workshop on Periodontitis and Systemic Diseases concludes that "there is consistent and strong epidemiologic evidence that periodontitis imparts increased risk for future cardiovascular disease; and while in vitro studies and animal studies support the interaction and biological mechanism, interventional trials to date are not adequate to draw further conclusions."[16]
   - b. Note, however, that insufficient evidence exists to show that the treatment of periodontal disease can reduce the risk for cardiovascular disease. The impact of treatment for periodontitis on the cardiovascular disease is an area for ongoing research.
4. **Implications for Dental Practice: ACVD and Periodontitis**
   - a. Dental health care providers should be aware of the emerging evidence that periodontitis is a risk factor for developing ACVD. All members of the dental team should be prepared to educate patients who are at risk for cardiovascular diseases about the possible impact of periodontal infection on cardiovascular health.[16,43]

b. Evidence suggests that periodontal therapy reduces systemic inflammation, but there is limited evidence on its effects on cardiovascular health in the long term.[44] Well-designed research studies are needed to clarify associations of poor periodontal health on ACVD.[45]
c. Comprehensive periodontal therapy should include patient education and advice on modifiable lifestyle risk factors such as smoking, diet, and exercise. Collaboration with appropriate specialists may facilitate the patient's efforts in making lifestyle modifications.[16,43]
d. Periodontitis patients who have other known risk factors for ACVD (such as smoking, hypertension, and obesity) *and* who have not been seen by a physician within the last year should be referred for a physical.[16,43]
e. **Table 15-1** summarizes clinical recommendations for the care of individuals with periodontitis and specific risk factors as published in the *American Journal of Cardiology* and the *Journal of Periodontology*.[16,43]

Table 15-1 Clinical Recommendations: Patients with Periodontitis and Risk Factors Associated with ACVD

Condition	ACVD Risk Factors	Recommendations
Moderate to severe periodontitis	None	Inform patient that there may be an increased risk for CVD associated with periodontitis.
Moderate to severe periodontitis	One known risk factor (smoking, family history of CVD, high cholesterol)	Recommend that patient seek a medical evaluation if he or she has not had one in the last 12 months.
Mild, moderate, or severe periodontitis	Two or more known risk factors	Refer patient for a medical evaluation if he or she has not had one in the last 12 months.

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Adverse Pregnancy Outcomes

1. **Overview of Adverse Pregnancy Outcomes**
   a. Adverse pregnancy outcomes that have been associated with periodontitis include preterm birth, low birth weight, and preeclampsia. The strength of this association, however, is modest.[14]
      1. Preterm delivery of low-birth-weight infants is a leading cause of neonatal death and long-term neurodevelopmental disturbances in children.
      2. **Preeclampsia** is a serious complication of pregnancy that can have serious (and possibly life-threatening) repercussions for both the mother and the unborn baby. For the unborn baby, preeclampsia can reduce the blood flow to the placenta, thereby decreasing the amount of oxygen and nutrients that flow to the fetus. If preeclampsia occurs prior to 37 weeks, the physician may decide that it is necessary to induce delivery to resolve this serious condition and save both the mother and unborn baby. This would result in the preterm birth of a low-birth-weight infant.
   b. Adverse pregnancy outcomes most likely involve additional shared risk factors with periodontitis, such as tobacco use, alcohol use, obesity, and diabetes.
2. **Two Proposed Biologically Plausible Mechanisms. How Is Periodontitis Related to Adverse Pregnancy Outcomes?** Research demonstrates a modest association between periodontal disease and adverse pregnancy outcomes; however, exact mechanisms remain unclear. Two major pathways have been proposed: direct and indirect.[46–48] Figure 15-2A–C shows these proposed biologic pathways.
   a. The direct biological pathway proposes that oral microorganisms and/or their components disseminate from the oral cavity and directly travel to the placenta–fetal unit. In turn, the placenta–fetal unit mounts

an inflammatory immune response by releasing increased levels of proinflammatory mediators. While these proinflammatory mediators have a protective role, they also have the potential to trigger a range of adverse pregnancy outcomes, such as hypercontractility of the uterine smooth muscle, cervical dilation, and loss of membrane integrity leading to preterm delivery or, even worse, spontaneous abortion, late miscarriage, or stillbirth (**Figure 15-2A**).

b. The indirect pathway may occur via two separate mechanisms:
    1. In response to bacterial challenge, the maternal periodontal tissues locally produce proinflammatory mediators (e.g., PGE_2, TNF-α) which enter the maternal bloodstream to initiate, intensify, and propagate the systemic inflammatory response. Ultimately, the proinflammatory mediators travel to the placenta–fetal unit, resulting in a potentially adverse outcome (**Figure 15-2B**).
    2. The oral bacteria enter the maternal bloodstream and circulate to the liver, enhancing cytokine production (e.g., IL-6) and acute phase protein reactants (e.g., CRP). The cytokines and acute phase protein reactants then spread systemically to eventually have an adverse impact on the fetal–placental unit (**Figure 15-2C**).

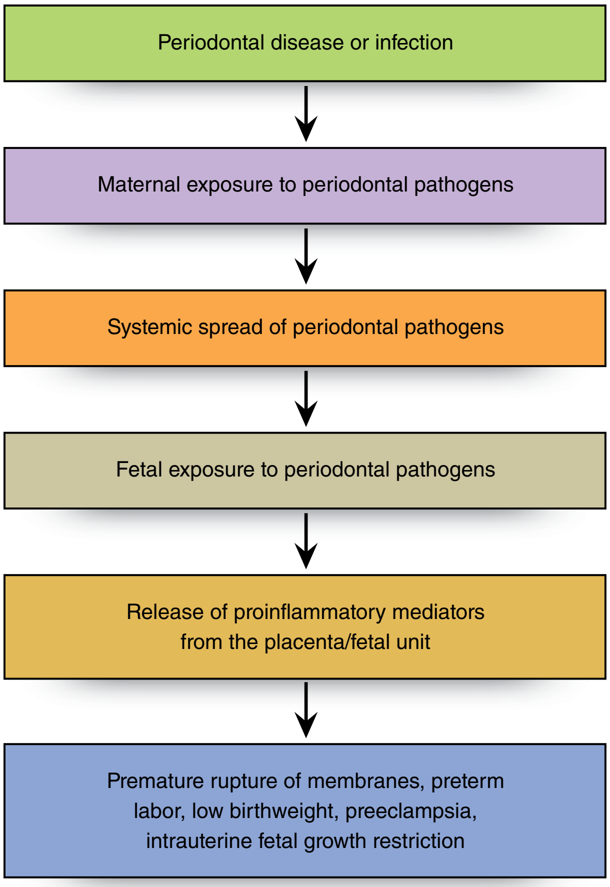
Periodontal disease or infection
Maternal exposure to periodontal pathogens
Systemic spread of periodontal pathogens
Fetal exposure to periodontal pathogens
Release of proinflammatory mediators from the placenta/fetal unit
Premature rupture of membranes, preterm labor, low birthweight, preeclampsia, intrauterine fetal growth restriction

Figure 15-2A **Direct Pathway 1.** The oral bacteria travel *directly* from the maternal periodontal tissues to the placenta–fetal unit.

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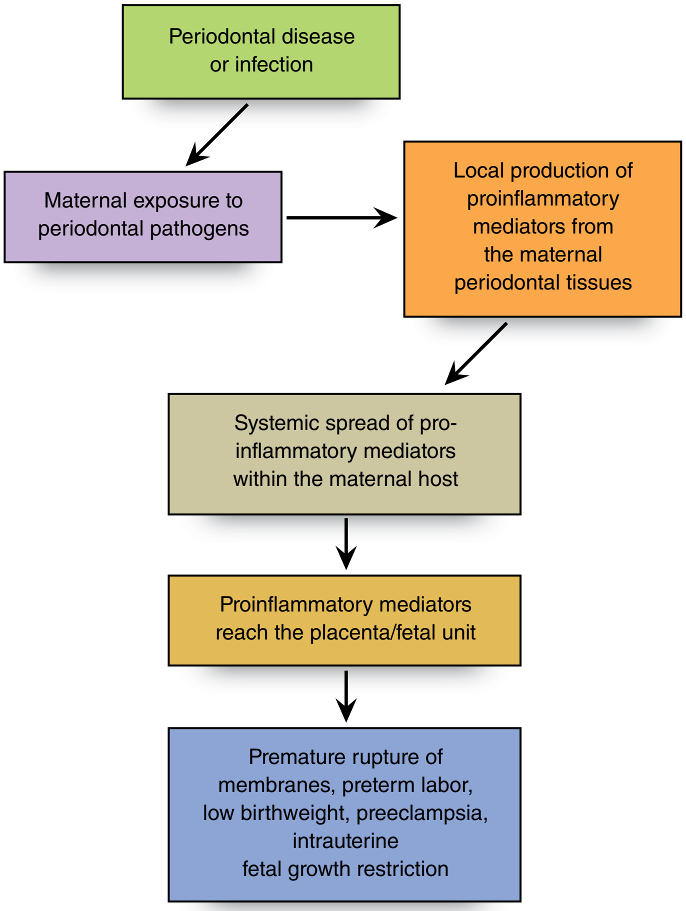
Periodontal disease or infection
Maternal exposure to periodontal pathogens
Local production of proinflammatory mediators from the maternal periodontal tissues
Systemic spread of pro-inflammatory mediators within the maternal host
Proinflammatory mediators reach the placenta/fetal unit
Premature rupture of membranes, preterm labor, low birthweight, preeclampsia, intrauterine fetal growth restriction

Figure 15-2B **Indirect Pathway 1.** Unlike the direct pathway, the oral bacteria do not directly travel to the placenta–fetal unit, but rather induce the *maternal periodontal tissues* to release elevated levels of proinflammatory mediators. From there, the proinflammatory mediators spread systemically to have an impact on the placenta–fetal unit.

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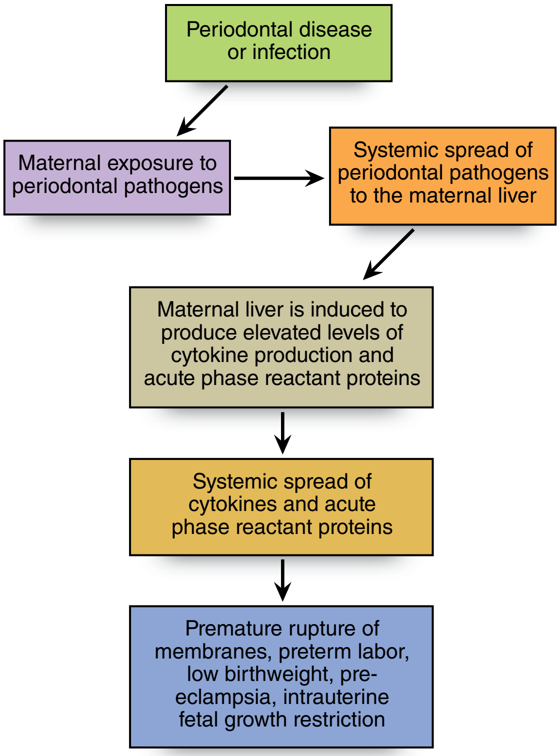
Periodontal disease or infection
Maternal exposure to periodontal pathogens
Systemic spread of periodontal pathogens to the maternal liver
Maternal liver is induced to produce elevated levels of cytokine production and acute phase reactant proteins
Systemic spread of cytokines and acute phase reactant proteins
Premature rupture of membranes, preterm labor, low birthweight, pre-eclampsia, intrauterine fetal growth restriction

Figure 15-2C **Indirect Pathway 2.** The oral bacteria spread systemically within the maternal host and reach the *maternal liver*. The liver responds by producing elevated levels of proinflammatory mediators and acute phase reactant proteins. From there, elevated levels of proinflammatory mediators and acute phase reactant proteins spread to the placenta–fetal unit to potentially induce premature contractions of the uterine wall, thus contributing to an adverse pregnancy outcome.

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3. **Clinical Relevance: Adverse Pregnancy Outcomes and Periodontitis**
   a. Periodontitis may be a significant, modifiable risk factor for adverse pregnancy outcomes. The 2013 Consensus Report of the EFP/AAP Workshop on Periodontitis and Systemic Diseases concludes that "it is very relevant to understand the extent of these associations, their possible biological mechanisms, and the implications for healthcare."[14]
   b. Note, however, that nonsurgical periodontal treatment of pregnant women does not significantly reduce the risk of preterm birth or low birthweight.[48]
4. **Implications for Dental Practice: Adverse Pregnancy Outcomes and Periodontitis**
   a. The American Academy of Periodontology issued a statement in 2004 recommending, "Women who are pregnant or planning pregnancy should undergo periodontal examinations. Appropriate preventive or therapeutic services, if indicated, should be provided. Preventive oral care services should be provided as early in pregnancy as possible. However, women should be encouraged to achieve a high level of oral hygiene prior to becoming pregnant and throughout their pregnancies."[49]
   b. Ideally, routine dental health procedures should be accomplished before conception. Dental team members assessing a female patient of childbearing age should inquire whether she is currently pregnant or trying to become pregnant. If the patient responds affirmatively, the dental health care professional should always factor in the pregnancy status when planning periodontal therapy.
      1. Health promotion information should be provided so that soon-to-be-mothers are armed with the knowledge and skillset necessary to prevent periodontal disease. Further, dental clinicians

should educate soon-to-be-mothers (and accompanying family members) about the possible effects of untreated periodontal disease to pregnancy outcomes. Dental clinicians should also be ready to provide prompt oral hygiene counseling services for pregnant women and those considering pregnancy.

2. The pregnant patient should be informed of the possible physiologic changes that take place in the periodontium during pregnancy, such as increased tissue response to biofilm, increased vascularity, and increased bleeding or gingival enlargement. The dental team should continuously counsel the soon-to-be-mother about the importance of a daily self-care regimen.
3. Emphasize that preventive, diagnostic, and nonsurgical periodontal therapeutic procedures are relatively safe throughout the second and third trimester. Elective procedures, however, should be avoided in the first trimester.
4. The pregnant patient with periodontitis should be scheduled for periodontal maintenance at a later stage during pregnancy (second or third trimester).
5. Interprofessional collaboration between the dental team and other health professionals involved with pregnancy care is strongly encouraged. It is important to remember that treatment is being rendered to two patients: mother and fetus. All treatment should be done only after consultation with the patient's obstetrician. It is best to avoid drugs and therapy that would put a fetus at risk.[50]

Diabetes Mellitus

Over the last several decades, a body of evidence supports significant associations between periodontitis and adverse effects on glycemic control in individuals with diabetes, glycemic status in individuals without diabetes, and complications of diabetes.[13,51–55] The 2018 American Diabetes Association's Standards of Medical Care in Diabetes emphasizes the importance of taking a history of past and current dental infections as part of the physician's examination.[56]

1. **Overview of Glycemic Control in Diabetes**
   a. **Glycemic control** is a medical term referring to the typical blood glucose levels in those with diabetes mellitus.
      1. Optimal management of diabetes relies on the patient's own efforts in monitoring and recording their daily blood glucose levels.
      2. If left unchecked and untreated, prolonged and elevated levels of glucose in the blood will result in serious health complications, or even death.
   b. Blood glucose level is measured by means of a glucose meter (known as a glucometer), with the result either in mg/dL (milligrams per deciliter in the United States) or mmol/L (millimoles per liter in Canada and Europe) of blood.
      1. A fasting blood glucose level (blood sugar level) less than 100 mg/dL (5.6 mmol/L) is normal. A fasting blood glucose level between 100 mg/dL and 125 mg/dL is considered prediabetes.
      2. Poor glycemic control refers to persistently elevated fasting blood glucose, greater than or equal to 126 mg/dL (7 mmol/L), or a glycosylated hemoglobin level greater than 7%.
   c. The **HbA1C level** is the average blood glucose level over the past 2 to 3 months (which corresponds to the average lifespan of the red blood cell). The HbA1C test measures the amount of glycosylated hemoglobin in the blood. Glucose in the blood enters the red blood cell and attaches to the hemoglobin. The more glucose in the blood, the more that will irreversibly bind to the hemoglobin. HbA1C is reported as a percentage of total hemoglobin in the blood. For example, an HbA1C of 8 means that 8% of the hemoglobin has glucose bound to it.
      1. A normal HbA1C level for nondiabetics is below 5.6%.[57]
      2. A reasonable HbA1C goal for many adults with diabetes is below 7%.[58]
2. **Proposed Biologically Plausible Mechanism. How Is Periodontitis Related to Diabetes Mellitus?** Diabetes is the pathological consequence of a number of physiological changes that result in impaired metabolic regulation, hyperglycemia, and inflammation that potentially impacts tissue integrity and repair. **Figure 15-3** illustrates this potential biologic pathway that provides a helpful perspective but needs further supportive evidence.

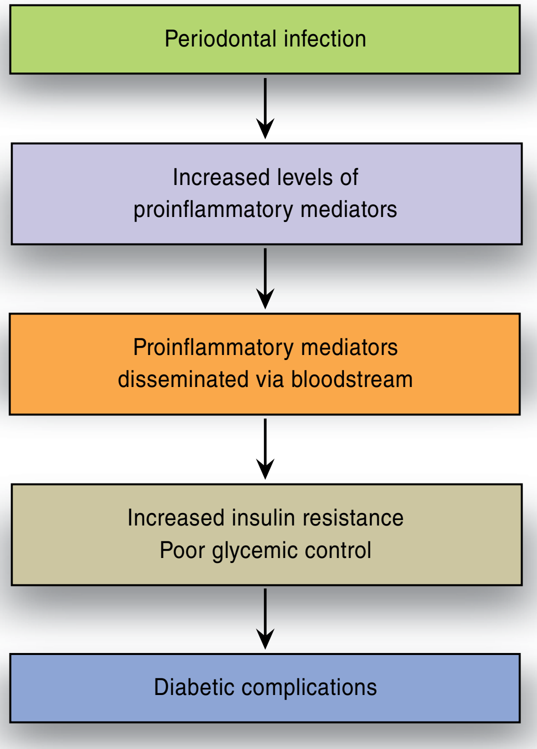
Periodontal infection
Increased levels of proinflammatory mediators
Proinflammatory mediators disseminated via bloodstream
Increased insulin resistance
Poor glycemic control
Diabetic complications

Figure 15-3 Proposed Biological Pathway for the Association Between Periodontal Disease and Diabetes. Periodontal diseases may serve as initiators of insulin resistance, thereby aggravating glycemic control.

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   a. Periodontal diseases may serve as initiators of insulin resistance, thereby aggravating glycemic control. In this proposed pathway, systemic inflammation provoked by periodontitis may increase insulin resistance, impair glycemic control, and contribute to the development of systemic complications in patients with diabetes mellitus.
   b. Evidence suggests that periodontitis raises the levels of proinflammatory mediators in the blood serum.[13,51,59–63]

3. **Clinical Relevance: Periodontitis and Diabetes Mellitus**
   a. Severe periodontitis adversely effects diabetes control. A direct relationship exists between periodontitis severity and diabetic complications in patients with diabetes, and emerging evidence suggests that severe periodontitis may predispose individuals to the development of diabetes.[13]
   b. Several studies indicate that periodontal therapy may result in improved insulin sensitivity and eventually lead to improved glycemic control and overall outcomes of diabetes mellitus.[64–66]
   c. Substantial information on potential mechanistic pathways supports a close association between diabetes and periodontitis, but there is a real need for longitudinal clinical studies using larger patient groups.[55]
4. **Implications for Dental Practice: Periodontitis and Diabetes Mellitus**
   a. Members of the dental team should be prepared to educate diabetic patients about the possible impact of periodontal infection on glycemic control and encourage oral disease prevention and treatment services.
   b. Diabetic patients should be informed of other associated oral conditions, such as xerostomia, burning mouth syndrome, fungal infections, and slower wound healing.

c. Collaboration with other health care professionals is encouraged to assist patients in managing diabetes. As our understanding of the relationship between diabetes mellitus and periodontitis deepens, collaboration among medical and dental professionals in the management of affected individuals becomes increasingly important.[51]

d. Many patients with diabetes remain undiagnosed, and oral findings may offer an opportunity for the identification of affected individuals unaware of their condition. Dental health care professionals have the opportunity to identify unrecognized diabetes or prediabetes in dental patients and refer them to a physician for further evaluation and care.[67]

Potential Associations with Other Systemic Conditions

In recent years, there has been intense interest in potential associations between periodontal disease and other various chronic systemic diseases and conditions. Diseases with possible associations to periodontitis include chronic obstructive pulmonary disease (COPD), pneumonia, chronic kidney disease, rheumatoid arthritis, cognitive impairment, obesity, metabolic syndrome, and cancer. Evidence of potential links of periodontitis to these systemic conditions is emerging, however no causal relationships can be inferred to date, with the exception that microorganisms found in the oral microbiome cause lung infections.[1,2]

Patients with periodontitis are increasingly aware of the possible links between periodontal disease and systemic diseases. Yet, to be clear, although current research demonstrates a possible association, there is no concrete evidence that periodontal disease causes COPD, chronic kidney disease, rheumatoid arthritis, cognitive impairment, obesity, metabolic syndrome, or cancer.

1. **Pneumonia.** Pneumonia is a serious inflammation of one or both lungs. It is caused by the inhalation of microorganisms and can range in severity from mild to life threatening. There are two types of pneumonia: community acquired and hospital acquired.
   a. **Overview of Pneumonia**
      1. **Community-acquired pneumonia** is pneumonia that is contracted outside of the hospital setting.
         a. Most cases of community-acquired bacterial pneumonia are caused by aspiration of oropharyngeal organisms such as *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Mycoplasma pneumoniae* into the lung.[68]
         b. Community-acquired bacterial pneumonia generally responds well to appropriate medical treatment.
         c. There is no evidence that periodontal disease or oral hygiene alters the risk for community-acquired pneumonia.
      2. **Hospital-acquired pneumonia** is an infection of the lungs contracted during a stay in a hospital or long-term care facility.
         a. Hospital-acquired pneumonia usually results from organisms called potential respiratory pathogens that are generally found in the gastrointestinal tract but may colonize the mouth and oropharynx. Bacterial plaque biofilms can serve as reservoirs of potential respiratory pathogens, particularly during prolonged hospitalization.[1,2,69–71]
         b. Oral colonization with potential respiratory pathogens increases during hospitalization, and the longer a patient is hospitalized, the greater their prevalence.
         c. **Ventilator-associated pneumonia** is a type of hospital-acquired pneumonia that develops after intubation for mechanical ventilation.
            1. In ventilator-associated pneumonia, insertion of the endotracheal tube can push oropharyngeal microorganisms deep into the lower airway, thereby triggering an immunoinflammatory response within the lung.
            2. The oral cavity may serve as an important reservoir of infection for ventilator-associated pneumonia.[1,2,70,72,73]
   b. **Proposed Biologically Plausible Mechanisms. How Is Periodontitis Related to Pneumonia?** There are four biologically plausible mechanisms to explain the possible role of oral pathogens in the onset and pathogenesis of respiratory infection.[74] These potential mechanisms are depicted in **Figure 15-4**.

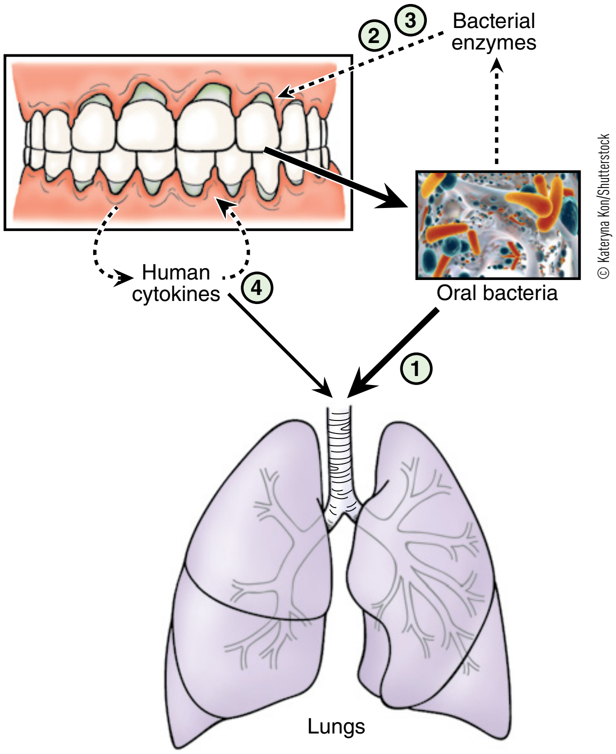

Figure 15-4 Biologically Plausible Mechanisms by Which Oral Bacteria Play a Role in Respiratory Infection. **(1)** Oral pathogens may be directly inhaled into the lungs. **(2)** Oral microorganisms may release enzymes that may damage oral mucosal surfaces, causing increased colonization by pathogenic bacteria. **(3)** Oral bacterial enzymes may reduce the protection of the protective salivary pellicle, resulting in increased colonization by pathogenic bacteria. **(4)** Salivary cytokines may alter the respiratory epithelium, promoting respiratory infection.

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1. **Direct Aspiration of Oral Pathogens into the Lung.** Oral bacteria are constantly shedding from the plaque biofilm into the saliva and subsequently aspirated into the lungs. In individuals with a competent immune system, this event ordinarily has no deleterious effect because the immune system can neutralize the oral bacteria invading the lungs. However, immunocompromised patients lack a capable immune system to fend off the bacterial invasion. As a result, these types of individuals may be more susceptible to developing a lung infection.
2. **Modification of Oral Mucosal Surfaces by Periodontal Disease–Associated Bacteria.** Patients with poor self-care and/or a deficient immune system have elevated levels of oral pathogens that have the ability to release proteolytic enzymes. The actions of the proteolytic enzymes alter the environment of the surface epithelium, thereby favoring the adhesion and colonization of potential respiratory pathogens to the mucosal surface. Subsequently, the potential respiratory pathogens can be aspirated into the lungs.
3. **Reduction of Protective Salivary Pellicle by Oral Bacteria.** Not only can bacterial-derived proteolytic enzymes alter the oral mucosal surface, but they can also have damaging effects on the salivary pellicle. Ordinarily, the salivary pellicle plays a role in defending the host by “sticking” to pathogens before they have a chance to adhere to oral mucosal tissue. In patients with poor oral hygiene, the increased bacterial levels result in elevated production of bacterial-derived proteolytic enzymes that enter the saliva from the gingival sulcus. Exposure of the salivary pellicle to the bacterial-derived

    proteolytic enzymes will cause pellicle destruction. As a result, the host has lost a means of limiting the adhesion of potential respiratory pathogens to the oral mucosa tissue. Periodontal pathogens, such as *Porphyromonas gingivalis*, have the capability to produce these damaging enzymes.
    4. **Alteration of Respiratory Epithelium by Salivary Cytokines.** Patients with poor oral hygiene have increased levels of plaque biofilm, which, in turn, stimulates cells of the periodontium and oral tissues to deploy a wide array of proinflammatory mediators. It is possible that the proinflammatory mediators originating from the host oral tissues may exit from the gingival sulcus and be mixed with the whole saliva. The contaminated saliva may travel to the respiratory epithelium and stimulate the respiratory cells to release more cytokines that have the potential to damage respiratory epithelium and make it more susceptible to colonization by both oral pathogens and respiratory pathogens.
  c. **Implications for Dental Practice: Pneumonia and Periodontitis**
    1. Ample evidence indicates that improved patient self-care and frequent professional care reduce respiratory diseases among high-risk elderly patients living in nursing homes and those in intensive care units.[69,75]
    2. Poor oral hygiene is common in patients residing in hospitals or long-term care facilities, especially in patients who are chronically ill. Dental health care providers should advocate for programs that enhance the access of dental care services to long-term care residents and collaborate with medical health care providers on the importance of improving daily oral hygiene care to this population.
    3. Application of 0.2% chlorhexidine gel to the teeth, gingiva, and other oral mucosal surfaces has been shown to significantly decrease the risk for pneumonia, especially in patients who are on ventilators.[76] Clinicians should take these findings into account when providing oral care to intubated patients.
2. **Chronic Obstructive Pulmonary Disease.** Chronic obstructive pulmonary disease (COPD) is a group of lung diseases, mainly emphysema and chronic bronchitis, characterized by an obstruction of airflow during exhalation. Several investigators have hypothesized that periodontal infections may increase the risk of COPD. Reviews of the current evidence, however, indicate that, at present, there is insufficient evidence to associate periodontal disease with COPD.[1,69,71]
3. **Chronic Kidney Disease.** Chronic kidney disease is a progressive loss of kidney function leading to permanent kidney failure. Several studies report an association between chronic kidney disease and periodontitis.[77–81] However, the complex pathogenesis of chronic kidney disease makes the role of periodontitis difficult to ascertain.
4. **Rheumatoid Arthritis.** Rheumatoid arthritis is an autoimmune disease that causes redness, warmth, swelling, and pain of the joints. If left untreated, rheumatoid arthritis can lead to debilitating deformities and functional disabilities which diminish the patient's quality of life. While the exact etiology of rheumatoid arthritis is still unknown, it is postulated that microorganisms, like *P. gingivalis*, may contribute to the pathogenesis of rheumatoid arthritis, thereby supporting the contention that there is a possible association between periodontal disease and rheumatoid arthritis.[82,83] However, there still remains contradictory evidence which question the strength of this relationship. Thus, more investigations are needed to further elucidate this relationship.[84]
5. **Cognitive Impairment.** Mild cognitive impairment is a slight but noticeable and measurable decline in cognitive abilities, including memory and thinking skills. The evidence from currently published studies for an association between periodontitis and cognitive impairment is weak.[1]
6. **Obesity.** Obesity is defined by the National Institutes of Health as a body mass index (BMI) of 30 and above. A BMI of 30 is about 30 pounds overweight. Two systematic reviews suggest a weak association between periodontitis and obesity.[85,86] In clinical practice, a higher prevalence of periodontal disease should be expected among obese adults. Overweight/obese individuals are more likely to suffer from periodontitis compared to normal weight individuals.[87]
7. **Metabolic Syndrome.** Metabolic syndrome is a cluster of conditions—increased blood pressure, high blood sugar, excess body fat around the waist, and abnormal cholesterol or triglyceride levels—that occur together, increasing the risk of heart disease, stroke, and diabetes. Emerging evidence implicates an association of metabolic syndrome, along with diabetes and cardiovascular disease, to periodontitis. However, more investigations are needed to further elucidate this relationship.[88]
8. **Cancer.** Cancer is a group of diseases involving abnormal cell growth with the potential to invade or spread to other parts of the body. Periodontitis has been identified as a possible risk factor for oral and oropharyngeal

cancer.[1] A recent review of the epidemiologic data seems to point to a possible association between periodontal disease and risk of cancer.[89] However, additional research needs to be conducted to strengthen our understanding of this association.

Chapter Summary Statement

Diseases impacting the oral cavity and systemic health share similar inflammatory pathways and similar risk factors (e.g., smoking) that exacerbate the progression and development of one another. Further, the treatment strategies for both are similar and involve a collaborative effort between medical and dental professionals.

Although research shows an association between periodontitis and various systemic diseases and conditions, albeit supported by modest evidence, well-designed research studies are still needed to better clarify associations; clinicians should keep abreast of the emerging evidence. Better patient education that emphasizes periodontal health and reinforces the importance of patient self-care, and periodontal therapy may play a role in improving overall systemic health. Profound understanding of the oral–systemic link will position the dental practitioner in a better position to safeguard a patient's systemic health.

Section 3

Focus on Patients

Clinical Patient Care

CASE 1

A new patient in your dental office is 3-months pregnant with her first child. She is 38 years old and has periodontitis. What counsel would you provide this patient about the association between adverse pregnancy outcomes and periodontitis?

Evidence in Action

You are a dental hygienist in a periodontal practice. Mr. O—a 45-year-old insurance executive—has been referred to the periodontal practice from his general dentist for treatment of periodontitis. Today is his initial appointment with you.

Mr. O tells you that there is a history of heart attacks in his family and that he has been reading all about "how gum disease causes heart attacks." In addition, Mr. O states that he wants the periodontist to prescribe antibiotics for his gum disease, as he is quite convinced that the antibiotics will eliminate the gum disease and prevent him from having a heart attack when he gets older.

What education would you provide on the association between periodontitis and cardiovascular diseases? What information would you provide to Mr. O. on use of antibiotic therapy for the treatment of chronic periodontitis?

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CHAPTER 16

Local Factors Contributing to Periodontal Disease

- Section 1 Introduction to Local Contributing Factors
- Section 2 Local Factors That Increase Biofilm Retention
- Section 3 Dental Restorations as Local Factors
- Section 4 Local Factors That Cause Direct Damage
- Section 5 Focus on Patients
  - Clinical Patient Care
  - Evidence in Action

CLINICAL APPLICATION

Periodontal diseases are inflammatory conditions that are primarily initiated by bacterial pathogens. Equally important are the roles contributing factors play in the progression of periodontal disease. Intrinsically, contributing factors do *not* initiate the onset of the disease. Rather, the presence of contributing factors (1) makes it more likely that an individual will develop periodontal disease/condition, (2) affects the progression of an already established preexisting periodontal disease/condition, or (3) predisposes a specific site of a tooth to periodontal disease/condition. The dental team must be able to identify and manage the contributing factors, when present, to minimize their impact on the periodontium. Chapters 14 and 15 highlighted several important *systemic* contributing factors that when superimposed on an existing periodontal disease/condition add to the impact of the progression of the disease/condition or heighten the likelihood of the disease/condition. This chapter shines light on several common *local (intraoral)* contributing factors associated with the onset of periodontal diseases/conditions and explains how these factors alter the progression and development of periodontal diseases/conditions.

LEARNING OBJECTIVES

- Describe how local factors contribute to the retention and accumulation of plaque biofilm.
- Explain how a local contributing factor differs from a systemic contributing factor.
- Identify and describe the location, composition, modes of attachment, mechanisms of mineralization, and pathologic potential of supra- and subgingival calculus deposits.
- Discuss how local contributing factors can lead to direct damage to the periodontium.
- Explain the role of trauma from occlusion as a possible contributing factor in periodontal disease.

KEY TERMS

Local (intraoral) contributing factors
Disease site
Dental calculus
Pellicle
Morphology
Cervical enamel projection (CEP)
Enamel pearl
Iatrogenic factor
Overhanging restoration

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Open margin
Embrasure space
Prosthesis
Supracrestal tissue attachment
Factitious injury
Malingering
Food impaction
Tongue thrusting
Mouth breathing
Traumatic toothbrushing
Dehiscence
Fenestration
Trauma from occlusion
Primary trauma from occlusion
Secondary trauma from occlusion
Functional occlusal forces
Parafunctional occlusal forces
Clenching
Bruxism
Frenal pull

Section 1

Introduction to Local Contributing Factors

In medicine, a primary etiological factor is the root cause(s) of a condition that initiates the pathologic effect (the disease). Hence, eliminating the primary etiological factor (the cause), should intuitively eliminate the effect. Equally important is the role contributing factors play in the progression of disease. In itself, a contributing factor does not initiate the disease (like a primary etiological factor); however, it can either increase the likelihood of disease, accelerate the progression of an already established disease, or aggravate the extent and severity of the already established disease. Eliminating a contributing factor alone may diminish the detrimental effects of the disease, but it will not eliminate the uninterrupted destructive effects of the disease unless the primary etiological factor is removed as well. For example, the primary etiology of diabetes mellitus is the complete or partial insufficiency of insulin secretion and/or insulin action whereas the contributing factors are poor diet, obesity, and sedentary lifestyle. Solely eliminating the contributing factors to diabetes without addressing the primary etiology may not result in demonstrable improvements in diabetic control.

Likewise, with respect to periodontal disease, the primary etiological factor of gingivitis and periodontitis is the microbial plaque biofilm challenge. But equally critical is the potential detrimental impact a contributing factor(s) can have in the development and progression of periodontal disease. As described above, a contributing factor does not initiate the onset of periodontal disease. Rather, a contributing factor plays a secondary role in either elevating the risk of an individual to developing gingivitis and periodontitis or elevating the extent and severity of an already established gingivitis and periodontitis.[1–3] Secondary factors that are present in the oral cavity and contribute to disease progression are known as **local (intraoral) contributing factors**.

Hence, it is critical for the dental team to search for any existing local contributing factors during a comprehensive periodontal assessment and understand their impact on the periodontium. The dental team should always eliminate or at least minimize the impact of existing local contributing factors during all phases of periodontal therapy.

The conditions discussed in this chapter refer to circumstances that favor periodontal breakdown and contribute to gingivitis or periodontitis in individual sites in the mouth. In the context of this discussion, a **disease site** is an individual tooth or specific surface of a tooth. For instance, a local contributing factor—such as a mesial root concavity—might predispose the mesial surface of a maxillary premolar tooth to periodontal breakdown, but not affect the periodontal support on the distal surface of the same tooth. Examples of local contributing factors include dental calculus, faulty dental restorations, developmental factors in teeth (such as a palatoradicular groove on a maxillary lateral incisor), plaque-retentive cavitated lesions, certain patient habits, and trauma from occlusion.

Local contributing factors can predispose an individual to developing gingivitis or periodontitis through several mechanisms or through combinations of these mechanisms. **Table 16-1** summarizes mechanisms for increased disease risk in local sites, and each of these mechanisms is discussed in detail in the following sections of this chapter. There are three primary mechanisms by which local factors can increase the risk of developing periodontal disease or increase the severity of existing periodontal disease:

1. A local factor can increase plaque biofilm retention.
2. A local factor can increase plaque biofilm pathogenicity (disease-causing potential).
3. A local factor can cause direct damage to the periodontium.

Table 16-1 Mechanisms for Increased Disease Risk in Local Sites

Mechanism	Clinical Example
Local factor that increases plaque biofilm retention	Rough edge on a restoration harbors plaque biofilm and/or hinders removal of the plaque biofilm with a brush and floss
Local factor that increases plaque biofilm pathogenicity (disease-causing potential)	Calculus deposits harbor plaque biofilm, allowing the biofilm community to grow uninhibited for an extended period
Local factor that inflicts damage to the periodontium	Ill-fitting dental appliance that puts excessive pressure on the gingiva History of traumatic toothbrushing Trauma from occlusion High frenal attachment

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Section 2

Local Factors That Increase Biofilm Retention

This section discusses local factors that increases plaque biofilm retention. Characteristically, these types of local contributing factors have rough or irregular exterior surfaces that favor increased plaque biofilm retention and limit the effectiveness of a patient's self-care.

1. **Dental Calculus.** Dental calculus is the most obvious example of a local contributing factor that can lead to increased plaque biofilm retention. **Dental calculus** is mineralized bacterial plaque biofilm that is covered by nonmineralized, viable (living) bacterial plaque biofilm on its external surface. Mineralization of plaque biofilm can begin from 48 hours up to 2 weeks after plaque biofilm formation.
    a. **Effects of Calculus on the Periodontium**
        1. Calculus is not as innocuous as once thought. Rather, the exterior surface of a calculus deposit, at the microscopic level, is quite irregular in contour and is always covered with disease-causing viable bacteria. Regardless of size, calculus deposits can act as plaque biofilm–retentive sites simply because their roughened and porous surfaces tend to readily harbor bacteria.
        2. As calculus deposits build up in size, they create larger plaque-retentive irregular ledges on the teeth which alter the contours of the teeth (**Figure 16-1**).

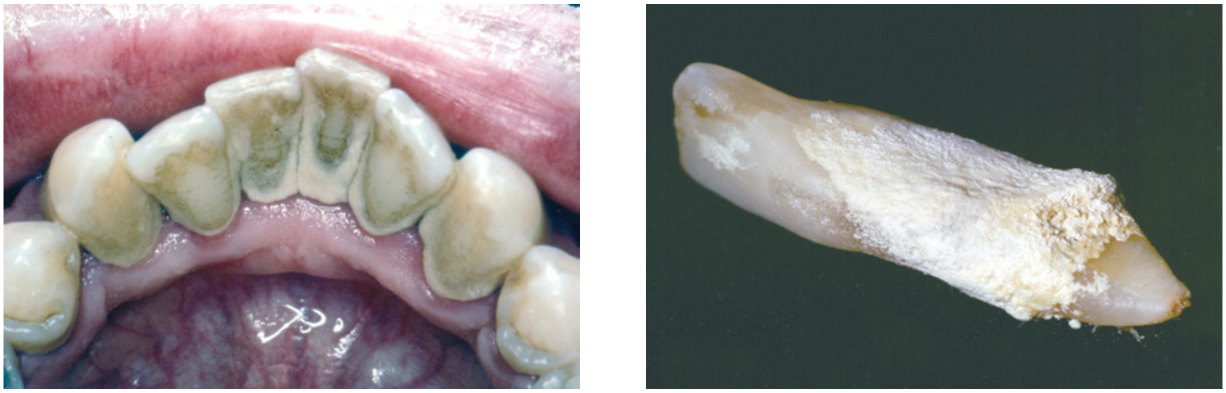

Figure 16-1 Irregular Surface of Calculus Deposits. The clinical photo on the left shows heavy calculus deposits on the lingual surfaces of the mandibular anterior teeth. These deposits are so large that they interfere with the patient's self-care efforts. In addition, calculus harbors living bacteria that act as constant irritants, which triggers the inflammatory reaction. The photo on the right depicts calculus on the crown and root surfaces of an extracted mandibular canine.

Courtesy of Dr. Don Rolfs, Periodontal Foundations, Wenatchee, WA.

3. Calculus deposits can even adhere to the roughened, threaded surfaces of dental implants. Both the roughened exterior surface of a calculus deposit and the exposed roughened, threaded surface of the dental implant act as plaque-retentive areas. As pathogenic bacteria accumulate on these plaque-retentive sites, the host inflammatory response ensues. If the inflammation is left unabated, peri-implant bone loss may occur with eventual loss of osseointegration. **Figure 16-2** shows two implants that failed due to peri-implantitis with large deposits of adherent calculus. Note how roughened and irregular the calculus deposits appear.

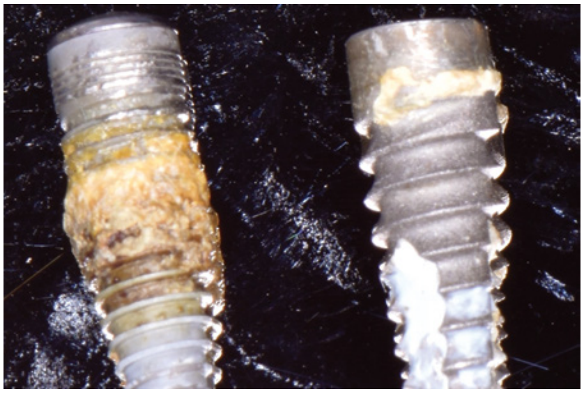

Figure 16-2 Calculus Deposits Adhering to Dental Implants. Note the irregular and jagged exterior surfaces of the calculus deposits adhering to the threaded implants. Microscopically, viable bacteria readily adhere to the roughened exterior surface of the deposit, thereby initiating the inflammatory response that led to loss of osseointegration.

b. **Pathologic Potential**
   1. Because a layer of living bacteria always covers a calculus deposit, dental calculus plays a significant role as a local contributing factor in periodontal disease. Moreover, with its close proximity to the periodontal tissues, calculus serves as a fixed source for continual plaque accumulation which can aggravate gingival inflammation.
   2. It is difficult to bring either gingivitis or periodontitis under control in the presence of dental calculus on affected teeth, and the importance of removing these deposits in patients with gingivitis and periodontitis cannot be overemphasized.

c. **Composition of Dental Calculus.** Calculus is composed of an inorganic (or mineralized) component and an organic component.
   1. **Inorganic Portion of Calculus**
      a. The inorganic part of calculus makes up 70% to 90% of the overall composition of calculus.
      b. This inorganic part of dental calculus is primarily calcium phosphate, but the dental calculus also contains some calcium carbonate and magnesium phosphate.
      c. The inorganic part of calculus is similar to the inorganic components of bone.
      d. Due to its high inorganic content, calculus can appear on a radiograph as a dense, radiopaque deposit attached to the interproximal surfaces of a tooth. Conventional radiographs can therefore be a valuable adjunctive means of detecting calculus on interproximal sites when used in conjunction with clinical detection methods (such as the use of a fine-tipped explorer).
      e. However, conventional radiograph detection methods are not always sensitive in detecting calculus deposits. The degree of mineralization of the deposit and the size of the deposit can be reasons why conventional radiography techniques are not foolproof methods in detecting calculus. At times when calculus deposits are too small, conventional radiography may not be sensitive enough to detect its presence. In addition, because a conventional radiograph—such as a bitewing radiograph and a periapical radiograph—is only a two-dimensional image of a

three-dimensional object, calculus located on the lingual or facial surfaces of teeth is not always radiographically apparent unless it is of significant size to be detectable on a conventional radiograph (refer to Figure 16-9B).

f. Note that differences in radiographic technique and procedures may also account for calculus deposits showing up on radiographs (**Figure 16-3**).

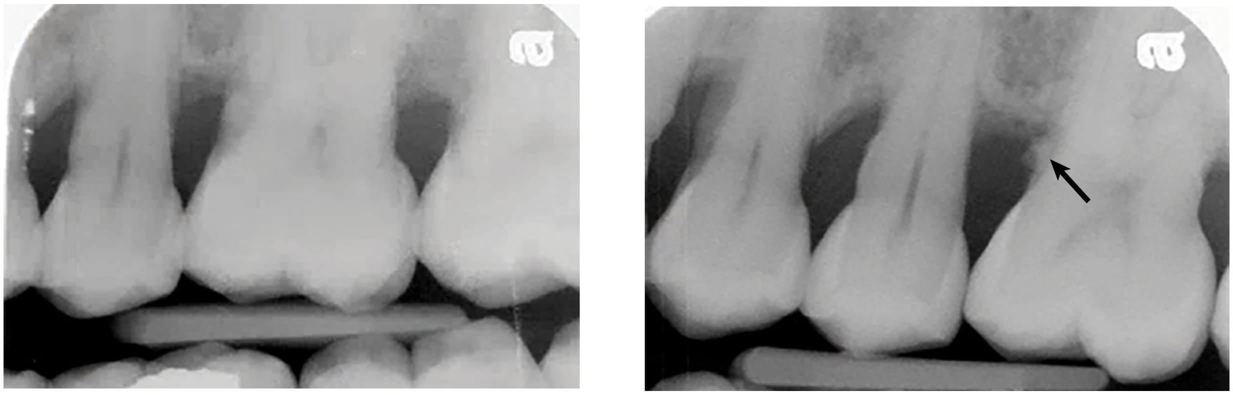

Figure 16-3 Challenges of Detecting Calculus from Conventional Radiography. Two radiographs of the *same* maxillary left posterior region with the x-ray tube oriented in different horizontal angulations. Both radiographs were taken in the same appointment. In the radiograph on the left, note the absence of radiographic calculus on the mesial surface of the maxillary first molar. However, in the right-hand radiograph, the calculus deposit is evident on the same surface (black arrow). The differences in the two radiographic images of the same teeth could be attributed to variations in radiographic technique, such as when an operator angles the x-ray tube in a slightly different orientation for each bitewing image.

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2. **Organic Portion of Calculus**
    a. The organic part of calculus makes up 10% to 30% of the overall composition of calculus.
    b. Components of the organic part include materials derived from plaque biofilm, dead epithelial cells, and dead white blood cells. It can also include living bacteria within the calculus deposit.

d. **Types of Dental Calculus**

1. **Crystalline Forms of Dental Calculus.** As calculus ages on a tooth surface, the inorganic component changes through several different crystalline forms. Some of the crystalline forms of calculus are quite similar to those found in the tooth.
    a. Newly formed calculus deposits appear as a crystalline form called brushite.
    b. In calculus deposits that are a bit more mature, but less than 6 months old, the crystalline form is primarily octacalcium phosphate.
    c. In mature deposits that are more than 6 months old, the crystalline form is primarily hydroxyapatite.
2. **Location of Calculus Deposits**
    a. Supragingival calculus deposits are located coronal to (above) the gingival margin. Other terms that have been used to refer to deposits coronal to the gingival margin are supramarginal calculus and salivary calculus. Because of its location relative to the gingival margin, supragingival calculus is visible during routine clinical examination.
        1. Although supragingival calculus deposits can be found on any tooth surface, they usually are found in localized areas of the dentition, such as lingual surfaces of mandibular anterior teeth, facial surfaces of maxillary molars, and on teeth that are crowded or in malocclusion (refer back to Figure 16-1 that shows calculus deposits adhering to crowded teeth). Note that supragingival calculus is frequently found in areas adjacent to large salivary ducts, such as the lingual surfaces of mandibular anterior teeth and the facial surfaces of maxillary posterior teeth.
        2. Although supragingival calculus can form in most any shape, these deposits most often are irregular, large deposits.

    b. In contrast, subgingival calculus deposits are located apical to (below) the gingival margin. Other terms that have been used for deposits apical to the gingival margin are submarginal calculus or serumal calculus. Subgingival calculus is not visible on routine clinical examination because it is apical to the gingival margin. Instead, subgingival calculus can only be detected by careful and delicate tactile perception with a fine-tipped explorer. Radiographs may be useful in confirming the presence of subgingival calculus, but should not be used as a substitute for clinical detection.
        1. The distribution of subgingival deposits may be localized in certain areas or generalized throughout the mouth.
        2. The shape of subgingival deposits is most often flattened. It is thought that the shape of the deposit may be guided by pressure of the pocket wall against the deposit.
        3. Generally, a deposit of calculus can be present supragingivally and extend subgingivally. However, sometimes the calculus deposit is present below the gingival margin, but absent supragingivally. This can occur if complete supragingival calculus removal is combined with ineffective subgingival instrumentation.
        4. It important to note that the classification of calculus is based on its location in relation to the gingival margin. If the gingiva recedes, then what was previously classified as subgingival calculus can be reclassified as supragingival calculus.

e. **Modes of Attachment to Tooth Surfaces.** Dental calculus attaches to tooth surfaces through several different modes, and different attachment mechanisms can even exist in the same calculus deposit.
    1. **Attachment by Means of Pellicle**
        a. Calculus can attach to the tooth surface by attaching to the pellicle on the surface. The **pellicle** is a thin, bacteria-free membrane that forms on the surface of the tooth during the late stages of eruption.
        b. This mode of attachment occurs most commonly on enamel surfaces.
        c. Calculus deposits attached via the pellicle are easily removed by the simple actions of toothbrushing because this attachment is on the surface of the pellicle (and not actually locked into the tooth surface).
    2. **Attachment to Irregularities in the Tooth Surface**
        a. Calculus can also attach to irregularities on tooth surfaces. These irregularities include cracks in the teeth, tiny openings left where periodontal ligament (PDL) fibers are detached, and grooves in cemental surfaces created as the result of faulty instrumentation during previous calculus removal procedures.
        b. Complete calculus removal in areas of irregularities in tooth surfaces is usually difficult because the deposits can be sheltered in these tooth defects.
    3. **Attachment by Direct Contact of the Calcified Component and the Tooth Surface**
        a. Calculus can also attach to tooth surfaces by attaching directly to the calcified component of the tooth. In this mode of attachment, the matrix of the calculus deposit is interlocked with the inorganic crystals of the tooth.
        b. Deposits that are firmly interlocked in the tooth surface are usually difficult to remove.

2. **Tooth Morphology. Morphology** is the study of the anatomic surface features of the teeth. A variety of local contributing factors relate to tooth morphology.
    a. **Tooth Grooves or Concavities**
        1. Naturally occurring developmental grooves and concavities in tooth surfaces frequently lead to difficulty in self-care at the site and can also be a local contributing factor for gingivitis and periodontitis because of the increased plaque biofilm retention at the site.
        2. During the natural development of some incisor teeth, a groove may form on the palatal surface of the tooth and extend apically onto the radicular (root) surface. This groove is a developmental anomaly called a palatoradicular groove (also known as radicular lingual groove) and is most frequently seen on maxillary lateral incisors. Palatoradicular grooves have been implicated as a contributing factor to periodontal disease due to their plaque-retentive features. Further, palatoradicular grooves are a challenge to maintain since they are difficult, if impossible, to clean effectively (**Figure 16-4**).

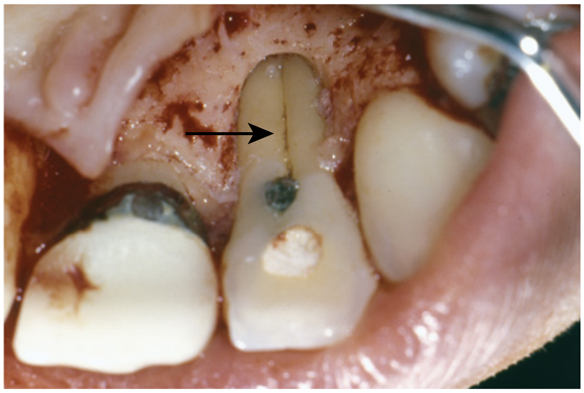

Figure 16-4 Palatoradicular Groove. Flap elevation (a periodontal surgical procedure that entails the lifting of the gingiva from the underlying bone and tooth root) reveals the presence of a long palatoradicular groove that runs down the length of this maxillary lateral incisor (black arrow). The palatoradicular groove facilitates plaque biofilm retention. Over time, the plaque biofilm can grow undisturbed in the groove and contribute to the extensive alveolar bone loss. Eliminating the palatoradicular groove via an odontoplasty at the time of flap surgery will render the root less plaque retentive and less susceptible to further attachment loss.

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3. Some tooth root surfaces have naturally developed concavities or depressions that can lead to plaque biofilm retention (**Figure 16-5**). Often, a pronounced developmental concavity is found on the mesial root surface of the maxillary first premolar. This concavity is part of the natural contour of the root of maxillary first premolar. It can also be part of the normal root anatomy of any other tooth. If exposed in the oral cavity, a root concavity can be a challenge for a patient to maintain effective self-care. The reader can refer to Figures 26-8 and 26-9 (in Chapter 26) to appreciate the difficulty in implementing adequate interproximal self-care measures of the root concavity.

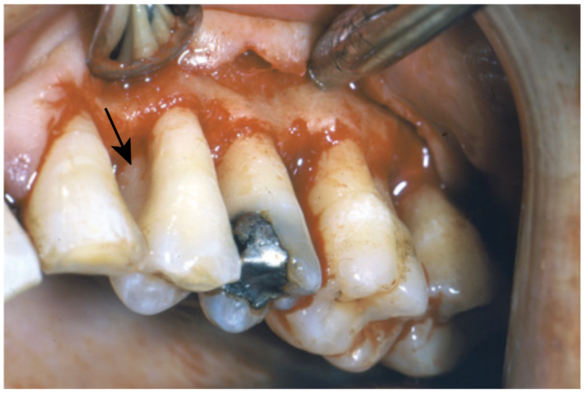

Figure 16-5 Root Concavity. The mesial root concavity on a maxillary first premolar (black arrow). This photograph was taken during a periodontal surgical procedure designed to allow better visualization and treatment of the root concavity.

Courtesy of Dr. Ralph Arnold, San Antonio, TX.

b. Cervical Enamel Projections and Enamel Pearls

1. A **cervical enamel projection (CEP)** is a flat, triangular-shaped projection of enamel that points in the direction of the furcation (Figure 16-6), whereas an **enamel pearl** is a well-defined ectopic, spherical-shaped deposit of enamel found on the root surface (Figure 16-7).

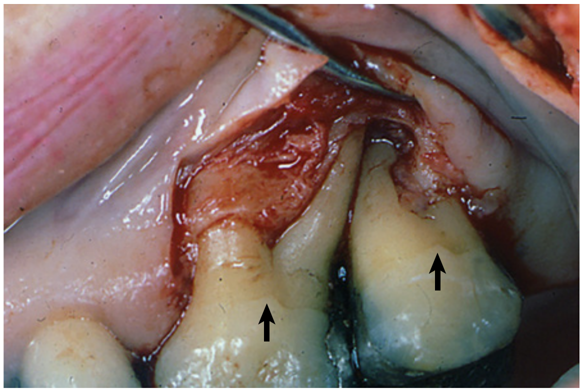

Figure 16-6 Cervical Enamel Projections. Note how the cementoenamel junction in the mid-buccal region of the maxillary first and second molars dips apically toward the furcation entrance (black arrows).

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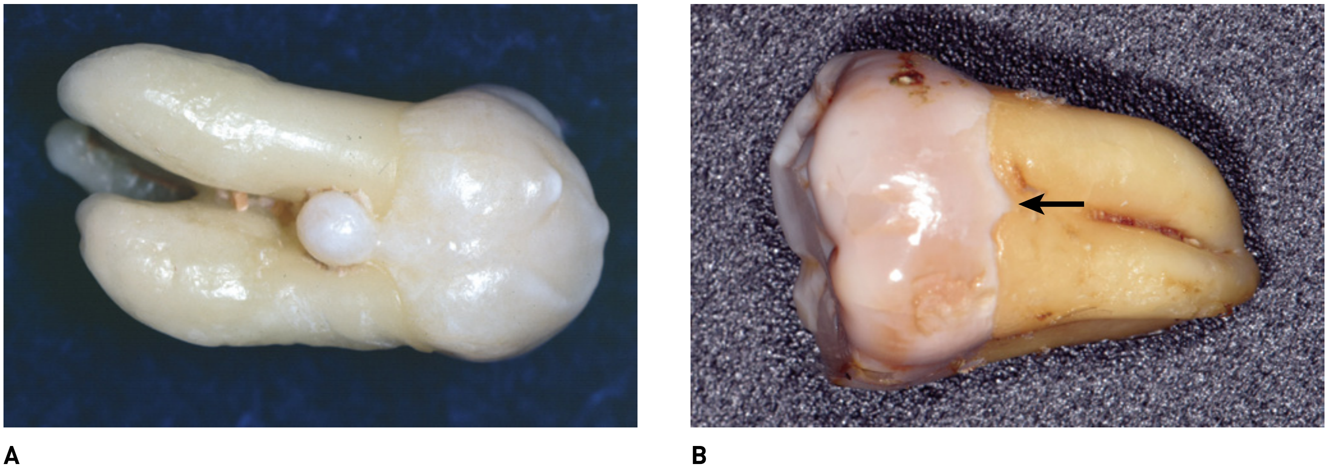

A

B

Figure 16-7 Enamel Pearl versus Cervical Enamel Projection. A. An extracted molar with an enamel pearl. Note the characteristic shape of the enamel pearl: a spherical-shaped globule of ectopic enamel on the root of this molar tooth. **B.** An extracted molar with a CEP (black arrow). Note the characteristic shape of the CEP: a flat, triangular shape of ectopic enamel that tapers as it approaches the furcation. Both enamel pearls and CEPs act as areas that retain plaque biofilm.

A: © Wolters Kluwer

2. Unlike an enamel pearl that bulges out from the surface of a root, a CEP is not easily discernible on a conventional radiograph because it is comparatively flatter than an enamel pearl. In contrast, an enamel pearl may be visually apparent because it appears as a dense radiopaque, circular globule superimposed on the root trunk of a tooth (Figure 16-8).
3. Both cervical enamel projections and enamel pearls have a predilection to be found on the root trunks of molar teeth.

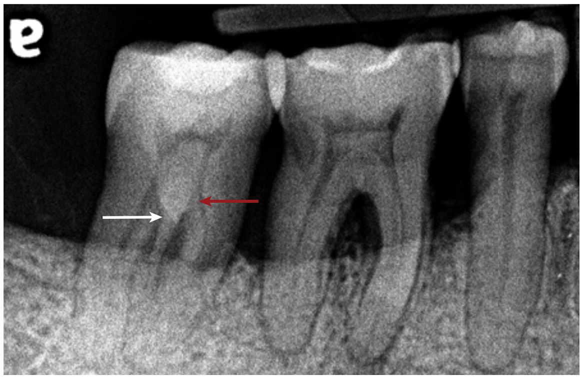

Figure 16-8 Radiographic Appearance of an Enamel Pearl. Note the well-defined circular-shaped radiodense nodule superimposed on the root trunk of the mandibular second molar (red arrow). Also note the proximity of the enamel pearl to the furcation invasion (white arrow). Because an enamel pearl retains plaque, pathogenic plaque does not have to travel too far before it affects the furcation and results in interfurcal bone loss.

4. Both a cervical enamel projection and an enamel pearl can potentially retain plaque biofilm, thereby increasing the likelihood of a furcation invasion. All efforts should be made to identify these local factors through thorough clinical and radiographic methods. Following detection, these local factors should be surgically removed.

c. **Malocclusion**

1. Dental malocclusion is a developmental anomaly that is associated with irregular alignment of teeth (also known as malalignment). Malignment poses challenges to a patient's ability to properly perform self-care practices such as tooth brushing and flossing. As such, teeth malignment predisposes the area to biofilm retention and, as a result, gingival inflammation (**Figure 16-9**).

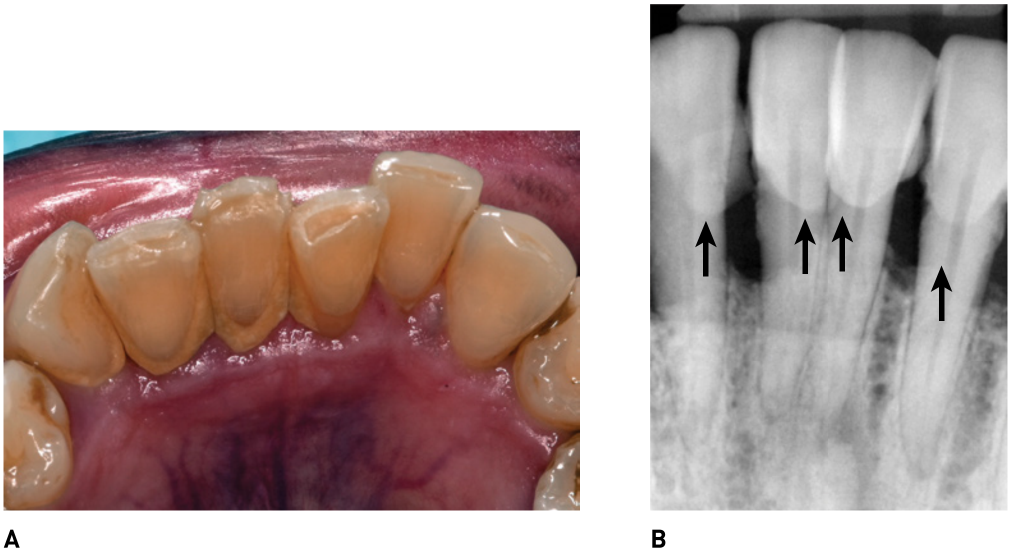

Figure 16-9 Plaque-Retentive Features of Malocclusion. Crowding of teeth leads to increased retention of plaque biofilm that can hinder the best attempts by the patient to keep the area clean. **A.** Visually note the supragingival plaque/calculus deposits and the gingival inflammation. **B.** On the accompanying radiograph of the same patient, note the significant calculus accretions on the root surfaces and the extent of bone loss. The calculus deposits are so large that the accretions on the lingual surfaces of the mandibular teeth are detectable on the conventional periapical radiograph (black arrows).

Courtesy of Dr. Brandi Herron, Indianapolis, IN.

2. In some cases, the malocclusion may be so severe that it necessitates a close collaborative, multidisciplinary approach involving the patient, the orthodontist, the general dentist, the periodontist, and the dental hygienist to correct the malocclusion and make the oral cavity more amenable to better plaque control (**Figure 16-10**).

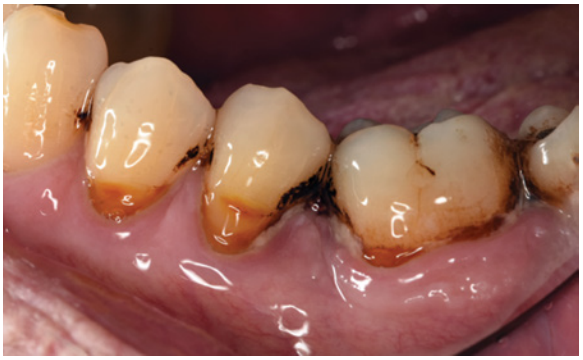

Figure 16-10 **Plaque Retentive Features of Dental Malocclusion.** A 52-year-old patient presented with severe periodontitis. Dental history revealed that the mandibular first molar was extracted over 30 years ago. Since then, the remaining molars tilted mesially. Presently, note the heavy band of plaque, staining (due to patient's current smoking status), and soft tissue inflammation.

d. **Dental Caries.** Untreated tooth decay is another example of a local contributing factor that can increase plaque biofilm retention. Because tooth decay results in a defect in tooth structure (dental cavities), a cavity can act as a protected habitat for bacteria to live and grow undisturbed (**Figures 16-11** and **16-12**). In such a case, management of the carious lesion is equally paramount to managing the patient's periodontal condition.

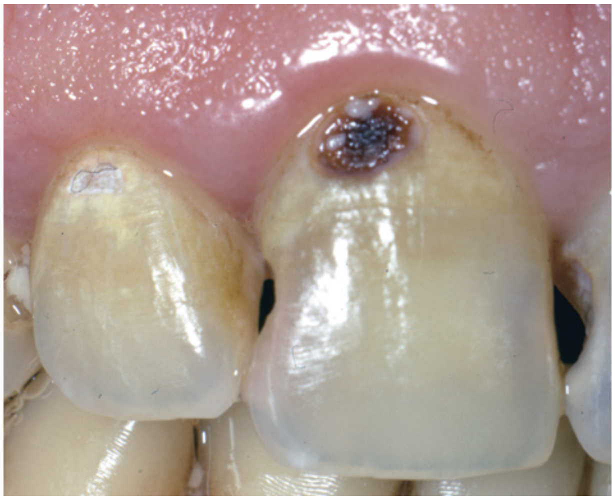

Figure 16-11 **Untreated Decay.** Untreated tooth decay leads to the creation of an actual concavity (cavity) on the tooth surface that can then harbor periodontal pathogens and can allow them to grow undisturbed by self-care efforts.

Courtesy of Dr. Ralph Arnold, San Antonio, TX.

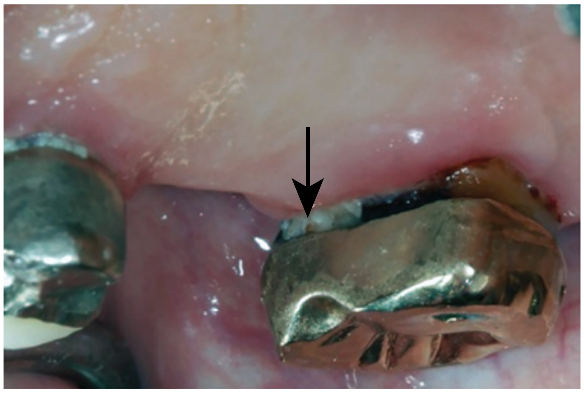

Figure 16-12 **Untreated Recurrent Decay and Its Implications to the Surrounding Periodontium.** Recurrent decay is a type of dental decay that develops around a preexisting restoration. Note the recurrent decay apical to the existing crown margin. Also note the plaque biofilm residing within the confines of the recurrent decay (black arrow). The cavitated lesion retains plaque well and handicaps self-care measures. Consequently, the gingival tissue becomes inflamed.

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e. **Orthodontic Appliances.** An unintended consequence of any orthodontic appliance (such as orthodontic brackets, archwires, and bonded retainers) is that it tends to harbor plaque due to its plaque-retentive properties. Additionally, because it makes the surfaces of teeth less accessible to the bristles of a toothbrush and conventional flossing, the orthodontic appliance creates more challenges for a patient to properly practice good self-care measures. Further, it limits the physiological self-cleaning mechanisms of the tongue and cheeks. As a result, periodontal complications are one of the more common unintended side effects linked to orthodontic appliances. **Figure 16-13** shows a patient during orthodontic treatment with an orthodontic bonded retainer in place on the lingual surfaces of the mandibular anterior teeth. **Figure 16-14** shows the same patient, post-treatment, immediately after removal of the retainer; note the calculus deposits on the lingual surfaces of the mandibular anterior teeth. To limit the occurrence of any unintended periodontal consequences, the patient's periodontal condition must be evaluated at every appointment. If an orthodontic patient exhibits diminished compliance, it is incumbent on the dental provider to collaborate with the patient and the orthodontist to reinforce and repromote the importance of good oral health.

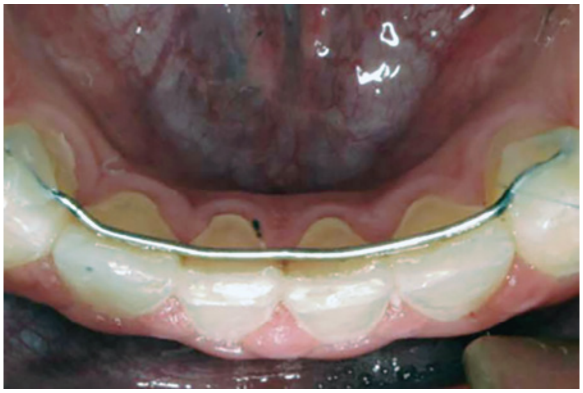

Figure 16-13 Orthodontic Appliance on Mandibular Anterior Teeth. Patient with orthodontic retainer bonded to the mandibular anterior teeth. Note mild gingival inflammation and marginal calculus deposits.

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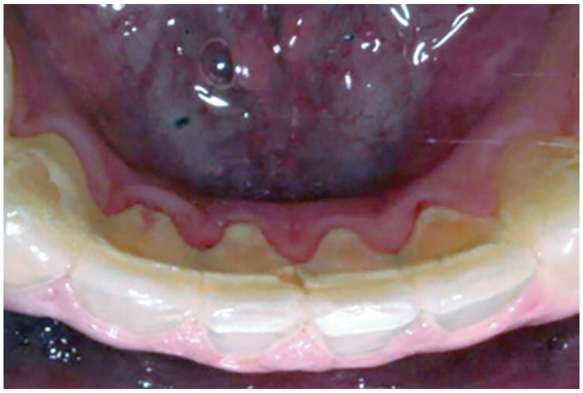

Figure 16-14 Calculus Deposits Evident on Same Patient. The same patient case (as in Figure 16-13) after the bonded retainer was removed. Note the light rim of supragingival calculus on the lingual tooth surfaces.

Courtesy of Dr. Corrie Swihart, Indiana University School of Dentistry, Indianapolis, IN.

Section 3

Dental Restorations as Local Factors

Most dental procedures have a positive impact on oral health. In some cases, however, dental treatment may have the opposite, unintended consequence of promoting the adherence and colonization of bacteria that causes tooth decay or periodontal disease. Treatment that results in an inadvertent, adverse outcome is known as an **iatrogenic factor**. Some well-known examples of iatrogenic factors that increase plaque retention are improperly contoured restorations, overhanging restorative margins, open restorative margins, open contacts, orthodontic appliances, or a poorly designed dental prosthesis.

1. **Damage Due to Improperly Contoured Restorations**
   a. **Overhanging Margins on Restorations**
      1. Ideally, the restorative margin should blend smoothly with the natural contours of the restored tooth. However, in some cases, it is not always possible for the dentist to contour the restoration smoothly with the surrounding tooth. When excess restorative material extends over the cavity margin or normal contours of the tooth, this condition is referred to as an **overhanging restoration**, or more simply as an "overhang" (**Figures 16-15**, **16-16**, and **16-17**). One can think of an overhang as the excess part of the restoration that "hangs over" the normal contours of the tooth.

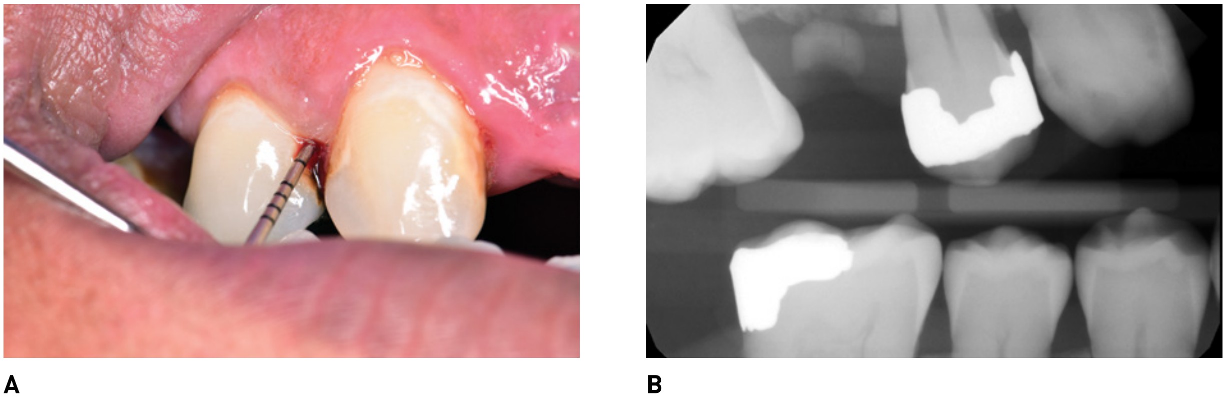

Figure 16-15 **Poorly Contoured Restorations. A.** Profuse bleeding noted on the mesial of the maxillary premolar. **B.** Accompanying bitewing reveals the presence of an overhang on the mesial site that harbors subgingival biofilm and contributes to the gingival inflammation apparent in the clinical photograph.

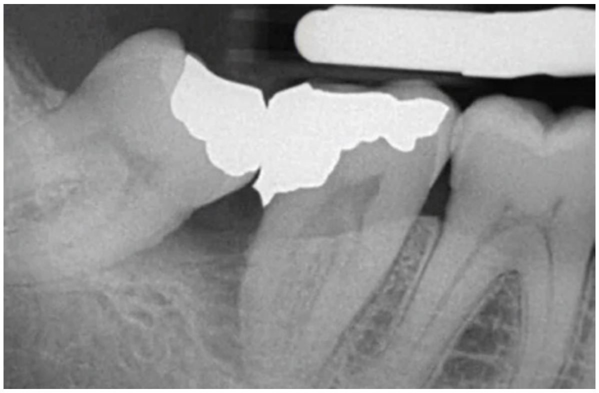

Figure 16-16 **Radiographic Evidence of a Poorly Contoured Restoration.** Large distal overhang on the mandibular second molar. The overhang triggered the inflammatory response that eventually led to irreversible periodontal breakdown (radiographically evident by the angular defect on the distal aspect of the affected tooth).

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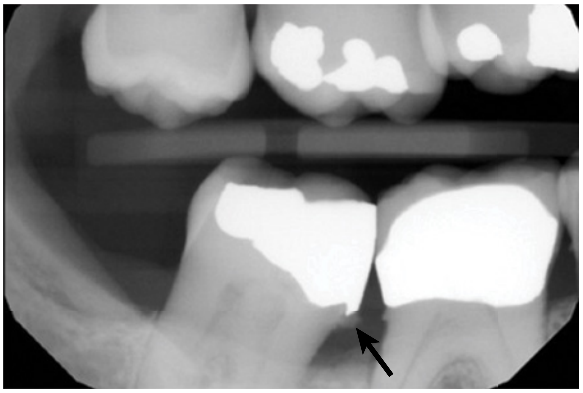

Figure 16-17 **Radiographic Evidence of a Poorly Contoured Restoration.** Note the large deposit of calculus just apical to the mesial overhang of the mandibular second molar (black arrow).

2. Because of the difficulty in accessing the tooth surfaces apical to the overhanging restoration, it is often impossible for a patient to effectively remove plaque biofilm from the tooth surface. This leads to the retention of bacteria that can cause either dental caries or periodontal disease.
3. If an overhanging margin is detected, the dental clinician should make every effort to smoothen the margin with either a rotary and/or hand instrument to make it more flush with the surrounding tooth structure. This will make the area less plaque retentive. If the overhanging margin is too large to be smoothened with a rotary and/or hand instrument, the clinician should remove the entire restoration and place a more well-contoured restoration that fits within the confines of the cavity preparation.

b. **Open Margins on Restorations**

1. There should be a smooth transition and seal between a restoration and the surrounding tooth without any gaps, spaces, grooves, or rough areas. Whenever there is a space or gap between the edge of a restoration and the natural, unprepared tooth structure, it is referred to as an **open margin** (Figure 16-18).

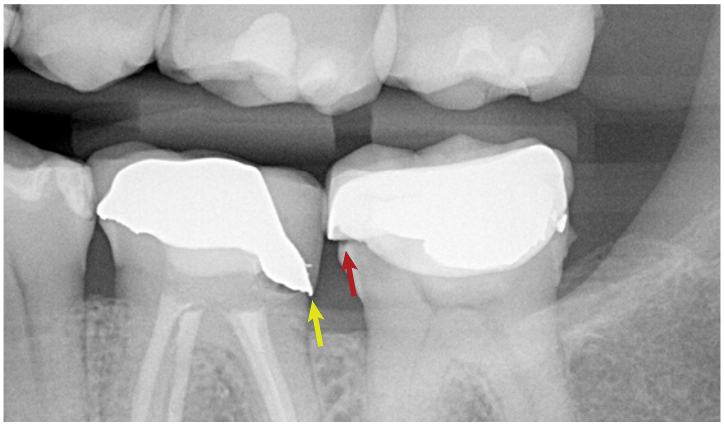

Figure 16-18 Radiographic Evidence of Poorly Contoured Restorations; Open Margin and Overhanging Margin: An open margin is a gap between the restoration and the prepared tooth surface. Note the presence of an open margin on the mandibular second molar (red arrow). An overhanging margin is an excess part of the restorative material that extends beyond the cavity preparation. Note the presence of an overhanging margin on the maxillary first molar (yellow arrow). The poor contours of an open margin and an overhanging margin create an environment that retains plaque, thereby contributing to recurrent decay and/or periodontal disease. Consequently, when either an open margin or overhanging margin is detected, the dental hygienist should bring this to the immediate attention of the dentist.

2. Open margins are a problem when plaque biofilm gets into this open gap. The accumulation of plaque biofilm in the open margin is enough to trigger gingival inflammation (**Figure 16-19**). Hence, like an overhang, an open margin has the potential to be plaque retentive.

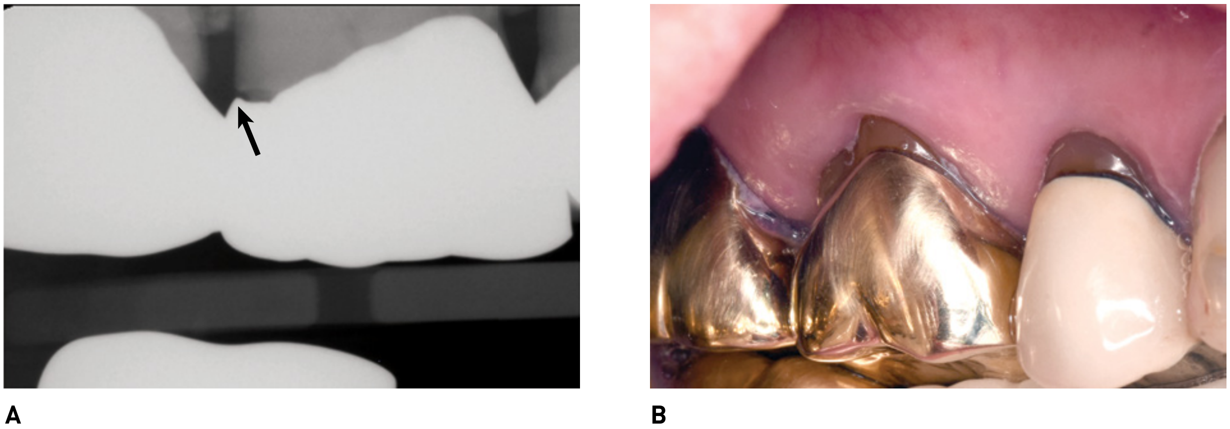

Figure 16-19 Consequences of an Open Margin. A. Radiograph reveals an open margin (black arrow) on the distal aspect of the full-coverage crown on the maxillary first molar. **B.** Accompanying clinical photograph of the same case. Note the edematous papilla between the two molars.

c. **Bulky or Overcontoured Crowns/Restorations**

1. Bulky or overcontoured crowns/restorations fail to reproduce the original anatomy of the natural tooth contour. Rather, the bulky restoration appears much more bulbous and much larger than a natural tooth. The poor contour of the restoration traps the bacteria which can cause decay or periodontal disease (**Figure 16-20**). Further, because the overcontoured restoration is unnaturally bulky and

protuberant, flossing is much more challenging as it easily shreds when snapped in between the difficult-to-reach interproximal areas.

a. The space apical to the contact area of two adjacent teeth is referred to as an **embrasure space**. In health, the embrasure space is filled by an interdental papilla. The ideal shape and size of the embrasure space is one that permits the introduction of interproximal cleaning devices that aids in the removal of plaque.

b. Bulky crowns impinge on the embrasure space by reducing the size of the embrasure space so that inadequate space exists between the teeth to accommodate adequate interproximal access. In this situation, the bulky crowns are described as encroaching on the embrasure space (**Figures 16-21** and **16-22**).

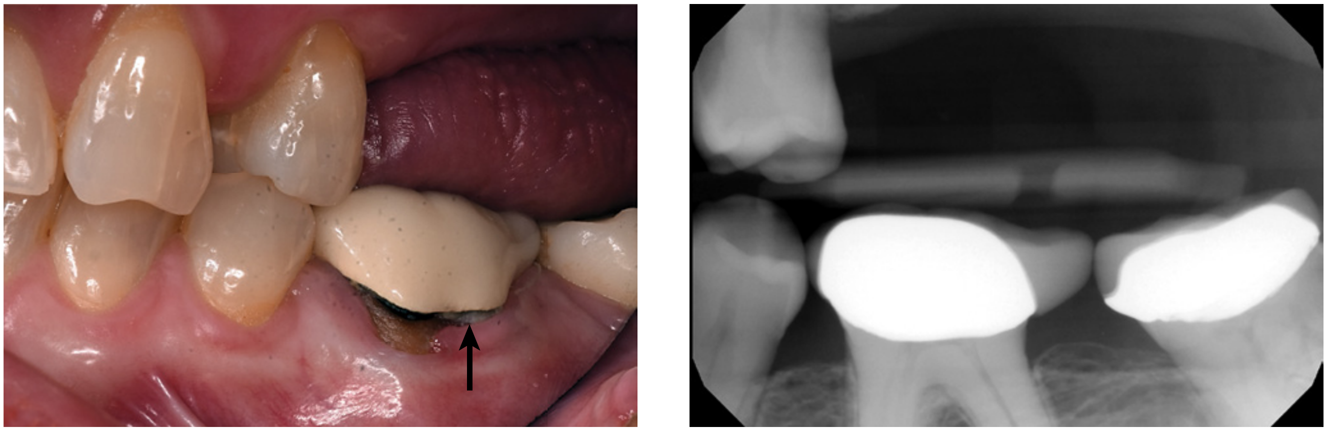

Figure 16-20 Periodontal Implications of a Bulky Crown. A. Note the abnormally, unnatural bulbous contour of the crown contributing to the edematous papillae and rolled margin of the mandibular first molar. Also note the plaque biofilm accumulation apical to the margin of the overcontoured crown (black arrow). **B.** Accompanying radiograph reveals the bulbous shape and bulky contour of the restoration.

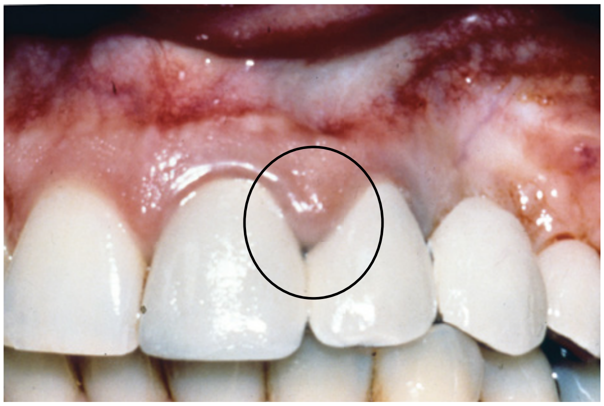

Figure 16-21 Bulky Crown Encroaching on Embrasure Space. The crowns shown here are so bulky in contour on their proximal surfaces that they overfill the embrasure space, leaving no room for the natural form of the papilla. Note the papilla between the central and lateral incisor appears enlarged because it is being pushed from between the teeth.

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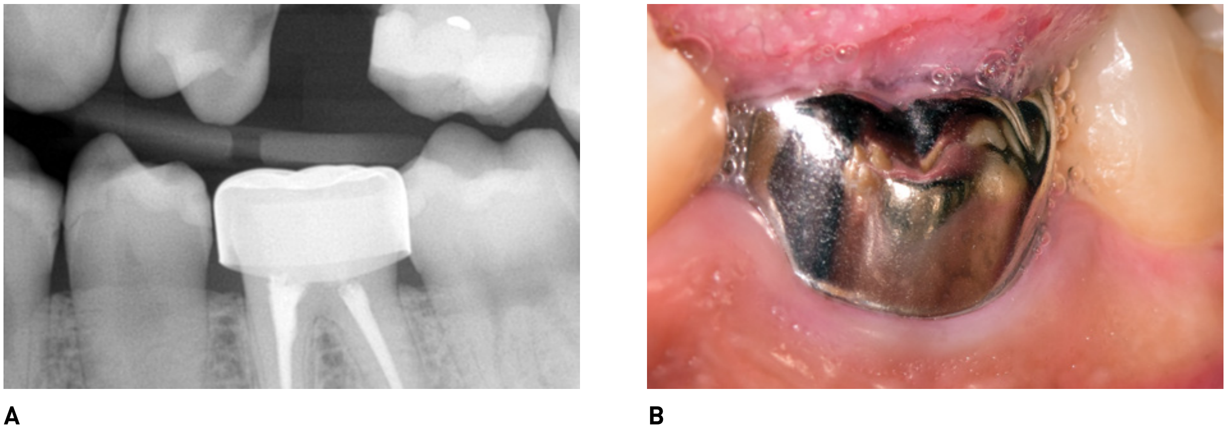

Figure 16-22 **Bulky Stainless Steel Crown Impinging on the Embrasure Space. A.** Radiograph of the overcontoured crown on the mandibular first molar. Note the rectangular shape of the crown. This shape does not conform to the natural contours of a tooth. **B.** Accompanying clinical photograph of the same case. Note the appearance of a rolled gingival margin and edematous (swollen) papillae.

2. A bulky contoured restoration acts as an area of biofilm accumulation and retention.
   a. The effectiveness of patient self-care is limited when a restoration is poorly contoured to the tooth surface. A poorly contoured restoration is a contributing factor to the development and spread of periodontal inflammation.
   b. It should be kept in mind that a poorly contoured restoration on a dental implant can have the same adverse effects as on a natural tooth (**Figure 16-23**).

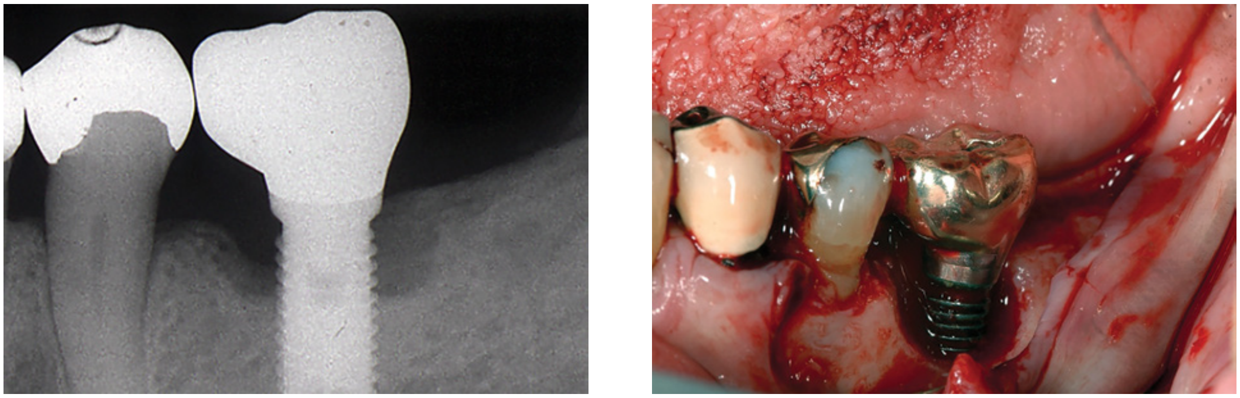

Figure 16-23 **Poorly Contoured Crown on an Implant.** This radiograph reveals a cup-like bony defect around the coronal third of a dental implant. Periodontal surgery (flap reflection) reveals an extensive defect in the alveolar around the implant.

Courtesy of Dr. Kazushi Yasumasu, Munakata, Fukuoka, Japan.

3. A restoration with a bulky contour may also act as an area of food impaction. Food impaction is the forceful trapping of food in between teeth. This can be unpleasant as it can create a wedging sensation between teeth. If not removed, the impacted food will serve as fuel for bacteria that causes tooth decay or periodontal disease.

2. **Damage Due to Faulty Prosthetics and Appliances.** A dental **prosthesis** is an intraoral substitute—such as crown, fixed bridge, or removable denture—that restores and replaces missing parts of teeth, missing teeth, and missing soft or hard tissues of the jaw and palate. The dental prosthesis can be a challenging area for the patient to keep clean (**Figures 16-24** and **16-25**). As a result, a dental prosthesis can potentially retain plaque biofilm that gives rise to periodontal inflammation. A dental practitioner should be aware of the risk associated with a faulty designed prosthesis and should make every effort to design a prosthesis that has minimal plaque biofilm retentive features.

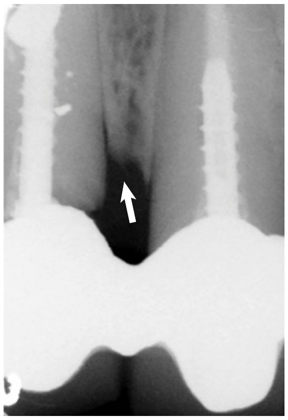

Figure 16-24 Splinted Crowns on the Maxillary Central Incisors. The incisor teeth have splinted crowns (the crowns are connected). This splinting makes interdental plaque removal difficult for the patient. As a result, an angular defect has formed in the alveolar bone between the two incisors (white arrow).

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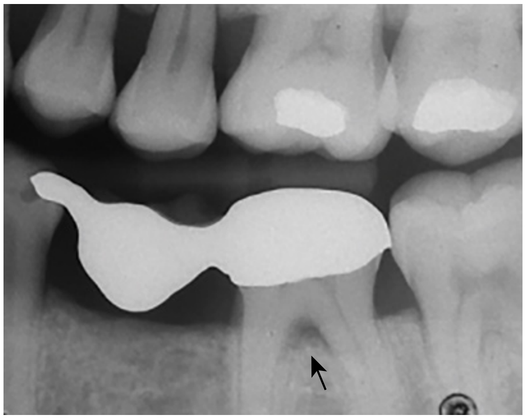

Figure 16-25 The Design of the Dental Prosthesis Can Facilitate Plaque Biofilm Retention. A less-than-ideally designed dental prothesis can hinder the patient's best self-care efforts. In this case, the plaque-retentive fixed bridge prothesis may have contributed to the furcation invasion seen on the abutment tooth (black arrow).

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a. **Inappropriate Crown Placement.** A crown is a metal, ceramic, or ceramic-bonded-to-metal covering for a badly damaged tooth. Placing a crown on a damaged tooth is a common way of restoring the form, function, and esthetics of the damaged tooth.

   1. A properly contoured crown with the edges of the crown (called margins) placed at least 2 mm coronal to the alveolar crest is paramount in maintaining gingival health and improving the prognosis of the previously damaged tooth.
   2. A crown margin that encroaches this 2 mm zone can result in alveolar bone resorption. This violates the supracrestal tissue attachment.
   3. **Supracrestal tissue attachment** (previously termed as "biologic width") refers to the zone of soft tissue occupied by the junctional epithelium plus the zone of connective tissue attachment fibers immediately apical to (below) the junctional epithelium (**Figure 16-26**). One can think of the supracrestal tissue attachment as the part of the periodontium that is coronal to the alveolar crest.

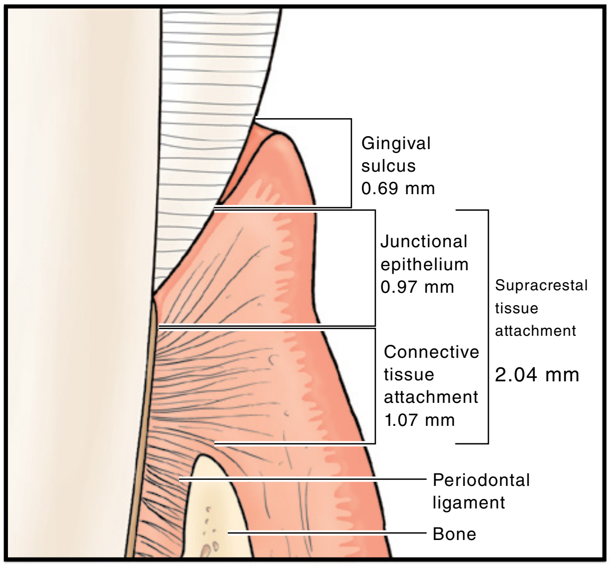
Gingival sulcus 0.69 mm
Junctional epithelium 0.97 mm
Supracrestal tissue attachment
2.04 mm
Connective tissue attachment 1.07 mm
Periodontal ligament
Bone

Figure 16-26 Supracrestal Tissue Attachment in Health. Illustration showing the supracrestal tissue attachment in health with average dimensions that have been reported in the literature. The 2017 AAP/EFP World Workshop replaced the previously termed "biologic width" with the more anatomically appropriate term "supracrestal tissue attachment" to reflect the zone of soft tissue coronal to the alveolar crest.

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a. This supracrestal tissue attachment is “violated” if the margin of a restoration encroaches upon this zone. If this occurs, the body will attempt to reestablish this zone by recreating room between the alveolar crest and the restorative margin to allow space for the junctional epithelium and the connective tissue to reform.

b. Unfortunately, when the supracrestal tissue attachment is violated, bone resorption and gingival recession are unintended consequences. **Figure 16-27** is an illustration of the ramifications of a restorative margin violating the supracrestal tissue attachment; **Figure 16-28** depicts the ramifications of an actual restorative margin violating the supracrestal tissue attachment.

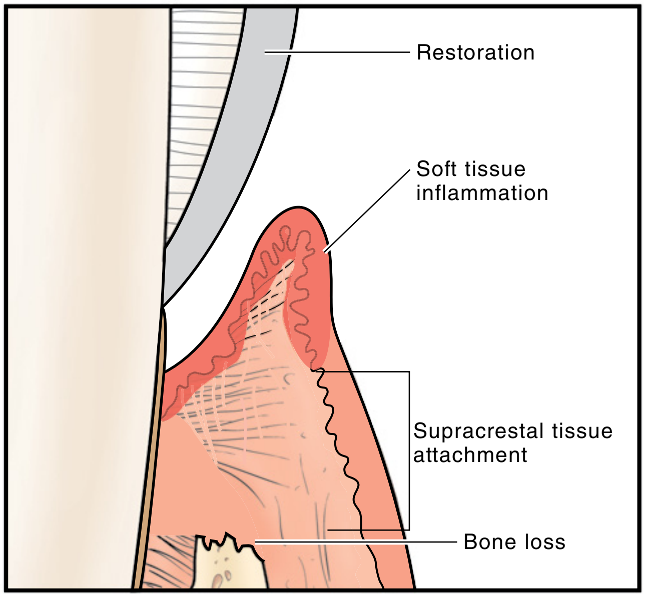

Figure 16-27 **Consequences of a Restorative Margin Violating the Supracrestal Tissue Attachment.** If a restorative margin is placed too subgingival and violates the supracrestal tissue attachment, two undesirable consequences may occur: uncontrollable bone loss and/or persistent gingival inflammation.

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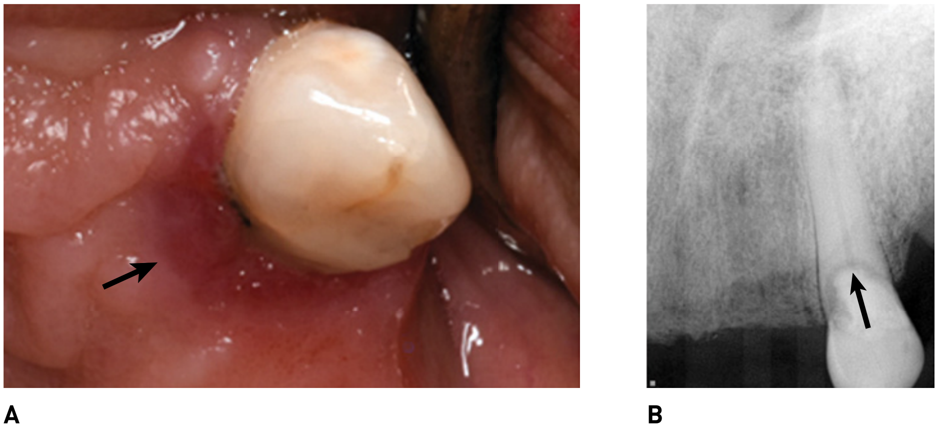

Figure 16-28 **Consequences of a Restorative Margin Violating the Supracrestal Tissue Attachment.** Several weeks prior to obtaining the above photograph, the lingual surface of the maxillary canine was restored with a resin restoration. The margin of the restoration extended subgingivally and impinged on the supracrestal tissue attachment. **A.** Clinical photograph of the actual case 1-month post restoration. Note the intense gingival inflammation associated with the lingual aspect (arrow). **B.** Accompanying radiograph depicts the encroachment of the restorative margin to the alveolar crest (arrow).

Courtesy of Dr. Erik Rose, Indianapolis, IN.

b. **Faulty Removable Prosthesis**

1. A removable prosthesis is one that the patient can remove for daily cleaning and before going to bed. A removable prosthesis that replaces a few teeth is commonly called a removable partial denture (commonly abbreviated as an RPD). A removable prosthesis that replaces an entire arch is referred to as a removable complete denture. A removable prosthesis that is attached to implants is known as an implant supported denture.
2. A removable prosthesis should be differentiated from a fixed prosthesis. A fixed prosthesis is permanently cemented to a single tooth (a crown), to several teeth (also known as a fixed bridge), or to an implant or several implants (known as implant-supported crown or bridge).
3. A damaged or poorly fitting removable prosthesis can impinge on gingival tissue and favor plaque biofilm accumulation. Eventually, this triggers periodontal inflammation (**Figure 16-29**).

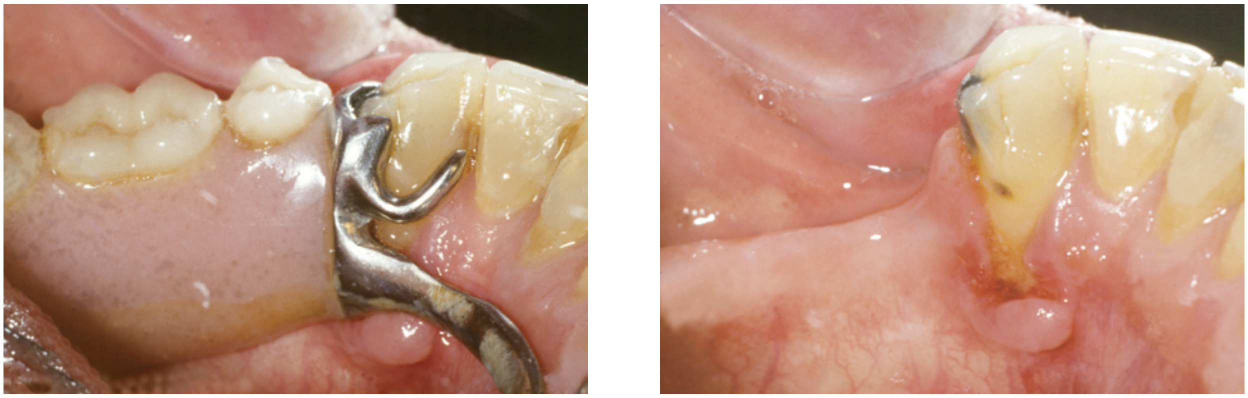

Figure 16-29 Tissue Damage by a Poorly Fitting Removable Prosthesis. The clinical photograph on the left shows a removable prosthesis (lower partial denture) seated in the patient's mouth. In the right photograph without the prothesis, the tissue damage to the mandibular canine is evident. Gingival recession on the canine is due in part to the clasp of the faulty prosthesis impinging upon the gingival tissue.

Courtesy of Dr. Don Rolfs, Periodontal Foundations, Wenatchee, WA.

4. At each recall visit of a denture-wearing patient, the dental clinician should inspect the intaglio surface (underside) of the denture to evaluate the cleanliness of the prosthesis. At the same time, the dental clinician should closely evaluate the denture-bearing areas, such as the maxillary palate, edentulous ridges, and buccal vestibule, for any signs of infection or inflammation. One of the more common complications seen in removable partial denture–wearing patients is a condition known as denture stomatitis. Denture stomatitis is an inflammatory condition that is typically painless and asymptomatic. In most cases, the patient does not even know he or she is suffering from the condition. Rather, it is often an incidental finding that is discovered during a routine dental examination. The causative factor is an overgrowth of *Candida* spp. (a type of fungus). The presence of denture stomatitis should be a red alert that the patient either is neglectful in practicing routine self-care measures or having difficulty in maintaining the cleanliness of the oral cavity and/or the denture. The characteristic presentation of denture stomatitis is a puffy, erythematous mucosal lesion that follows the outline of the denture (**Figure 16-30**). Dental practitioners should be vigilant for the characteristic appearance of denture stomatitis and, if present, should make every effort to educate and forewarn the denture-wearing patient of the possibility of denture stomatitis. If the denture-wearing patient is suffering from denture stomatitis, the condition can be reversed with resumption of good mechanical plaque control of the remaining teeth and the denture along with appropriate denture-wearing habits. In addition, topical antifungal agents (e.g., 2% ketoconazole in Orabase) may be prescribed. The denture may also need to be relined, refitted, or replaced, depending on the fit and retention of the prosthesis.

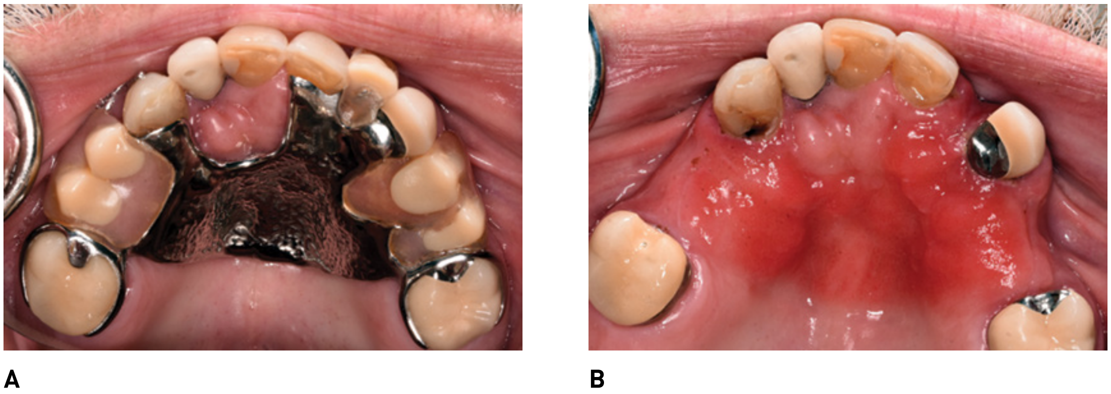

Figure 16-30 **Denture Stomatitis. A.** Patient wearing removable partial denture**. B.** Same patient without the denture in the mouth. Note the reddish, inflammatory mucosal lesion that follows the outline of the overlying removable partial denture. This is pathognomonic of denture stomatitis.

Section 4

Local Factors That Cause Direct Damage

This section discusses a few local contributing factors that may actually cause direct damage to the periodontium. These factors also may alter the progress of periodontal disease at individual sites. Some local contributing factors that can directly damage the periodontium include food impaction, patient habits, and faulty restorations or appliances.

In some patients, habits such as tongue thrusting, mouth breathing, or the improper use of toothbrushes, toothpicks, and other dental cleaning aids can cause direct damage to the periodontium. Most oral injuries are committed inadvertently without malingering intent and without patients realizing that they are performing the habit. However, some patients deliberately damage their own oral tissues with premediated, malingering intent. These types of injuries are known as **factitious injuries** and may be seen in patients suffering from maladjusted cognitive and behavioral processes.

In the context of damage to the periodontium, **malingering** is defined as the intentional injury to the tissues by the patient to deliberately feign or exaggerate a physical or psychological symptom with the goal of receiving a reward. The motives for intentional trauma may include such incentives as insurance settlement, avoidance of work, or to seek sympathy from others. Malingering is difficult to diagnose because patients do not admit to causing their injury. In this case, proper consultation with a mental health care expert may be warranted.

1. **Direct Damage Due to Food Impaction**
   a. **Definition. Food impaction** refers to the forceful wedging of food (such as pieces of tough meat) between teeth during mastication, trapping the food in the interdental area (**Figure 16-31**). Food impaction commonly occurs when the opposing teeth come together to force food into the interdental area. This forceful wedging of food may trigger a gingival inflammatory response (**Figure 16-32**).

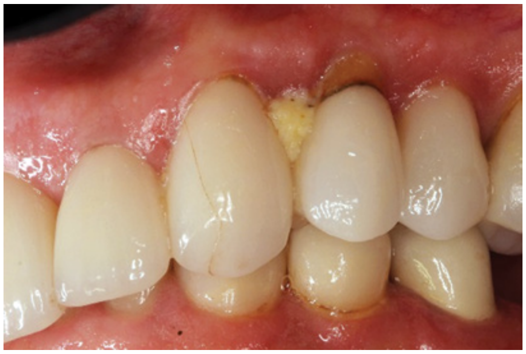

Figure 16-31 **Food Impaction.** Note the wedging of food into the embrasure space (interdental space) by occlusal forces in between the canine and first premolar.

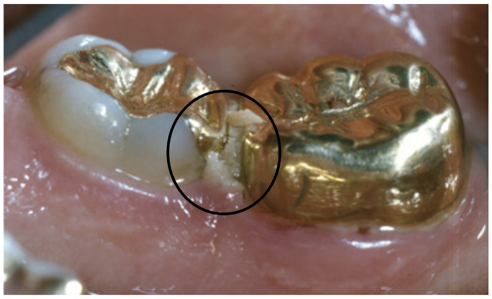

Figure 16-32 **Food Impaction.** Note the food impaction between the two molar teeth. As this patient chews food, the food is forced between these teeth and produces direct damage to the periodontium. Additionally, note the edematous papilla associated with the food impaction site.

Courtesy of Dr. Don Rolfs, Periodontal Foundations, Wenatchee, WA.

b. **Effect of Food Impaction**
   1. Food forced into a tooth sulcus can strip the gingival tissues away from the tooth surface and contribute to periodontal breakdown in addition to the more obvious danger of serving as nutrients for tooth decaying bacteria.
   2. Food impaction not only damages the gingival tissues directly but can also lead to alterations in gingival contour that make interdental areas more difficult to clean.

c. The most conservative, noninvasive approach to managing food impaction is to educate the patient on the daily use of a interdental brush, waxed floss, irrigating syringe, or water irrigation device. If the most conservative, noninvasive approach does not alleviate the patient's chief concern, then consultation with the dentist for restorative assessment or a specialist such as an orthodontist (to realign and reposition the teeth) may be warranted.

2. **Direct Damage from Patient Habits and Oral Piercings**

   a. **Improper Use of Plaque Biofilm Control Aids.** Improper use of plaque biofilm control aids can result in direct damage to the gingival tissues, causing alteration of the natural contours of the tissues (**Figure 16-33**).

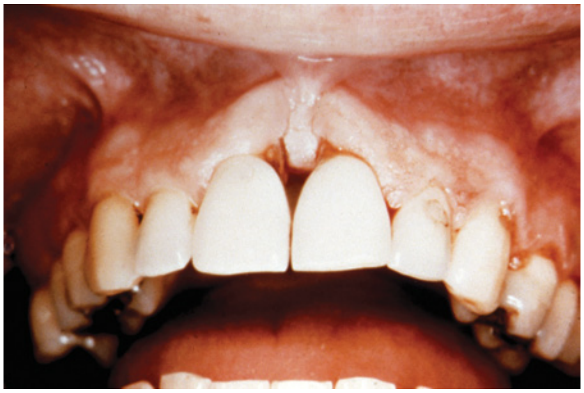

Figure 16-33 **Misuse of Toothpick.** The interdental papilla between the two central incisors has been destroyed by the patient's habit of repeatedly forcing a toothpick between the teeth. This damage to the papillae is an example of direct damage to the periodontium.

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   b. **Tongue Thrusting.** **Tongue thrusting** is the application of forceful pressure against the anterior teeth with the tongue (**Figure 16-34**).

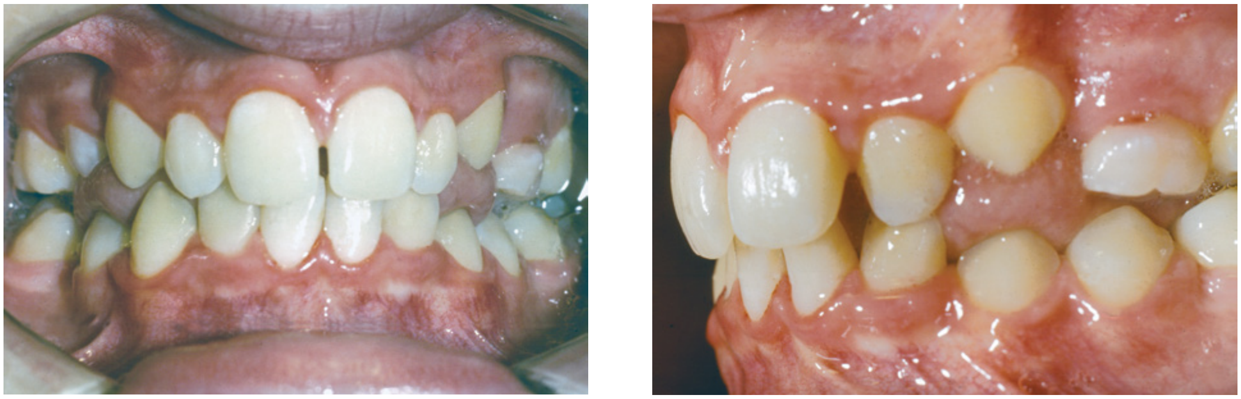

Figure 16-34 **Tongue Thrust.** Pictured on the left, the facial view of a patient with a tongue thrust. As this patient swallows, the patient applies lateral pressure with her tongue against the teeth. The right-hand photo shows a side view of the tongue thrust. The tongue is visible in the canine region of the mouth as the patient presses her tongue forward when swallowing.

Courtesy of Dr. Don Rolfs, Periodontal Foundations, Wenatchee, WA.

1. Tongue thrusting is often the result of an abnormal tongue positioning during the initial stage of swallowing.
2. This oral habit exerts excessive lateral pressure against the teeth and may be traumatic to the periodontium.

c. **Mouth Breathing.** **Mouth breathing** is the process of inhaling and exhaling air primarily through the mouth, rather than the nose, and often occurs while the patient is sleeping. Mouth breathing has a tendency to dry out the gingival tissues in the anterior region of the mouth.

d. **Traumatic Toothbrushing.** **Traumatic tooth brushing** is the aggressive, forceful use of a toothbrush in a horizontal or rotary fashion (**Figure 16-35**). This trauma may be a result of either improper toothbrushing habits or overzealous toothbrushing practices.

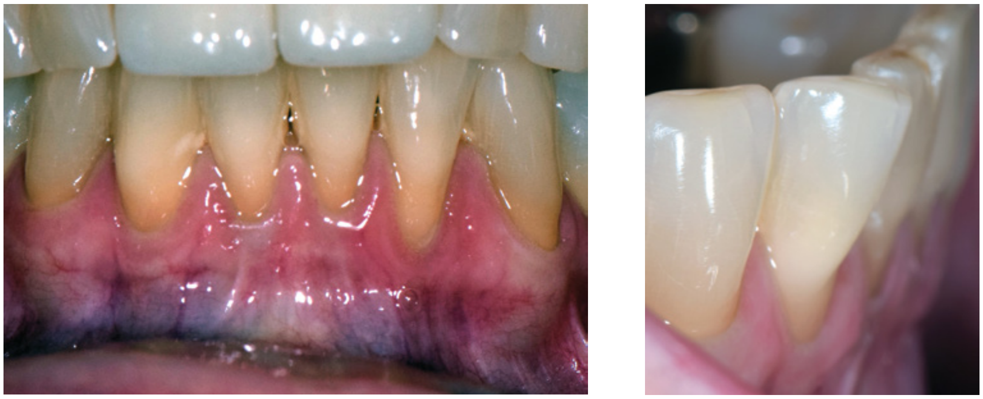

Figure 16-35 **Traumatic Toothbrushing** This patient overzealously brushes her teeth with a hard-bristle toothbrush. Although overzealous toothbrushing has resulted in minimal gingival inflammation and plaque accumulation, it has also traumatized the periodontal tissues and caused gingival recession. Note how thin the gingival tissue appears from the facial and the lateral views.

Courtesy of Dr. Hawra AlQallaf, Indianapolis, IN.

1. While traumatic toothbrushing carries a possible risk of harming the gingival tissues (in the form of gingival recession), equally important is the effect of traumatic toothbrushing on the tooth itself. Take for instance the potential impact of the abrasive ingredients in many of the common over-the-counter toothpastes. Abrasives (such as hydrated silica, calcium carbonate, alumina, sodium metaphosphate, and dicalcium phosphate dehydrate) are regular ingredients added to toothpaste formulations to help efficiently remove stain and plaque during toothbrushing. Reputable toothpaste companies have developed toothpaste formulations that add just enough abrasive ingredients to safely and gently clean teeth without causing any irreversible damage. Further, sound non-carious enamel is not susceptible to abrasion due to its highly mineralized content. On the other hand, when exposed to the oral environment, dentin and cementum may be highly susceptible to irreversible breakdown due to the lower mineralized content relative to enamel. Thus, along with actively monitoring the patient's toothbrushing habits, toothbrushing technique, and type of toothbrush used (soft bristle toothbrush vs. hard bristle toothbrush), the dental clinician should make every effort to help the patient select the right type of toothpaste that does not carry with it a risk of harming the teeth and the surrounding periodontium.
2. If left unabated, traumatic toothbrushing may lead to gingival recession with concomitant loss of alveolar bone on the labial side. This type of root-exposed defect from the cementoenamel junction (CEJ) apically is known as a **dehiscence** (**Figure 16-36**).
3. A dehiscence should not be confused with another type of bony defect known as a fenestration. A **fenestration** is a "window" of bone loss bordered by alveolar bone on its coronal aspect (Figure 16-36). Unlike a dehiscence, a fenestration is not commonly associated with gingival recession.
4. A dehiscence or a fenestration may affect either a natural tooth or a dental implant.

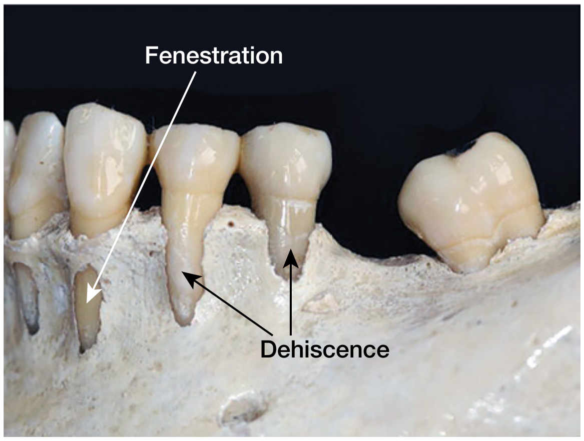

Figure 16-36 **Fenestration and Dehiscences.** A fenestration—a "window" of bone loss—on the facial aspect of the mandible. Note that the marginal bone is intact in the fenestrated area. Dehiscences, pictured here, are V-shaped defects apical to the cementoenamel junctions extending through the marginal bone.

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e. **Oral Jewelry.** Body piercing is the placement of a foreign object through tissue to add to an individual's esthetics or enhance an individual's culture or identity. Oral jewelry is a type of body piercing that has enjoyed growing popularity, especially among the younger generation. Broadly, oral jewelry placed *in* the mouth is classified as intraoral piercings (**Figure 16-37** and 16-38), whereas oral jewelry placed *around* the opening of the mouth is known as perioral piercings (Figure 16-39). Both intraoral and perioral piercings are not without risks. Systemically, they can increase the risk of disease transmission (such as endocarditis, hepatitis, and HIV), trigger hypersensitivity reactions to the type of material composition of the oral jewelry, or cause nerve damage that can cause either paresthesia or anesthesia to the affected body part (e.g., the tongue or lip). Intraorally, they pose risks to an individual developing dental and/or periodontal problems. Further, the longer the piercings are worn, the higher the risk of these complications.

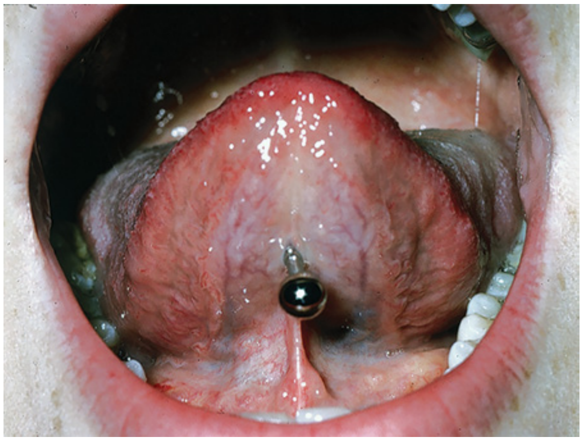

Figure 16-37 **Intraoral Piercing.** The metal tongue barbell, pictured here, is a popular object placed in a pierced tongue, usually worn for esthetic, cultural, or identity purposes. However, intraoral piercings can pose dental and periodontal complications that can compromise the health of the oral cavity.

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1. Intraoral piercings (such as a tongue piercing) or perioral piercings (such as a lip or cheek ring) can cause direct mechanical damage by repeatedly traumatizing the periodontal tissues. Common adverse events associated with oral piercing include gingival recession, infection, swelling, bleeding, tooth fractures, and allergic reactions. **Figures 16-38** and **16-39** illustrate the amount of damage an oral piercing can inflict on the periodontium.

2. Recent microbial studies have shown that individuals with oral piercings tend to exhibit higher levels of periodontal pathogens, such as *Eikenella corrodens*, *Streptococcus oralis*, *Treponema denticola*, and *Prevotella intermedia*.[4,5] This may be because an oral piercing, itself, is an unnatural artificial surface that can serve as a nidus of bacterial colonization (similar to how a restorative overhang is a potential hospitable habitat for pathologic bacteria). Consequently, the piercing directly disrupts microbial symbiosis within the oral cavity, thereby disturbing microbial homeostasis and creating a dysbiosis microbial environment. Further, the bacteria that first accumulated around the piercing can potentially migrate to different areas of the oral cavity like the teeth and surrounding periodontal tissue.
3. Thus, along with the possible mechanical trauma that an oral piercing can inflict on the periodontal tissue, an oral piercing may be a hospitable niche for pathologic bacteria. The case depicted in Figure 16-38 illustrates the combined destructive effects of mechanical trauma and bacteria-induced periodontal disease inflicted by an intraoral piercing.
4. Hence, all members of the dental team should be at the forefront of educating patients of the possible deleterious complications of such piercings so as to prevent exacerbating problems and deliver proper oral education. Ideally, oral jewelry–wearing individuals should be motivated to remove the piercing before the damage is done. If such individuals are unwilling to remove the oral jewelry, the dental practitioner should foreworn them that the piercing will require constant attention and monitoring.

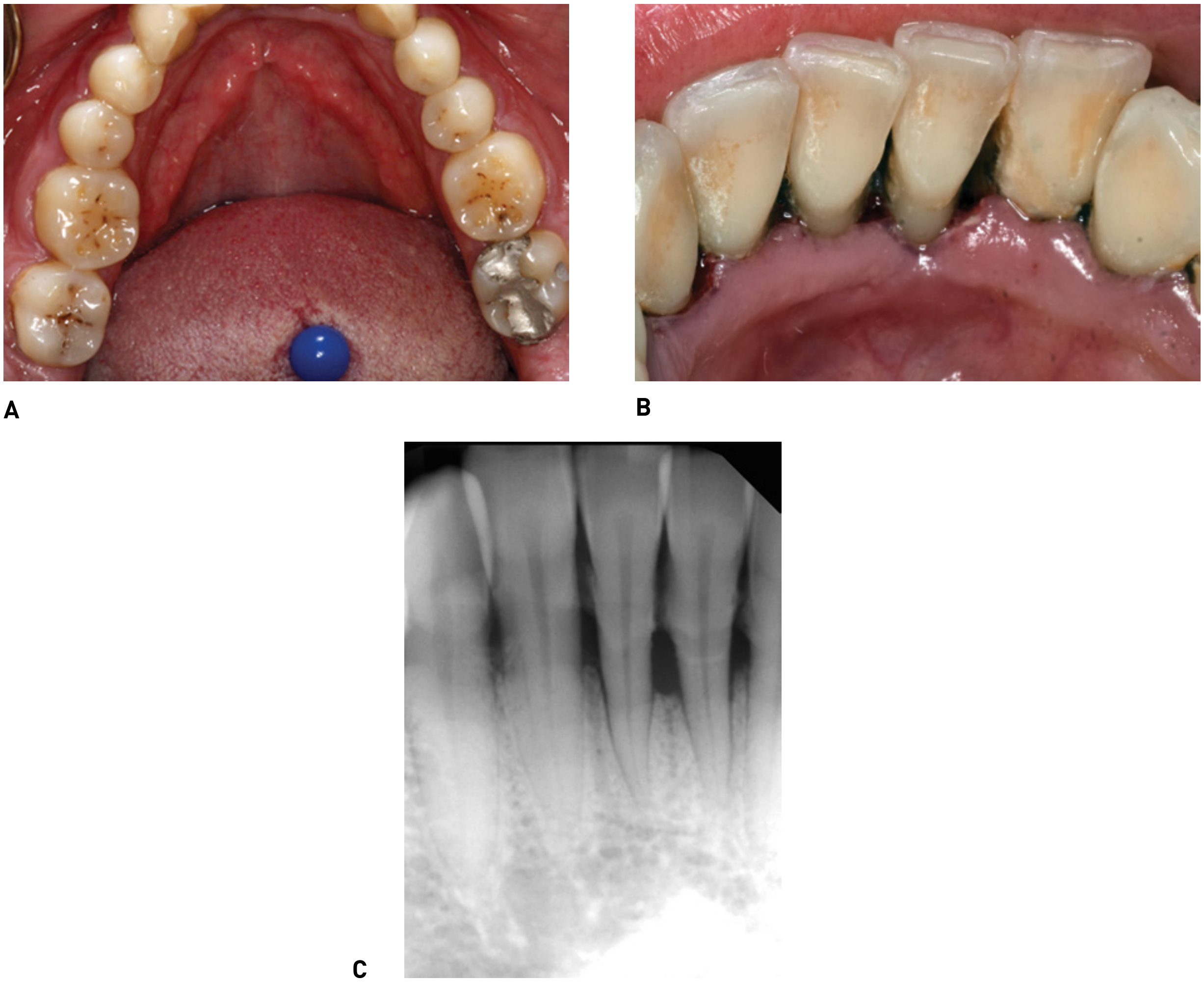

Figure 16-38 Periodontal Complications Associated with an Intraoral Piercing. This 26-year-old patient reported wearing an intraoral piercing (a "tongue ring") for the past 10 years. **A.** Photograph of the intraoral piercing on the tongue. **B.** Close-up view of the lingual surface of the mandibular anterior sextant directly opposing the intraoral piercing. Note the soft tissue destruction present on the lingual of the mandibular anterior teeth and the accumulation of supragingival calculus deposits. **C.** Radiographic evidence of pathologic bone loss. Note the heavy deposits of subgingival calculus. From this case, it is evident that the intraoral piercing not only contributed to the gingival recession, but also contributed to the patient's periodontitis due to its plaque-retentive features.

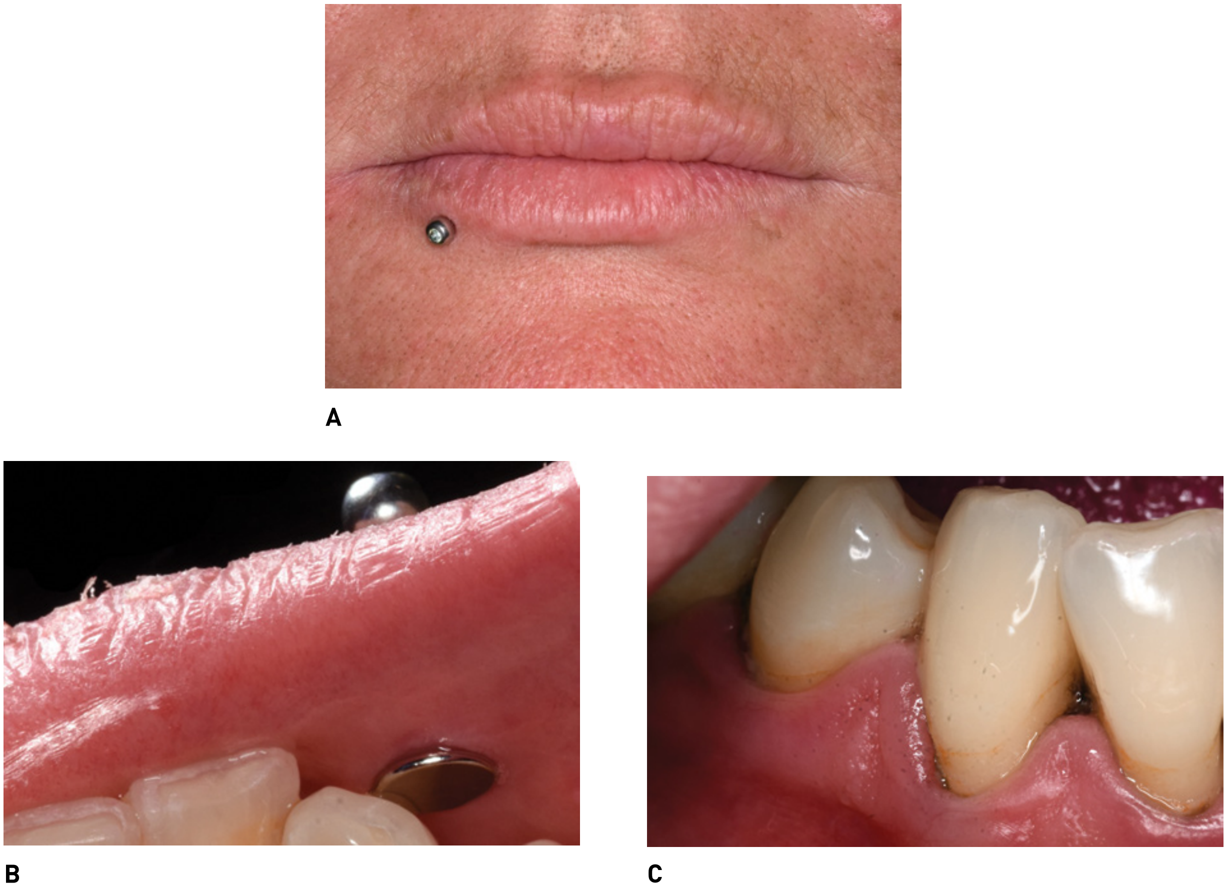

Figure 16-39 **Periodontal Complications Associated with a Perioral Piercing.** This 34-year-old patient presented with the chief complaint, "I want to do something to cover up my canine root" (patient specifically pointed to the mandibular right canine). At the time of the appointment, patient presented with a perioral piercing and reported wearing the piercing for the past 8 years. **A.** Extraoral facial view of the perioral piercing. **B.** Intraoral occlusal view of the perioral piercing. Note the proximity of the oral end of the perioral piercing to the affected mandibular canine. **C.** Intraoral facial view of the mandibular right canine. Note the gingival recession around the affected canine. Thus, from these photos, it is clear the perioral jewelry is not as benign as previously assumed. Rather, it can play a direct role in mechanically traumatizing the tissues and causing direct permanent damage.

3. **Direct Damage from Occlusal Forces**
    a. **Trauma from Occlusion**
        1. Direct damage to the periodontium can result from excessive occlusal (or biting) forces on the teeth.
        2. When excessive occlusal forces cause damage to the periodontium, it is referred to as **trauma from occlusion**. **Table 16-2** summarizes definitions of some terms used to describe trauma from occlusion.
            a. When trauma from occlusion occurs, some alveolar bone resorption can result simply because of increased pressure placed on the surrounding alveolar bone.
            b. When there is loss of some alveolar bone due to trauma from occlusion, there can be a more rapid destruction by any existing periodontitis.
        3. A thorough clinical and radiographic exam can frequently reveal signs of trauma from occlusion.
            a. Some of the clinical signs of trauma from occlusion that have been reported include the following:
                1. Tooth mobility.
                2. Sensitivity to pressure.
                3. Migration of teeth.
            b. Some of the radiographic signs of trauma from occlusion that have been reported include the following:
                1. Enlarged, funnel-shaped PDL space.
                2. Angular alveolar bone resorption.

4. Trauma from occlusion has been classified in the dental literature for many years as either primary trauma from occlusion or secondary trauma from occlusion.

Table 16-2 Terms Associated with Trauma from Occlusion

Term	Definition
Trauma from occlusion	Injury to the periodontium resulting from excessive occlusal forces
Primary trauma from occlusion	Injury to a healthy periodontium resulting from excessive occlusal forces
Secondary trauma from occlusion	Injury from normal or excessive occlusal forces applied to a periodontium previously damaged by periodontitis

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a. **Primary trauma from occlusion** (also known as primary occlusal trauma) occurs when a greater than normal occlusal load is applied to a tooth with a sound, healthy, noninflamed periodontium with no previous history of periodontal breakdown (**Figure 16-40**).

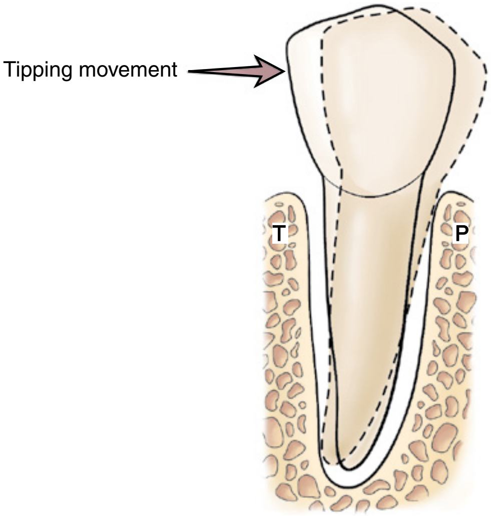

Figure 16-40 Primary Trauma from Occlusion. Primary trauma from occlusion refers to excessive, abnormal forces applied to a tooth with normal supporting structures. In this illustration, the excessive, abnormal force is being applied in a lateral direction. Tipping of a tooth within the socket due to the lateral occlusal forces often accompanies trauma from occlusion. This tipping can result in areas of pressure and tension within the PDL. In the illustration, "P" indicates an area of pressure in the PDL and alveolar bone. The letter "T" indicates an area of tension. Bone under pressure tends to undergo resorption.

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1. Examples of causes of primary trauma from occlusion include inadvertent placement of a high restoration or insertion of a fixed bridge or partial denture that places excessive force on periodontally healthy supporting teeth.
2. The changes seen in primary occlusal trauma include a wider PDL space, tooth mobility, and even tooth and jaw pain. These changes are reversible if the trauma is removed.

b. **Secondary trauma from occlusion** (also known as secondary occlusal trauma) occurs when normal or excessive occlusal load(s) are applied on a periodontally damaged tooth already weakened by periodontitis, thus leading to more harm to an already damaged periodontium.
   1. Secondary trauma from occlusion occurs to a tooth in which the surrounding periodontium has experienced apical migration of the junctional epithelium, loss of connective tissue attachment, and loss of alveolar bone. In this type of trauma, the periodontium was unhealthy prior to experiencing excessive occlusal forces.
   2. A tooth with an unhealthy, inflamed periodontium that is subjected to excessive occlusal forces is thought to be more susceptible to more rapid bone loss and pocket formation.
   3. Teeth can be tipped laterally easily when subjected to lateral occlusal forces. These tipping forces frequently accompany trauma from occlusion and can create areas of pressure and tension within the PDL that are transmitted to the bone. **Figure 16-41** illustrates how this tipping can occur. As alveolar bone loss progresses, this lateral tipping becomes even more likely because of the longer lever arm created by the part of the tooth out of the bone compared to the part of the tooth encased in bone.
   4. Teeth with reduced alveolar bone support can have additional damage to the periodontium because of the tipping action of lateral forces placed on the teeth. **Figures 16-42** and **16-43** show radiographs of teeth that have been subjected to secondary occlusal trauma.

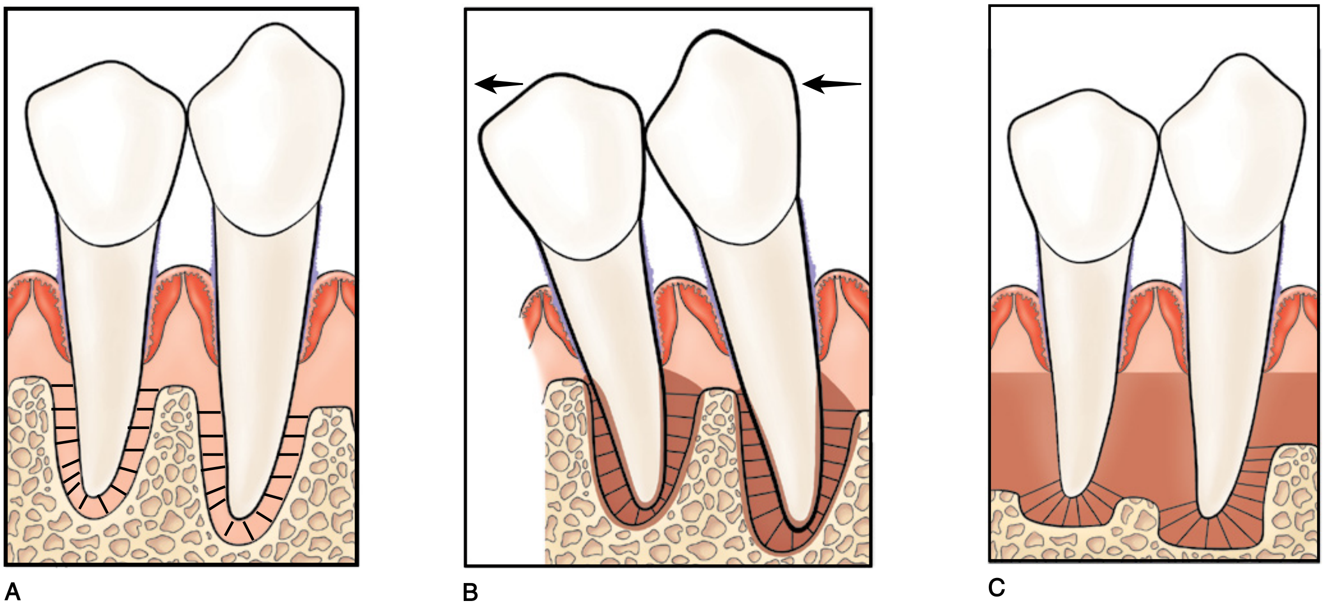

Figure 16-41 Secondary Trauma from Occlusion: Damage to Teeth with Existing Periodontitis. A. Teeth with reduced bone height caused by a preexisting periodontitis. **B.** Lateral forces applied to teeth. In response to these forces, the teeth tip laterally. **C.** Additional bone loss to the periodontium occurs as a result of pressure on bone from the lateral forces. The additional bone loss that occurs from secondary trauma from occlusion can further compound the periodontal destruction that occurs due to periodontitis.

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b. **Parafunctional Occlusal Forces**
   1. A series of other terms has been used to describe occlusal forces. Two of these terms are functional and parafunctional occlusal forces.
      a. **Functional occlusal forces** are the normal forces produced during the act of chewing food (also known as mastication).
      b. **Parafunctional occlusal forces** result from unconscious or conscious tooth-to-tooth contact made when not in the act of eating (**Figure 16-44**).

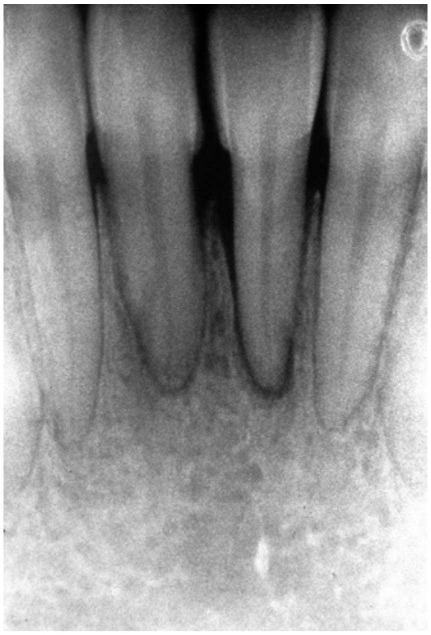

Figure 16-42 Radiographic Evidence of Trauma from Occlusion. Note the dramatic widening of the periodontal ligament space along the lateral root surfaces on the mandibular left central incisor (center tooth on radiograph). The alveolar bone has been destroyed because of the pressure resulting from trauma from occlusion.

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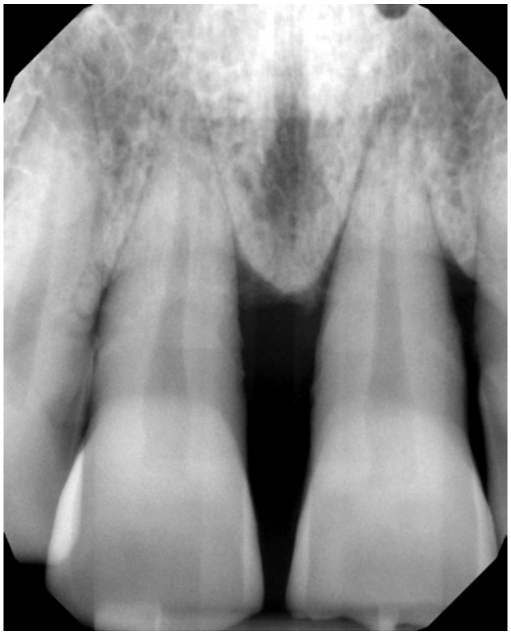

Figure 16-43 Enlarged, Funneling of the PDL Space. The maxillary left central incisor has been subjected to trauma from occlusion. Note the enlarged, funnel-shaped widening in the coronal and apical regions of the PDL space. This is the radiographic hallmark of occlusal trauma.

1. Examples of these parafunctional habits are clenching of the teeth together as a release of nervous tension or grinding the teeth together for the same release.
   a. **Clenching** is the continuous or intermittent forceful closure of the maxillary teeth against the mandibular teeth.
   b. **Bruxism** is forceful grinding of the teeth.
   c. These parafunctional habits can occur without the person having conscious knowledge of the habit. Some individuals exhibit these habits while asleep.
2. Parafunctional habits can exert excessive force on the teeth and to the periodontium.

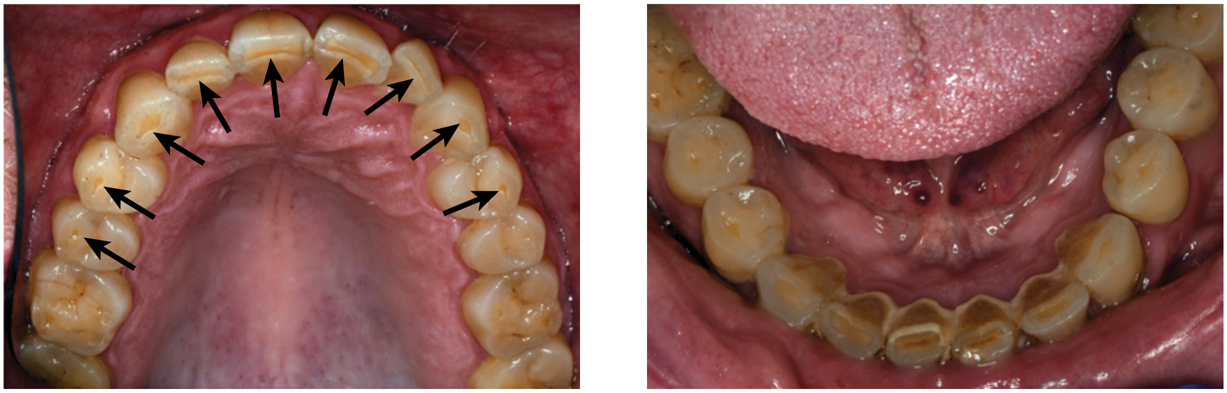

Figure 16-44 Parafunctional Habit. Patient reports a history of prolonged clenching on a daily basis due to workplace environment. On both maxillary and mandibular photographs, note the flattened occlusal surfaces of all teeth, presence of wear facets, and enamel wear.

3. The dentist can use several clinical therapies to help control the damage from trauma from occlusion.
   a. When the trauma is a result of a faulty bite (referred to as a faulty occlusion), the dentist can make minor adjustments in the bite by selectively reshaping (also known as selective grinding) the occlusal surfaces of the teeth to minimize the damaging forces and eliminate occlusal interferences. This procedure is called an occlusal adjustment.
   b. When the trauma is a result of bruxism, the dentist can fabricate an acrylic appliance known as an occlusal appliance (also referred to as an occlusal splint or a night guard) that can protect the teeth during part of each day. It should be emphasized that an occlusal appliance does not cure bruxism, but rather intervenes by protecting the teeth and implants (if present) from further wear.
4. **Direct Damage Caused by Normal Anatomic Factors.** Typically, normal anatomic factors are not injurious to the periodontium. One exception, however, is a high frenal attachment.
   a. A frenum is a thin fold of mucous membrane with enclosed muscle fibers that attaches the lips to the alveolar mucosa and underlying periosteum (**Figure 16-45**). A frenum's primary function is to provide stability to the lips, cheeks, and tongue.

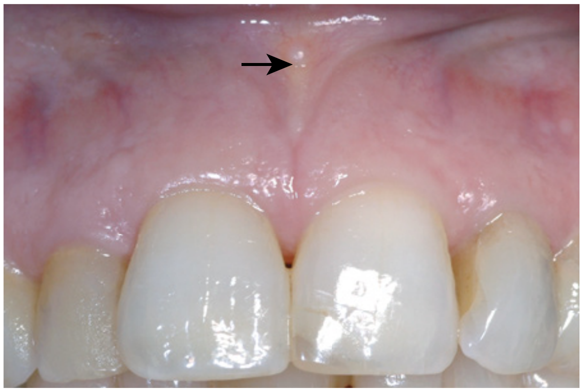

Figure 16-45 Normal Frenal Attachment. Note the healthy band of keratinized tissue separating the maxillary labial frenum from the gingival margin of the maxillary central incisors. Also note the lack of tissue blanching or "pulling" of the gingival margin as tension is applied to the upper lip.

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      1. Intraorally, there are four major oral frena: (1) the maxillary labial frenum (connects the upper lip to the gingiva); (2) the mandibular labial frenum (connects the lower lip to the gingiva); (3) the buccal frenum (connects the cheek to the gingiva); and (4) the mandibular lingual frenum (connects the tongue to the gingiva).
      2. Depending on the location, the frenum provides stability to the upper lip, lower lip, cheek, or tongue.
   b. The frenum can be clinically examined by applying light tension to the upper lip, lower lip, or cheek.
   c. A frenum can become a significant problem if tension from lip movement pulls the gingival margin away from the tooth (**Figures 16-46** and **16-47**). An abnormal frenal attachment located in close proximity to the gingival margin will distend the gingival sulcus (as the muscular fibers in the frenum repeatedly pull on the gingival margin). This is known as a **frenal pull**. Frenal problems occur most often on the facial surface between the maxillary and mandibular central incisors and in the canine and premolar areas.

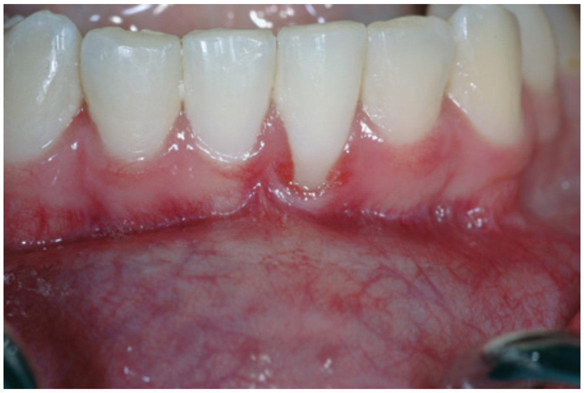

Figure 16-46 **Direct Periodontal Damage Caused by a High Frenal Attachment.** Note the high frenal attachment between the central incisor teeth. Tension of the lower lip pulls down the gingival margin and contributes to the gingival recession seen on the mandibular left central incisor. Continued tension by the frenum, if not removed, will lead to further progression of tissue breakdown.

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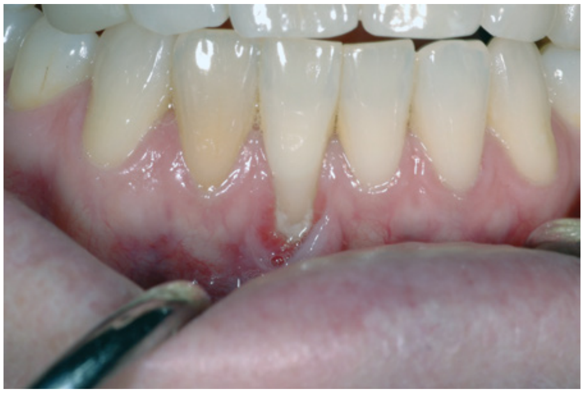

Figure 16-47 **Abnormal Frenal Attachment.** Note the close proximity of the frenal attachment to the gingival margin of the right central incisor. This patient complains of tooth sensitivity whenever he brushes the exposed root surface, so he does not brush the area very well. Consequently, plaque biofilm has accumulated over the root surface and the marginal gingiva has become severely inflamed.

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d. The presence of any abnormal frenal attachments can be corrected through a surgical procedure known as a frenectomy. (Chapter 30 provides a more in-depth discussion of the indications and the steps of the frenectomy procedure.) However, if the abnormal frenal attachment is not surgically eliminated, the frenum will continually pull down the marginal gingiva and make the exposed root surface more susceptible to plaque biofilm accumulation. This may lead to root caries and/or periodontal disease. If not removed, the health of the tooth and the periodontium may be jeopardized.

Chapter Summary Statement

Although local contributing factors per se do not initiate the onset of disease, they can certainly elevate the risk of developing periodontal disease or aggravate the extent and severity of an already preexisting gingivitis or periodontitis. Hence, as will be discussed in Chapter 19, during each routine dental evaluation every effort must be made by the dental clinician to closely scrutinize for the presence of any local contributing factors. If present, the local contributing factor(s) should be eliminated or its effects should be minimized before the disease progresses to a stage that results in permanent damage to the dentition and surrounding periodontium.

Section 5

Focus on Patients

Clinical Patient Care

CASE 1

Examination of a patient reveals gingivitis. In addition, the patient has generalized calculus deposits and numerous restorations with overhangs. What steps might be necessary to bring the gingivitis under control in this patient?

CASE 2

Examination of a patient reveals periodontitis. The patient also has a severe tooth clenching habit. Explain how the tooth clenching habit could be related to the progress of the periodontitis.

Evidence in Action

Case 1: In a dental hygiene journal, you find an article that refers to mature dental plaque biofilm. Explain what the author means by this term *mature dental plaque biofilm*.

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CHAPTER 17

Tobacco, Smoking, and Periodontal Disease

- Section 1 Introduction
- Section 2 Nicotine Dependence and Addiction
  - Biochemical Basis Underlying Nicotine Dependence and Addiction
  - Chemical Composition of Tobacco: A Storehouse of Thousands of Chemicals
- Section 3 Categories of Smoking Delivery Products
  - Combustible Versus Noncombustible Delivery Systems
  - Prevalence of Combustible and Noncombustible Usage in the United States
- Section 4 Smoking as a Risk Factor for Periodontal Disease
  - Epidemiology of Smoking Use Among Periodontal Patients
  - Effects of Combustible Tobacco on the Periodontium
  - Effects of Noncombustible Tobacco on the Periodontium
  - Effects of Noncombustible Nicotine on the Periodontium
  - Effects of Smoking on Periodontal Therapy
- Section 5 Cannabis as a Risk Factor for Periodontal Disease
  - Overview of Cannabis
  - Epidemiology of Cannabis in Periodontal Patients
  - Effects of Cannabis on the Periodontium
- Section 6 Smoking and Peri-Implant Disease
  - The Impact of Smoking on Dental Implants
- Section 7 Smoking Cessation for the Periodontal Patient
  - Effects of Smoking Cessation on the Periodontium
  - Smoking Cessation Counseling in Periodontal Therapy
  - A User-Friendly Model for Counseling the Periodontal Patient
- Section 8 Focus on Patients
  - Clinical Patient Care
  - Clinical Evidence in Action: Clinical Relevance

CLINICAL APPLICATION

Although it is well established that smoking is a risk factor for many debilitating systemic and oral diseases, smoking continues to be a grave and persistent threat that overburdens the global public health system. In the United States alone, smoking is responsible for half a million deaths annually due to tobacco-associated cancers, emphysema, heart disease, and stroke. Equally sobering is that over 50 million Americans struggle with their addiction and dependence to nicotine. On top of this is the mass-marketing of new noncombustible nicotine delivery systems (e.g., e-cigarettes) and noncombustible tobacco delivery systems (e.g., "heat-not-burn" tobacco products)

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that have been heavily commercialized and promoted by the tobacco industry. Hence, being at the forefront of health care delivery, dental hygienists can play a leading role in blunting the impact of the global smoking epidemic by keeping abreast of the rapidly evolving classes of newer tobacco delivery systems. Additionally, dental hygienists are well positioned to take the lead in educating patients on the potential hazards associated with traditional combustible products (i.e., conventional cigarettes), newer noncombustible products, and even cannabis (marijuana). Any help that we, as health care practitioners, can offer to a smoker will not only inevitably diminish the likelihood of smoking-related health complications but also have an enormous positive impact on the individual's livelihood.

LEARNING OBJECTIVES

- Discuss the implications of smoking and use of other tobacco products on periodontal health status.
- Describe the different categories of tobacco/nicotine delivery systems and provide examples of each.
- Discuss the implications of smoking on the host response to periodontal disease.
- Discuss the implications of cannabis on the host response to periodontal disease.
- Discuss the effects of smoking on periodontal treatment outcomes.
- Discuss current theories as to why smokers have more periodontal disease than nonsmokers.
- Explain why tobacco cessation counseling is a valuable part of patient care in the dental setting.
- Value the importance of providing smoking cessation counseling as a routine part of periodontal treatment.

KEY TERMS

Combustible tobacco delivery systems
Noncombustible tobacco delivery system
Noncombustible nicotine delivery system
Dental implant
Environmental tobacco smoke (ETS)
Smoking cessation counseling

Section 1

Introduction

In 2021, the U.S. National Institutes of Health (NIH) published a comprehensive report titled *Oral Health in America: Advances and Challenges* that explores the nation's oral health over the past 20 years.[1] Among the many concerns highlighted in the report is the protracted toll smoking has had on the oral health of Americans over the last several decades, and which is being further exacerbated by the advent of heavily promoted noncombustible nicotine products (e.g., e-cigarettes), noncombustible tobacco products (e.g., "heat-not-burn" tobacco products), and cannabis (marijuana). This is in line with several key points stressed in the 2020 U.S. Surgeon General's Report titled *Smoking Cessation: A Report of the Surgeon General*—namely, that there are many unknowns with respect to the new smoking delivery systems that circumvent our traditional understanding of "smoking." For example, we do not yet know how dangerous these novel products are when used over the long term; we do not know if they are truly effective at helping addicted adults quit smoking traditional cigarettes, or even if some types of smoking products—such as e-cigarettes—are less harmful than traditional cigarettes; nor do we know to what extent they can serve as a "gateway" to the use of combustible cigarettes and/or illicit substances.[2]

Thus, the purpose of this chapter is to investigate in-depth the pathophysiologic mechanisms underlying smoking and smoking addiction, review the new classes of nicotine/tobacco delivery systems that have skyrocketed in popularity over recent years and their impact on systemic and oral health, and explore the biologic consequences of cannabis and its impact on general health. Finally, this chapter concludes with an important section that outlines a user-friendly model to proactively counsel smokers on the link between smoking and oral/systemic diseases and motivate them to choose the best cessation plan that would put them onto the road to recovery.

Section 2

Nicotine Dependence and Addiction

Biochemical Basis Underlying Nicotine Dependence and Addiction

Smoking-related physical addiction and psychological dependence lies in the addictive properties of one of tobacco's major chemical components: nicotine.

1. By itself, nicotine is not a carcinogen. However, repeated, prolonged use of any form of smoking product, via the *addictive properties of nicotine*, creates the conditions necessary to drastically impair normal physiologic mechanisms and contribute to protracted psychological dependence of the drug.
2. According to the U.S. Centers for Disease Control and Prevention (CDC), nicotine can be as addictive as alcohol, cocaine, or heroin.[3] Prolonged, habitual addiction to smoking can lead to *dependence*—in which a person suffers from acute physiological withdrawal symptoms and adverse emotional changes when not taking the drug. Symptoms that emerge when prolonged and excessive smoking consumption is discontinued include irritability, mood and sleep difficulties, decreased appetite, cravings, restlessness, and/or various forms of physical discomfort that peak within the first week after quitting and last for several weeks.[3,4]
3. Smoking dependence occurs when the brain adapts to large amounts of the nicotine binding to acetylcholine receptors on neurons. This interaction facilitates the release of the neurotransmitter known as dopamine, which provides an intense feeling of pleasure and reward. Over time, prolonged, repeated exposure to nicotine likely results in desensitization, which leads to greater dependence to higher doses of nicotine or more frequent consumption of nicotine to compensate for the desensitization.[2–4]
4. Nicotine is hazardous to pregnant women and developing fetuses because it can potentially disrupt normal fetal development and have potentially lasting consequences on brain, heart, lung, and other major organ function of the newborn. As such, the U.S. Food and Drug Administration (FDA) classifies nicotine as a Pregnancy Category D drug, which means that there is positive evidence of human fetal risk, based on adverse reaction data from investigation or marketing studies in humans, to the drug. Unless the benefits outweigh the risks (i.e., life-threatening conditions to the mother or developing fetus), nicotine, and any other Pregnancy Category D drug, is absolutely contraindicated in pregnant mothers.

Chemical Composition of Tobacco: A Storehouse of Thousands of Chemicals

Tobacco is a versatile and chemically active leafy plant that grows in all corners of the planet. As the tobacco grows, it readily absorbs various chemicals from soil and fertilizer, including cadmium, lead, arsenic, and nitrates, which can be absorbed through the respiratory lining when consumed. Furthermore, more than 7,000 toxins are present in cigarette smoke, including hazardous toxins such as carbon monoxide; oxidizing radicals; carcinogens (e.g., nitrosamines); and addictive psychoactive substances, such as nicotine, that "feed" the dependence and addiction to smoking.[5]

1. Nicotine is the major addictive chemical compound found in all tobacco-containing products and nicotine-containing products. Although nicotine is a highly addictive compound, it is only one of the many thousands of harmful ingredients found in all tobacco-containing products.
2. When tobacco is heated or burned, it releases a cocktail of noxious and harmful by-products, such as benzene, carbon monoxide, carbon dioxide, nitrogen oxides, ammonia, hydrogen cyanide, volatile sulfur containing compounds, and volatile hydrocarbons, that have the potential to cause many types of cancer (carcinogenic), including lung cancer, nasopharyngeal cancer, oral cancer, and other forms.
3. Although not a chemical compound per se, tar is the sticky, resinous, semi-combusted residue that adheres to the epithelial lining of the oral cavity, teeth, lungs after the tobacco leaves within the tobacco-containing product are burned. Unlike nicotine, tar is not addictive. However, tar can be dangerous when it builds up inside the lungs and impairs normal respiratory functions. Tar is also associated with the dark discolored stains found on teeth of smokers, discoloration of the tongue, and possible halitosis due to the "burning" of the tobacco product. All tobacco-containing products can generate tar.

Section 3

Categories of Smoking Delivery Products

Combustible Versus Noncombustible Delivery Systems

For centuries, smoking involved burning (combusting) tobacco leaves to release the complex cocktail of ingredients upon inhalation. However, over the past decade the landscape has dramatically changed with newer, faster, and easier systems that more readily deliver nicotine directly to the consumer. The three main types of delivery systems currently on the market are detailed below and in **Figure 17-1**.

1. **Combustible Tobacco Delivery Systems.** **Combustible tobacco delivery systems** are the traditional method of nicotine delivery. Combustible delivery systems are composed of tobacco leaves that are actively burned upon being lit. The burning of the tobacco leaves creates an aerosol that the user can inhale. Included in this group are conventional cigarette smoking, cigarillos, cigar smoking, waterpipe smoking (hookah), and pipe smoking products.
2. **Noncombustible Nicotine Products.** **Noncombustible nicotine products** (also known as electronic nicotine delivery systems, or ENDS) are a novel nicotine delivery system that neither contains tobacco nor undergoes an active burning process. Rather, ENDS are battery-powered devices composed of a heating element and a container filled with liquid nicotine derived naturally from tobacco (referred to as the "e-liquid" or "e-juice") or synthetic liquid nicotine. The heating element electronically heats up the liquid nicotine, which then creates nicotine-containing vapor that is then inhaled by the consumer. Included in this category are e-cigarettes (also known as e-cigs), vaping devices, and vape pens. Although manufacturers purport that noncombustible nicotine products are safer than conventional combustible tobacco-products, current evidence suggests otherwise.
   a. Because noncombustible nicotine products contain nicotine, they are as addictive as combustible tobacco-products.
   b. The burning of nicotine produces noxious chemicals that are potentially harmful, including volatile organic compounds, carcinogenic chemicals, heavy metals such arsenic, tin, lead, and ultrafine particles that can be inhaled deep into the lungs. Also, flavoring agents (such as diacetyl) added to these products have been linked to lung disease.
   c. Some nicotine vaping manufacturers make the alleged claim that their products do not contain nicotine. However, an analysis of various e-cigarettes from the United States, South Korea, and Poland did not find any evidence to substantiate this claim. In fact, products labeled as "nicotine-free" had detectable levels of nicotine.[6]
   d. A large majority of adult e-cigarette users in the United States use it concurrently with routine cigarette use, rather than in place of cigarettes. In an epidemiological survey examining the prevalence of electronic cigarette use among U.S. adult workers, the dual use of e-cigarettes with other types of tobacco products, most notably combustible tobacco products, was highly prevalent.[7] This is consistent with another survey that found that 1 out of every 3 users of e-cigarettes were also current cigarette smokers.[8] As such, dual use of combustible tobacco and e-cigarettes is a common pattern seen in e-cigarette users. Although some try to use e-cigarettes to quit, others use it in lieu of smoking combustible tobacco in smoke-free areas (like work areas), thus having the effect of sustaining their smoking behavior.[9]
   e. Because nicotine vaping products are relatively new to the market, the scientific community still has much to learn about their long-term health effects. Thus, as of 2022, these products have not received approval from the FDA nor Health Canada as smoking cessation aids.
3. **Noncombustible Tobacco Products.** Noncombustible tobacco products are another novel form of nicotine delivery. They include the following products.
   a. **Heated Tobacco Products That Heat Tobacco Without Combustion**
      1. Commonly referred to as "heat-not-burn" tobacco products.
      2. Unlike noncombustible nicotine delivery systems, the **noncombustible tobacco delivery system** contains tobacco that is heated using a battery-operated electronic device (instead of being ignited and burned, as with a traditional cigarette).

        3. Compared to traditional combustible products, the purported advantage of this product is that because heated tobacco products are not burned, they do not produce as many harmful chemicals.
        4. In 2019, the FDA permitted the sale of Philip Morris's IQOS® device (IQOS® stands for "I Quit Ordinary Smoking"), which was the first product of its kind to be sold in the U.S. market.[10]
            a. Unlike combustible tobacco products that burn tobacco, IQOS® is composed of a battery-operated electronic device and tobacco plug that is wrapped in a special paper, known as a heatstick. When the battery-operated device is turned on, the heatstick heats up to release an inhalable aerosol that contains nicotine, flavoring agents, and other tobacco components.
            b. In 2020, the FDA classified IQOS® as a "modified tobacco product."[11] A modified tobacco product is a special type of tobacco product that has a potential reduced risk and/or reduced exposure from using a tobacco product supported by scientific evidence. Indeed, early evidence seems to suggest that IQOS® produces lower levels of harmful and potentially harmful chemicals compared to regular cigarettes. Although the FDA authorizes the marketing of IQOS® as a modified tobacco product, it does not mean that the FDA deems the delivery system to be safe for use. It also does not mean that IQOS® or other types of heat-not-burn products are FDA approved. (Note that as of the publication of this textbook, the U.S. International Trade Commission imposed a temporary suspension on the sale and import of IQOS® in the U.S. market due to potential patent infringements filed by a rival's heated tobacco product. Nevertheless, IQOS® continues to be marketed and sold to consumers in other countries.)
            c. Although heat-not-burn tobacco products such as IQOS® and noncombustible nicotine products such as e-cigarettes are both battery-operated devices that do not burn tobacco, their similarities end there. Noncombustible nicotine products are in a different category of delivery system because they contain synthetic nicotine without the tobacco, whereas heat-not-burn products contain real tobacco leaves.
    b. **Waterpipe Tobacco Smoking**. Waterpipe tobacco smoking (hookah smoking) falls under the noncombustible tobacco category. Waterpipe tobacco smoking involves the use of a hookah (the waterpipe) that contains charcoal and special tobacco leaves laced with honey, molasses, glycerin, and other flavoring agents. When the charcoal is burned, it heats up the special tobacco leaves, releasing vapors that can be inhaled by the consumer.
    c. **Smokeless Tobacco Products.** Smokeless tobacco products deliver tobacco products through means other than smoking, such as chewing, sniffing, or placing between the teeth, periodontal tissues, and/or buccal mucosa. Included in this group is snuff (a smokeless tobacco that is inhaled through the nose or placed in the mouth to produce saliva) and dissolvable tobacco products, which includes tobacco sold as lozenges, strips, or sticks that look like candy. Like all tobacco products, smokeless tobacco contains hazardous carcinogens and nicotine.

4. Neither noncombustible tobacco nor noncombustible nicotine products have yet to be deemed safe by leading national drug regulatory bodies, such as the FDA and Health Canada, for the following reasons:
    a. Because both delivery systems are still relatively new, there is presently a lack of research that verifies the safety and efficacies of these products. Moreover, there are questions regarding the long-term health impact associated with these products. Preliminary studies to date have found evidence that noncombustible products may have a comparable, if not greater, hazardous impact on systemic health, such as an increased risk of cardiovascular disease,[12] cancer,[13] and diabetes.[14] Equally important are the health consequences from secondhand exposure to these products.
    b. No formal safety requirements exist regarding ingredient disclosure or information on nicotine levels and thresholds of different types of noncombustible tobacco or noncombustible nicotine products. Further, not all noncombustible products have the same nicotine content or concentrations. In fact, the concentrations of nicotine levels vary among different brands of noncombustible products.[15] This raises serious doubt that tobacco and noncombustible nicotine companies are being 100% accurate on their manufacturer labels when it comes to documenting the true quantity of nicotine content in their individual products.
    c. Thus, there needs to be more ongoing research in the health effects of these newer delivery systems when compared to traditional combustible smoking systems.

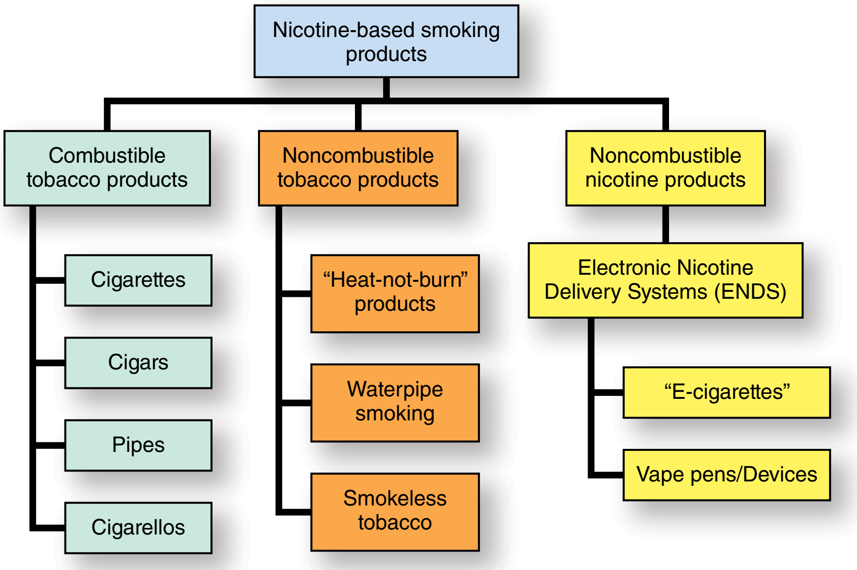
Nicotine-based smoking products
Combustible tobacco products
Noncombustible tobacco products
Noncombustible nicotine products
Cigarettes
Cigars
Pipes
Cigarellos
"Heat-not-burn" products
Waterpipe smoking
Smokeless tobacco
Electronic Nicotine Delivery Systems (ENDS)
"E-cigarettes"
Vape pens/Devices

Figure 17-1 Categories of Nicotine-Based Smoking Products. Nicotine-based smoking products can be classified according to the method that nicotine is delivered upon inhalation: (1) combustible tobacco product, (2) noncombustible tobacco product, and (3) noncombustible nicotine product. Note that cannabis is not included in this chart because it does not contain nicotine. Cannabis will be discussed in Section 5 of this chapter.

Combustible and Noncombustible Usage in the United States

1. In spite of all the evidence that demonstrates the health risks associated with smoking, epidemiologic data from the 2019 National Health Interview Survey (NHIS) estimated that close to 51 million American adults currently use tobacco products.[16] Of these 51 million, about 34 million reported the use of combustible tobacco products, about 10.9 million used e-cigarettes, 8.7 million used cigars, 5.9 million used smokeless tobacco, and 2.4 million used cigars. Combustible products were used by 81% of current tobacco product users.[16]
2. The popularity of smoking is not limited to adults. In the 2021 National Youth Tobacco Survey (NYTS), 2.55 million U.S. middle and high school students reported current (past 30-day) use of tobacco product in 2021.[17] Moreover, among the study population, e-cigarettes were the most used tobacco product, followed by cigarettes, cigars, smokeless tobacco, hookahs, nicotine pouches, heated tobacco products, and pipe tobacco. The emergence of e-cigarette use among adolescents is a new public health threat, especially with the growing concern that e-cigarette use among youth and adolescents can potentially lead them down the path to lifelong nicotine addiction. Further, a U.S. study has shown that e-cigarette use among youths and adolescents may potentially serve as a gateway to future use of combustible tobacco products in later years.[18]

Section 4

Smoking as a Risk Factor for Periodontal Disease

Both combustible and the new noncombustible tobacco/nicotine products potentially have the same negative effects on the tissues of the oral cavity like that seen with other types of tobacco products.[19] Thus, all forms of smoking—whether combustible or noncombustible—adversely affect the oral cavity to the same extent and same degree. Much of smoking's destructive effects on the periodontium centers around its ability to alter the progression, severity, and outcome of the host immunoinflammatory response. Therefore, a comprehensive understanding of how smoking affects inflammation is vital to implementing preventive and therapeutic strategies.

Epidemiology of Smoking Use Among Periodontal Patients

1. Evidence accumulated over the five decades has thoroughly implicated cigarette smoking as a very strong risk factor for periodontal disease.[20–23] Cigarette smoking increases the risk for periodontal disease by at least two to three times.[20]
2. Tooth loss is the ultimate outcome of untreated periodontitis. Smokers are at higher risk for tooth loss due to periodontal disease.[22,24–26]
3. Cigar and pipe smoking are also significant risk factors for attachment loss.[26–28]
4. Smokeless tobacco use is associated with severe recession and loss of attachment to buccal surfaces of teeth where the smokeless tobacco was placed.[29]
5. Several important in vitro studies have revealed that vaping (e-cigarettes) has comparable damaging consequences as cigarette smoking on the periodontium. The available evidence confirms an increased risk of periodontal disease from vaping.[30]

Smoking and Periodontitis

1. A wealth of literature has outlined the role that cigarette smoking has on periodontal disease and treatment, and several reviews cover this topic in detail.[23,31,32]
2. Results from the first National Health and Nutrition Examination Survey (NHANES) demonstrated that smokers have greater periodontal destruction than former and never smokers.[27]
3. More recently, the NHANES III study concluded that approximately half of periodontitis cases could be attributed to either current (42.9%) smoking or former smoking (10.9%).[27,33] Current estimates indicate smoking also increases the prevalence of periodontitis in excess of 20% in younger segments of the adult population.[26]
4. The risk of smoking-related periodontal destruction is dose-dependent with total exposure to cigarette smoking being a widely used measure of dose.[26] Heavy smokers (more than 10 cigarettes/day) have greater odds for more severe attachment loss compared to nonsmokers.[34]
5. The risk of smoking-related periodontal destruction is also duration dependent. As an example, long-term smokers tend to be at higher risk of developing periodontitis compared to nonsmokers. A cohort study found that individuals who started smoking tobacco by age 15 tend to be at higher risk of periodontal disease compared to never smokers or individuals who started smoking at a later period of their lives.[35] The same investigators concluded that the effects of smoking over a prolonged period accumulate through a smoker's lifetime with cumulative damage to the periodontal tissues.
6. In longitudinal maintenance studies, smoking is strongly correlated to higher rates of tooth loss due to periodontal disease even in patients who were consistently receiving regular periodontal maintenance over a 5-year period.[36–38]
7. The Task Force of the 2017 AAP/EFP World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions recognizes that cigarette smoking is a major risk factor for periodontal diseases and peri-implant diseases: (1) smoking increases the rate of progression of periodontitis, (2) it alters the patient's responsiveness to standard therapeutic principles, (3) it negatively impact general health or systemic disease, and (4) it could cause the disease to progress from one stage to the next.[39] Therefore, the clinician should make every effort to assess a patient's smoking status and integrate this pertinent information, along with other risk factors, to formulate the grade of periodontitis. For reference, the reader is referred to Chapter 6 to review the proposed framework for staging and grading periodontitis, according to the 2017 Classification System.

Effects of Combustible Tobacco on the Periodontium

Oral problems associated with combustible smoking include halitosis, dry mouth, dental staining periodontal disease, and cancer (**Figures 17-2** and **17-3**).[22,26,32,33] **Figure 17-4** illustrates the pathophysiologic mechanisms that underlie the detrimental effects of smoking on the host.

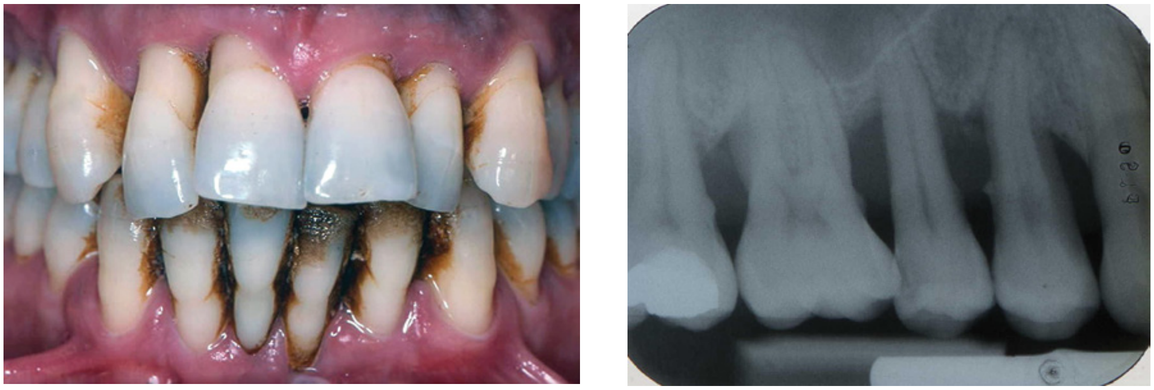

Figure 17-2 Bone Loss Associated with Combustible Tobacco Use. Clinical and radiographic findings show severe horizontal and vertical bone loss that effects the masticatory function of a 37-year-old male who smoked a pack of cigarettes a day for 20 years. These findings are consistent with the diagnosis of Stage IV, Grade C periodontitis.

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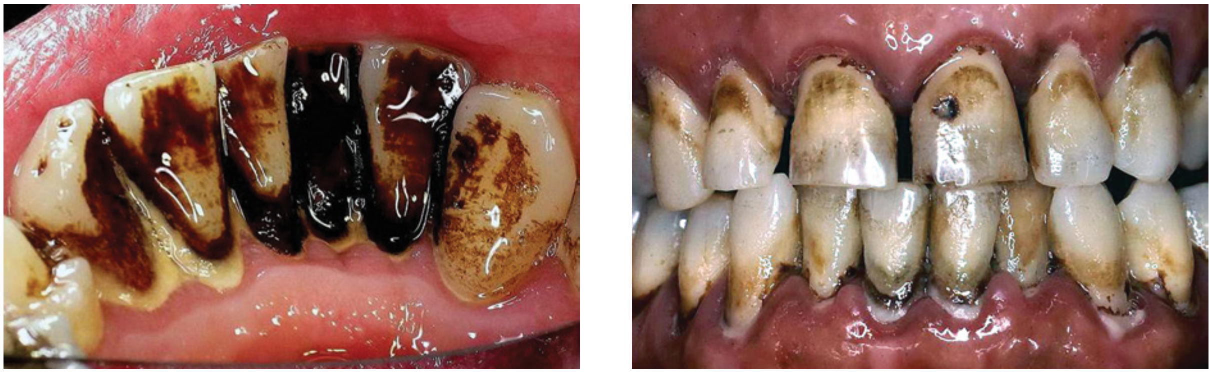

Figure 17-3 Oral Problems Associated with Combustible Smoking. Heavy tobacco staining and plaque biofilm accumulation along with accompanying signs of periodontitis are evident in a smoker. The staining is due to the buildup of tar residue.

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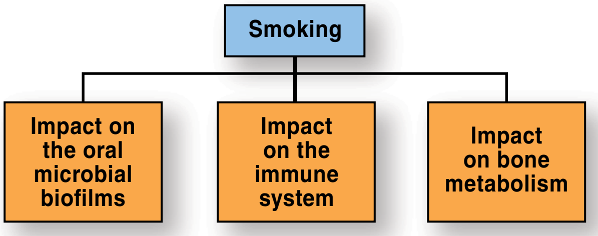
Smoking
Impact on the oral microbial biofilms
Impact on the immune system
Impact on bone metabolism

Figure 17-4 Mechanisms of Smoking-Mediated Destruction. Possible impacts of smoking on the periodontium include effects on oral biofilms, the immune system, and alveolar bone.

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1. **Impact of Combustible Smoking on the Oral Microbial Biofilm**
   a. There are conflicting reports on how combustible smoking affects the oral microflora. A few studies indicate there are no differences in the subgingival bacteria between smokers and nonsmokers.[40,41]
      1. Other investigations, however, show that the subgingival microbial profile associated with periodontitis in smokers is diverse and distinct from that of nonsmokers.[43–45] Hence, because of the differences in subgingival microbial profiles, demonstrable differences in gingival bleeding are apparent when comparing the periodontal inflammatory status of smokers versus nonsmokers, with smokers displaying a significantly lower bleeding occurrence than nonsmokers.[42]

    2. Multiple studies have shown that plaque biofilm in smokers is more likely to be colonized by *Porphyromonas gingivalis* and other potential periodontal pathogens.[46–50]
    3. More recent studies indicate that smoking is also responsible for depletion of beneficial bacteria along with an increase in pathogenic bacteria such as *Treponema* spp.[51]
  b. Cigarette smoking is associated with a lower oxygen tension in the periodontal pocket, thereby creating a subgingival environment favorable to anaerobic bacteria.[33]
  c. A recent study by Kumar and colleagues examined the impact of smoking on composition and proinflammatory characteristics of the biofilm during formation. The authors reported that smoking favors early acquisition and colonization of periodontal pathogens in oral biofilms.[43]

2. **Impact of Combustible Smoking on the Immune System**
   a. From both a biologic and epidemiologic viewpoint, numerous studies suggest that combustible smoking increases the risk of periodontal disease.[22,32,52] Smoking affects both the human immune system and cellular and humoral inflammatory response systems.[22,53,54]
   b. Smokers have decreased signs of inflammation and decreased gingival crevicular blood flow that is indicative of impaired gingival blood flow in smokers. This is due to the vasoconstrictor properties of nicotine.
   c. Although smokers have a higher number of neutrophils, neutrophil function in the peripheral circulation is impaired. Neutrophils have shown decreased adherence, chemotaxis, and phagocytosis in smokers.[55]
   d. Antibody production is another protective mechanism impacted by smoking.[55] Smoking generally decreased IgG2 antibody production that, in turn, leads to decreased serum immunoglobulin G (IgG) concentrations. IgG2 antibody production is also reported to occur in patients with severe forms of periodontitis.[48,55,56]
3. **Smoking and Bone Metabolism**
   a. Bone is one of the tissues most affected by smoking.[57] Smoking is associated with a greater amount of alveolar bone destruction in comparison to nonsmokers.[55,57–59] In a 10-year longitudinal study, reduction in bone height was 2.7 times greater in adult smokers compared to nonsmokers.[57]
      1. At least one longitudinal cohort study has reported the bone mineral content among smokers was 10% to 30% lower compared to nonsmokers.[21]
      2. Bone loss in smokers also appears to be dose dependent, with odds ratios ranging from 3.25 for light smokers to 7.28 for heavy smokers.[60]
   b. Although the mechanisms of how nicotine contributes to alveolar bone damage are not fully understood, several pathways have been proposed.
      1. In vitro studies have shown that nicotine suppresses osteoblasts while stimulating alkaline phosphatase activity.[61,62]
      2. Nicotine also increases the secretion of interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α) in osteoblasts.[63]
      3. Finally, nicotine is also known to alter normal bone remodeling by increasing the release of matrix metalloproteinases.[59]
4. **Impact of Environmental Tobacco Smoke**
   a. Nonsmokers exposed to **environmental tobacco smoke (ETS)**—“secondhand smoke” or “passive smoking”—are at increased risk for periodontitis. An analysis of NHANES III data concluded that the odds of having periodontitis are 1.6 times higher for nonsmoking adults who are exposed to ETS than adults who are not exposed to passive smoke.[64]
      1. Sanders and colleagues conclude that exposure to ETS and the presence of severe periodontitis among nonsmokers had a dose-dependent relationship.[65] Their research findings show that individuals exposed to ETS for 1 to 25 hours per week have a 29% increased risk of severe periodontitis. For those exposed to ETS for 26 hours or more per week, the odds were twice as high for severe periodontitis as individuals not exposed.
      2. An investigation of 3,137 subjects evaluated the association between ETS and periodontitis in nonsmokers. This study concluded that adults with high ETS exposure had two times the odds of periodontitis in comparison with subjects with negligible exposure.[66]
      3. A recent cross-sectional study utilizing NHANES data from 2009 to 2012 concludes that there is a 28% increase in the odds for moderate periodontitis for those with any ETS exposure.[67]
   b. Nishida and colleagues determined that passive smoke exposure was correlated to an elevation of interleukin-1β, albumin, and aspartate aminotransferase (AST) levels in the saliva.[52]

Effects of Noncombustible Tobacco on the Periodontium

Noncombustible tobacco products expose individuals to the same health risk as traditional combustible products. Like combustible products, noncombustible tobacco products have nicotine (which make it addictive) and many of the same toxic chemicals, such as cadmium, lead, arsenic, and others. Hence, prolonged exposure to noncombustible tobacco products is not any safer than combustible tobacco producs and may actually pose the same level of risk to many kinds of diseases. (Note that because heat-not-burn products such as IQOS® are new to the market there is insufficient long-term research on the impact of these products on the periodontium. Thus, for the purposes of this chapter, heat-not-burn products will not be discussed because it remains an open question as to what impact these products have on the periodontium.)

1. **Waterpipe Tobacco Smoking.** Waterpipe tobacco smoking (WTS; also known as hookah, shisha, maassel, or argileh) commonly involves a hookah (waterpipe), special flavored tobacco (shisha tobacco), and charcoal. The hookah tobacco leaves, themselves, are not acutally combusted (burned). Rather, the burning of charcoal inside the waterpipe heats up the flavored tobacco to vaporize it. The vapored tobacco then bubbles through water before being inhaled. In some countries, the hookah bowl is filled with cannabis, opium, or hashish instead of tobacco.
    a. WTS has been found to have significant amounts of nicotine and 27 known or suspected carcinogens.[68]
    b. In recent years, along with e-cigarette use, WTS has been gaining in popularity.[69] WTS is associated with respiratory and cardiovascular problems and has a significant impact on the oral cavity.
    c. Higher amounts of nicotine are delivered utilizing WTS compared to cigarettes.[70] During a single WTS episode, waterpipe smokers can inhale over 40 L of smoke compared to 1 L or less for a single cigarette.[68,71]
    d. WTS has been consistently associated with an increase in periodontal pocket depths, loss of clinical attachment, and bone loss compared to nonsmokers.[72–74] Hence, the negative impact of WTS on the periodontium is the same as tobacco smoking.[75]
    e. The prevalence of noncombustible tobacco use is on the rise across the globe. Most patients do not associate these habits with smoking. As a result, practitioners should modify their health history questionnaire to include questions to elicit information about other sources of nicotine. Although cigarette smoking has been steadily but slowly declining in the United States and Canada, the use of alternate tobacco products and their impact on the periodontium cannot be ignored.
2. **Smokeless Tobacco**
    a. Smokeless tobacco includes a broad category of noncombusted tobacco products. They are used orally as chewed tobacco and occasionally inhaled nasally. Smokeless tobacco products available in the United States fall into one of five categories: moist snuff, loose-leaf chewing tobacco, plug or twisting tobacco, snus, and scotch or dry snuff.
    b. Smokeless tobacco use in the United States is most prevalent among adolescent and young males.
    c. Smokeless tobacco is associated with oral carcinoma.[76]
    d. Large-scale studies of athletes and adolescents who use smokeless tobacco have demonstrated a strong relationship to white oral mucosal lesions (**Figure 17-5**). The lesions are commonly found in areas of the mouth where smokeless tobacco products are placed. These lesions are found in 50–60% of smokeless tobacco users.[77]

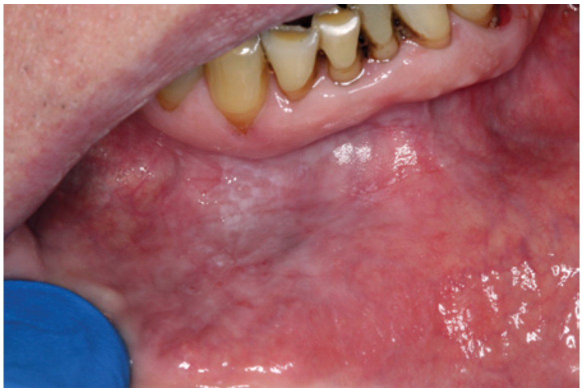

Figure 17-5 White Mucosal Lesion. This clinical photograph shows a white lesion on the oral mucosa of this patient who routinely uses smokeless tobacco.

Effects of Noncombustible Nicotine on the Periodontium

The e-cigarette was introduced to the U.S. market in 2007. E-cigarettes heat nicotine (extracted from tobacco), flavorings, and other chemicals to create an aerosol that is inhaled. Smoking e-cigarettes is also called vaping, which refers to the use of a vaporizer or e-cigarette to inhale a vapor that contains nicotine. E-cigarettes are highly addictive. In the United States, e-cigarette use among high-school students increased by 900%, and 40% of young e-cigarette users had never smoked tobacco.[78] The popularity of e-cigarettes among youth and their unknown long-term effects are worrisome.[69]

1. Laboratory analysis of e-cigarettes conducted by the FDA shows quite clearly that the fluid and aerosol in e-cigarettes contain known toxins, including propylene glycol, heavy metals, volatile organic compounds, and tobacco-specific nitrosamines.
    a. At this time, the possible side effects of inhaling nicotine vapor, as well as other health risks e-cigarettes may pose, both to users and to the public, are unknown.
    b. Jeong et al. confirmed that both electronic and conventional cigarette use was associated with an increased rate of periodontal disease.[79] They concluded that vaping may not be a safe alternative to smoking and that cessation of both types of cigarettes is necessary for maintaining oral health. **Figure 17-6** shows the extent of periodontal destruction in a 34-year-old patient who uses e-cigarettes daily. Note the lack of staining or discoloration on the periodontium and teeth; because e-cigarettes do not contain tobacco, they do not leave behind sticky, discoloring tar residue.
    c. Some cigarette smokers are utilizing e-cigarettes as smoking cessation aids. However, the extent to which e-cigarettes can help with smoking cessation is unclear.[69]

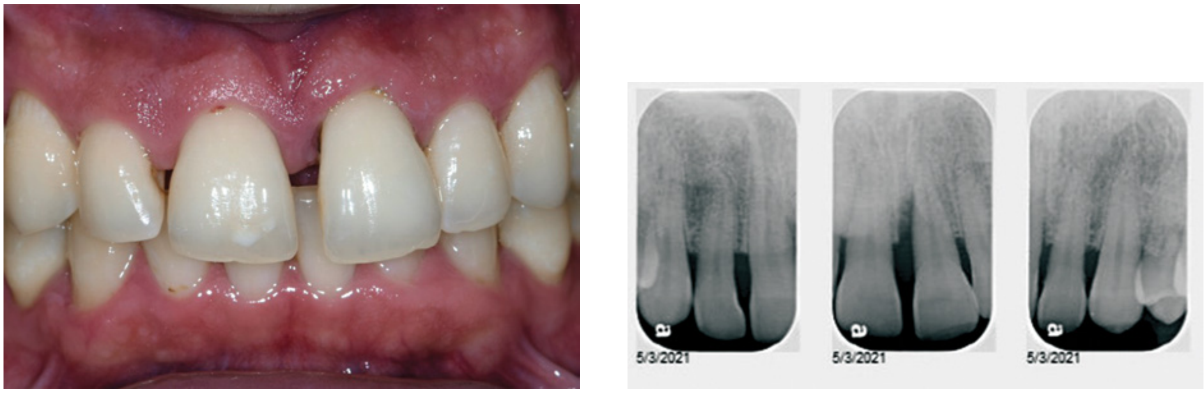

Figure 17-6 **Presentation of a 34-Year-Old Habitual E-Cigarette Smoker.** Although staining is minimal, the clinical presentation shows signs of inflammation with radiographic bone loss.

Effects of Smoking on Periodontal Therapy

The effects of smoking on periodontal therapy are the same whether an individual uses a combustible tobacco product, a noncombustible tobacco product, or a noncombustible nicotine product.

1. **Impact of Chemical Products and Toxins in Cigarette Smoke on Periodontal Therapy**
    a. Chemical products and toxins in tobacco smoke may delay wound healing by impairing normal repair functions necessary for the initiation of wound healing.[55]
    b. Volatile components of cigarette smoke—namely acrolein and acetaldehyde—may inhibit gingival fibroblast attachment and proliferation. Fibroblasts exposed to nicotine produce less extracellular matrix, less collagen, and more collagenase. These negative effects on fibroblast functions influence wound healing and the progression of periodontitis.[22,32]
2. **Smoking and Response to Periodontal Treatment.** Smoking not only increases the risk for developing periodontal disease but also impacts the response to periodontal treatment. Smokers show a poorer response to periodontal therapy compared to nonsmokers.[80–82]
    a. Smokers exhibit less reduction in probing depth and less gain in clinical attachment after treatment compared to ex-smokers or nonsmokers.[83]

b. In 6-year longitudinal study, nonsmokers had approximately a 50% higher rate of improvement in probing depth and clinical attachment levels after periodontal therapy than did active smokers.[60]
c. Periodontal treatment in smokers, both surgical and nonsurgical therapies, has been associated with improvements in periodontal outcomes. Comparison of the outcomes, however, showed significantly less improvement in the smokers compared with nonsmokers.[82–90]

Section 5

Cannabis as a Risk Factor for Periodontal Disease

Overview of Cannabis

1. Cannabis is one of the most commonly used substances for medical and recreational purposes. Cannabis preparations are derived from the plant *Cannabis sativa*. The three main forms of cannabis are marijuana, hashish, and hash oil. Marijuana is the most common.[91]
2. Cannabis and tobacco share many similarities. They are both derived from dried leafy plants primarily consumed through smoking, release hazardous compounds that can be potentially carcinogenic, and are potentially addictive. However, unlike the combustible tobacco products and noncombustible nicotine/tobacco products, marijuana does not contain nicotine. Rather, when marijuana leaves are burned (or consumed through edible products laced with marijuana), psychoactive (mind-altering) THC (delta-9-tetrahydrocannabinol) is released, which is responsible for most of the intoxicating euphoric and relaxed effects associated with the substance. Other common marijuana-associated effects, which may vary among different individuals, is heightened sensory perception, laughter, altered perception of time, and increased appetite. When used under the strict supervision of a medical practitioner, marijuana may have medicinal value in managing patients undergoing cancer chemotherapy or in increasing appetite among AIDS patients who are suffering from wasting syndrome. However, when used recreationally, marijuana can potentially lead to cognitive impairments and irreversible damage to the brain, lungs, and heart.

Epidemiology of Cannabis in Periodontal Patients

1. Cannabis smoking may be a risk factor for periodontal disease that is independent from tobacco use.
2. In a review examining attachment levels of adults between 26 and 32 years of age, the incidence of attachment loss in the no exposure group (defined as individuals never been exposed to cannabis), "some occasional" (defined as individuals who were exposed to cannabis on 1 to 40 occasions during the previous year), and high cannabis exposure (defined as individuals who were exposed to cannabis on 41 or more occasions in the previous year) groups was 6.5%, 11.2%, and 23.6%, respectively.[92]
3. The conclusion reached by the above systematic review and meta-analyses determined that smoking cannabis is related to a higher occurrence of periodontal disease.[92,93] Hence, cannabis use carries with it the same, if not higher, risk of periodontitis than combustible and noncombustible products.

Effects of Cannabis on the Periodontium

1. Generally, cannabis users have poorer oral health than noncannabis users, having more decayed, missing, and filled teeth; higher plaque scores; and less healthy gingiva.[94]
2. Smoking cannabis is associated with gingival enlargement, erythroplakia, and chronic inflammation of the oral mucosa with hyperkeratosis and leukoplakia. This is sometimes referred to as cannabis stomatitis.[94]
3. According to the NHANES study utilizing data from 2011–2012, frequent use of recreational marijuana is associated with deeper probing depths, more clinical attachment loss, and higher odds of having severe periodontitis.[95]
4. With the trend toward legalization, the use of cannabis has increased significantly in recent years.
5. **Figure 17-7** summarizes several factors a clinician must take into account when treating a cannabis-using patient. **Figure 17-8** shows the periodontal condition of a cannabis-using patient.

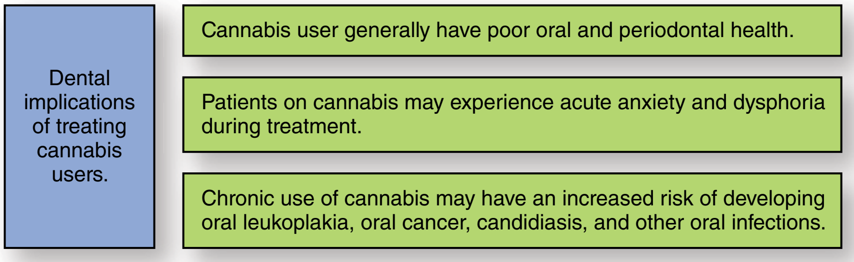
Dental implications of treating cannabis users.
Cannabis user generally have poor oral and periodontal health.
Patients on cannabis may experience acute anxiety and dysphoria during treatment.
Chronic use of cannabis may have an increased risk of developing oral leukoplakia, oral cancer, candidiasis, and other oral infections.

Figure 17-7 **Treating Cannabis Users.** The oral health care provider must be aware of the dental implications of treating cannabis users.

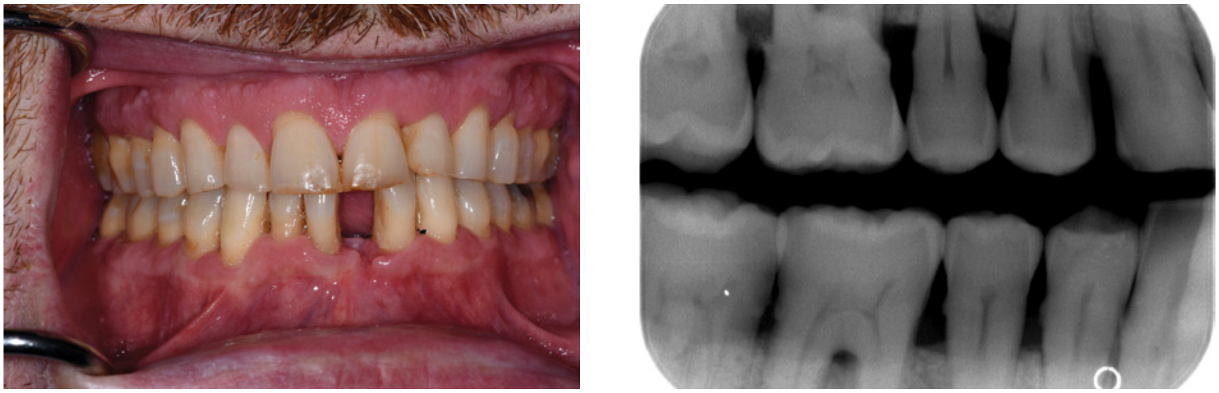

Figure 17-8 **Patient with Cannabis Addiction.** A cannabis-addicted patient with severe periodontal involvement and missing teeth with radiographic bone loss. The patient became addicted to cannabis following severe nerve injury as the result of a motorcycle accident.

Section 6

Smoking and Peri-Implant Disease

A **dental implant** is a nonbiologic (artificial) device surgically inserted into the jawbone to (1) replace a missing tooth or (2) provide support for a prosthetic denture. The peri-implant tissues are the tissues that surround the dental implant. In many ways, the peri-implant tissues are like the periodontium of a natural tooth. Dental implants and peri-implant disease categories are discussed in detail in Chapter 8.

The Impact of Smoking on Dental Implants

1. Heat produced by smoking, as well as the toxic by-products of cigarette smoking, such as nicotine, carbon monoxide, and hydrogen cyanide, have been implicated as risk factors for impaired healing after implant surgery.[96]
2. Smokers experience almost twice as many implant failures compared with nonsmokers and are more prone to peri-implant bone loss in the maxilla.[97–104]
3. Risk indicators associated with increased peri-implant mucositis and peri-implantitis include poor plaque biofilm control, a history of periodontitis, diabetes, and smoking.[96,100,102–104]
4. The combination of smoking and a history of periodontitis (treated or untreated) increase the risk of peri-implant bone loss (**Figures 17-9** and **17-10**).[97,103]
5. In a recent study, the health of the implant was compromised both in smokers and individuals who vaped. This change was due to increased levels of proinflammatory cytokines.[105]

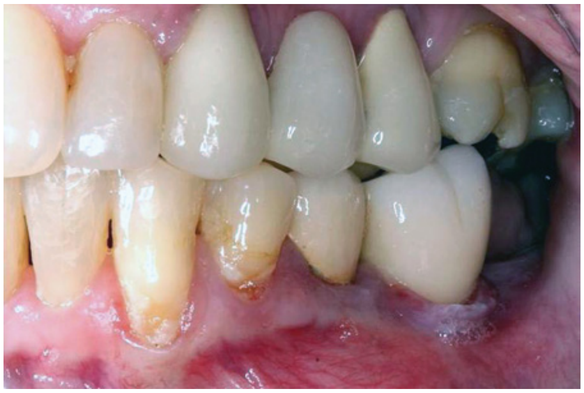

Figure 17-9 Peri-Implant Mucositis. Clinical signs of peri-implant mucositis of the mandibular first molar.

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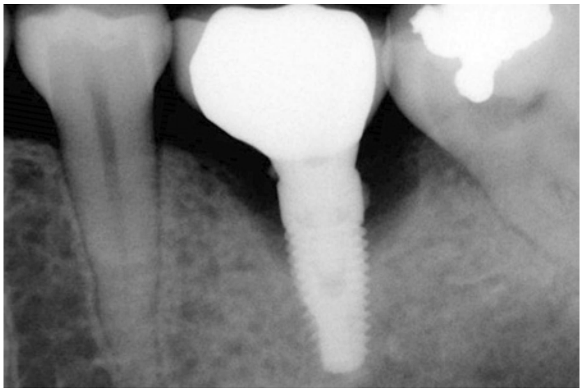

Figure 17-10 Peri-Implantitis. Radiographic evidence of peri-implantitis featuring circumferential angular bone loss.

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Section 7

Smoking Cessation for the Periodontal Patient

Effects of Smoking Cessation on the Periodontium

Few publications in the periodontal literature specifically address the impact of smoking cessation on the periodontium, most likely because of the common challenges in motivating patients to quit smoking. Fiorini and colleagues conducted a systematic review of the literature to evaluate the effect of smoking cessation on periodontitis progression and response to periodontal therapy. Based on the limited available evidence, Fiorini and colleagues concluded that smoking cessation seems to have a positive influence on periodontitis occurrence and periodontal healing.[20]

1. **The Effect of Smoking Cessation on Periodontal Status**
   a. Current smokers usually have significantly worse periodontal conditions (greater probing depths, more attachment loss, and alveolar bone loss) than either never smokers or former smokers. The NHANES III study concluded that approximately half of periodontitis cases could be attributed to either current smoking (42.9%) or former smoking (10.9%).[27,33]
   b. In general, the periodontal health status of former smokers is not as good as that of never smokers, but is better than that of current smokers.[106] These findings suggest that although the past effects of smoking on the periodontium cannot be reversed, smoking cessation is beneficial to periodontal health.[107]
   c. The American Academy of Periodontology strongly recommends inclusion of tobacco cessation counseling as an integral part of periodontal therapy.[108]
2. **The Effect of Smoking Cessation on Periodontal Treatment Outcomes**
   a. Studies have confirmed that treatment outcomes in former smokers are generally similar to those that can be expected in never smokers, but are usually better than those that can be expected in current smokers.[107]
   b. The benefits of smoking cessation on the periodontium likely result from (1) a reduction in pathogenic bacteria in the subgingival plaque biofilm, (2) improved vascular circulation in the gingiva, and (3) improvements in the host immunoinflammatory response.
   c. It cannot be overstated that smoking is one of the greatest risk factors to the onset and progression of periodontitis. The evidence for this association is insurmountable, as countless epidemiologic studies have substantiated that the likelihood of developing periodontitis is higher in smokers compared to nonsmokers. Equally important, cessation of the smoking habit is one of the most important actions a smoker can take as it can lead to immediate and substantial benefits in reducing the risk of periodontitis. In a

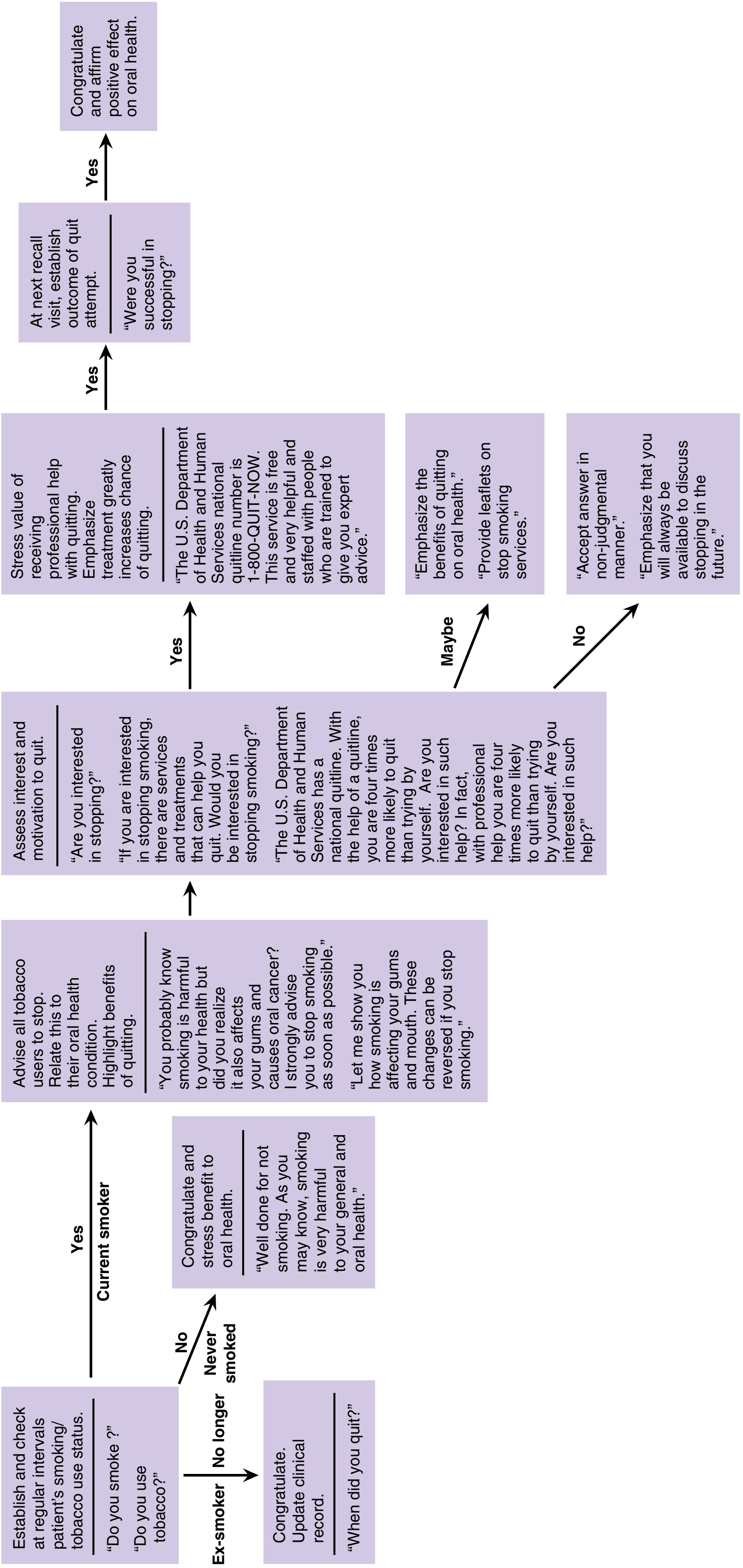
recent study using NHANES data, investigators found that among former smokers, the time since quitting the smoking habit (in other words, the number of years since the individual quit the smoking habit) significantly reduces the risk of developing periodontitis.[109] To put this in perspective, each additional year of having quit the smoking habit was associated with an additional 2.5% to 5.2% reduction in the odds of a former smoker developing periodontitis. Hence, the time since quitting the habit is significantly protective against periodontitis.

Smoking Cessation Counseling in Periodontal Therapy

1. **Smoking Cessation and the Prevention of Periodontal Disease**
   a. The knowledge that smoking is a significant risk factor suggests that in smokers, smoking cessation might prevent more periodontal disease than daily plaque control self-care. All patients should be assessed for smoking status and smokers should be given smoking cessation counseling.[110]
   b. **Smoking cessation counseling** includes information on smoking cessation and prevention of tobacco use, as well as referrals to other health professionals for tobacco cessation programs.
2. **The Role of the Dental Team in Tobacco Cessation Counseling**
   a. The World Health Organization (WHO) advocates that all health providers must be involved in tobacco cessation efforts, including oral health professionals who reach a large proportion of the healthy population.[111]
   b. Dental team members have regular contact with patients, are the first to see the effects of tobacco in the mouth, and are the only health professionals who frequently see “medically healthy” patients. Thus, dental hygienists are in an ideal position to reinforce the antitobacco message, as well as being able to motivate and support smokers willing to quit.
   c. Dental hygienists have a professional responsibility to provide tobacco cessation services as a routine component of dental hygiene practice. Smoking cessation guidelines recommend that all health professionals, including dental team members, should check the smoking status of their patients at least once a year, and should advise all smokers to stop smoking.[20,111,112]

A User-Friendly Model for Counseling the Periodontal Patient

1. **Counseling Time Commitment.** In the clear majority of cases, dental teams will only be involved in delivering brief advice to smokers. This should take less than 5 minutes of their time.
2. **Key Elements in Providing Brief Advice**
   a. All patients should have their smoking/tobacco use status (current, ex-, never smoked) established and checked at regular intervals. This information should be recorded in the patient’s chart.
   b. Smokers should then be asked some simple questions to assess their degree of interest in stopping smoking/tobacco use.
   c. All smokers and chewers of tobacco should be advised of the value of stopping and of the health risks of continuing. The advice should be clear, firm, and personalized.
   d. Although most people know of the risks of tobacco use in relation to cancers and heart disease, fewer are aware of the detrimental effects on the mouth. Dental teams thus have a unique opportunity to highlight the dangers of tobacco use. The early signs of tobacco use—such as tooth staining, changes to the soft tissues, and halitosis—are easily identified and are reversible, and this provides a useful means of motivating smokers to stop.
   e. All smokers and chewers of tobacco should be advised of the value of the support offered by quitlines. Quitlines are toll-free telephone centers staffed by trained smoking cessation experts. It takes as little as 30 seconds to refer a patient to a quitline. Smokers who are interested and motivated to stop should be referred to these services. The U.S. Department of Health and Human Services has a national quitline number: 1-800-QUIT-NOW (1-800-784-8669).
3. **A Pathway and Sample Dialogs for Cessation Counseling.** A smoking cessation care pathway for dental practice is summarized in **Figure 17-11**.[113] **Boxes 17-1** to **17-3** provide sample dialogues for cessation counseling.



Establish and check at regular intervals patient's smoking/ tobacco use status.

“Do you smoke ?”

“Do you use tobacco?”

Advise all tobacco users to stop. Relate this to their oral health condition. Highlight benefits of quitting.

“You probably know smoking is harmful to your health but did you realize it also affects your gums and causes oral cancer? I strongly advise you to stop smoking as soon as possible.”

“Let me show you how smoking is affecting your gums and mouth. These changes can be reversed if you stop smoking.”

Congratulate and stress benefit to oral health.

“Well done for not smoking. As you may know, smoking is very harmful to your general and oral health.”

Congratulate. Update clinical record.

“When did you quit?”

Assess interest and motivation to quit.

“Are you interested in stopping?”

“If you are interested in stopping smoking, there are services and treatments that can help you quit. Would you be interested in stopping smoking?”

“The U.S. Department of Health and Human Services has a national quitline. With the help of a quitline, you are four times more likely to quit than trying by yourself. Are you interested in such help? In fact, with professional help you are four times more likely to quit than trying by yourself. Are you interested in such help?”

Stress value of receiving professional help with quitting. Emphasize treatment greatly increases chance of quitting.

“The U.S. Department of Health and Human Services national quitline number is 1-800-QUIT-NOW. This service is free and very helpful and staffed with people who are trained to give you expert advice.”

“Emphasize the benefits of quitting on oral health.”

“Provide leaflets on stop smoking services.”

“Accept answer in non-judgmental manner.”

“Emphasize that you will always be available to discuss stopping in the future.”

At next recall visit, establish outcome of quit attempt.

“Were you successful in stopping?”

Congratulate and affirm positive effect on oral health.

Figure 17-11 A Flowchart for Smoking Cessation Counseling.

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Box 17-1 Sample Cessation Counseling Dialogue 1

The patient is a 22-year-old radiology technician who has come to the dental office for a biannual exam. The patient has no significant health history but has periodontitis on exam. The patient began smoking at age 12 and has smoked three-quarters to one pack a day for almost 10 years.

Clinician: Your oral exam shows evidence of damage from smoking. How do you feel about quitting?
Patient: I want to quit. My fiancé hates that I am smoking. Every day I tell myself not to smoke but I just cannot seem to quit.
Clinician: What is the most difficult part about quitting? What is your biggest barrier?
Patient: I feel like cigarettes are my best friends! The only time I relax is when I smoke—or when I am out drinking and smoking with my friends. Most of my friends smoke.
Clinician: Have you ever tried to quit?
Patient: Every day! But nothing works! And then my fiancé yells at me so I feel worse and end up smoking more. I feel so guilty and so embarrassed!
Clinician: It sounds like you are in a vicious circle that is probably making it harder to quit. Let's put this in perspective. Of all addictions, it is harder to take control over the nicotine addiction than any other. But the good news is that there are more things available to help smokers quit than ever before. And quitting at your young age will be so beneficial in every way—including your oral health! The longer we do anything, the harder it is to stop. So, stopping now would be the best thing you will ever do for yourself, and while it will be very difficult, it will be easier than if you continue to smoke for another 20 years.
Patient: What is the best way to quit?
Clinician: Probably the most important thing is for you to do is make the decision to quit. That is even more important than wanting to quit. Rather than telling yourself, "today I am not going to smoke," set a firm quit date within 1 to 2 weeks of this appointment and make plans on how not to smoke. Think of quitting as taking on a new job. With all the other responsibilities you have, quitting should be your priority for about 3 months. And get help—the more support you have the better.
Patient: What kind of help? My fiancé tells me to just stop—that if I really wanted to quit I could.
Clinician: A lot of people think smokers should just stop. But that is like telling an alcoholic to just stop drinking, and we wouldn't do that. We tell other addicts to get quit therapy, and that's what smokers should do! It is about learning how to quit. We rarely get what we want in life unless we work at it.
Patient: I never thought of it that way. I thought I couldn't stop because I am weak or just don't have any will power.
Clinician: Not at all. I would suggest calling 1-800-QUIT-NOW. This is a free service that provides counseling on how to quit. This quitline can also tell you about programs near you if you are interested in more intensive treatment. And as your provider, I will assist you in any way I can—including educate you about the medications that help people quit.
Patient: Thank you. I will call the quitline and let you know what I decide to do.
Clinician: Good for you. You will miss your "best friend" but your life will be so much better once you have made the break!

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Box 17-2 Sample Cessation Counseling Dialogue 2

The patient is a 50-year-old grant writer who has come to the dental office for follow-up care. The patient has a history of type 2 diabetes, COPD (chronic bronchitis), and severe periodontal disease that has resulted in tooth loss. The patient began smoking at age 13 and has smoked a pack a day for many years. About a year ago the patient was successful at cutting the daily consumption to half a pack.

Clinician: Congratulations on being able to cut down on your smoking! Have you thought about quitting completely? We might be able to save your remaining teeth if you are able to quit.
Patient: I know I should quit but I really love my cigarettes.
Clinician: Has it been hard for you to cut down?
Patient: Actually, it's been horrible.
Clinician: What has been the most difficult part for you?
Patient: I can't stop thinking about my next cigarette. I find myself thinking about when I can get the next cigarette in as soon as I put one out!

(*continues*)

Box 17-2 Sample Cessation Counseling Dialogue 2 (continued)

Clinician: It almost sounds like the cigarettes have become even more important to you since you cut down! While quitting is the most difficult thing you might ever do, it might in fact make your life easier to not have to worry about when you can get in your next cigarette.

Patient: That's true—it would be so nice to not to have to think about them anymore.

Clinician: So, you still love your cigarettes but don't love that you are smoking!

Patient: Yes that is exactly it. I hate that I am still smoking. I know it is so bad for my health. I feel that I must quit but I have tried to quit before and the longest I have ever gone without a cigarette has only been 4 hours! Cigarettes are the only things that calm me down.

Clinician: How soon do you smoke when you first get up in the morning and do you ever smoke if you wake during the night?

Patient: I smoke before my feet hit the floor every morning and I often take a couple of drags if I wake up during the night.

Clinician: It sounds like you have a strong physical addiction as well as a significant psychological addiction. Have you considered using medication to help you quit? You would be a good candidate for pharmacotherapy.

Patient: No. I take insulin for my diabetes and I don't want to take anything that would interfere with that.

Clinician: Actually, smoking interferes with controlling your diabetes much more than a quit-smoking medication. And were you aware that with every cigarette your blood sugar goes up? So, your diabetes would be much easier to control if you quit.

Patient: I had no idea that was the case.

Clinician: One suggestion would be to look at quitting the same way you look at having diabetes. A nicotine addiction is a chronic condition just as diabetes is. You do whatever you can to control your blood sugar with diet, exercise, and medication to enhance your quality of life and decrease risks—even though you may not want to do all those things. Try to look at quitting that way. Even though you love cigarettes, you will benefit by taking control! Look at quitting as something you are doing for yourself rather than to yourself. That approach may make it easier to let go of smoking.

Patient: I like that. It puts me in control of the situation—rather than letting the cigarettes control me! Maybe I will consider trying a medication to help me.

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Box 17-3 Sample Smoking Cessation Counseling Dialogue 3

The patient is a 48-year-old attorney who has come to the dental office for care. The patient has a history of hypertension, high cholesterol, and periodontitis. The patient reports smoking from age 17 and has smoked two packs a day for 30 years.

Clinician: As you know, your oral exam shows significant damage from smoking and your blood pressure is elevated today. Have you tried to quit since our last visit?

Patient: Not really. And I don't want to talk about it. Everyone is on me. My doctors, my significant other, my kids, and now I suppose you are going to give me a hard time too.

Clinician: I don't want to give you a hard time. But I do want you to encourage you to at least try and quit. The more times you try, the more chance you have of success. I am not going to tell you how dangerous it is to smoke. But I am going to remind you that by quitting you can reverse so much of the damage cigarettes have caused in you.

Patient: Look I really don't want to quit. I exercise and I watch what I eat. I'll keep coming to you for my teeth and take medicine for my heart problems and hope for the best.

Clinician: You are such a "take charge" person in every other aspect of your life. How about taking control over this addiction rather than hoping for the best?

Patient: I will quit when I am ready. I know myself. When I decide to do something, it gets done.

Clinician: Take advantage of that! Consider what is available to help you quit now. It is possible to quit even if you don't want to quit or don't feel ready.

Patient: Well quitting would get people off my back anyway. My kids nag me every day.

Clinician: People in your life are concerned about you. But you cannot quit for them. You can use the fact that you will have a longer, better quality life with your kids as a motivation though the decision to quit is yours. No one, nothing, can make you quit but there are plenty of us who can help you quit.

Patient: If I do this, I am doing it on my own.

Clinician: I understand your wanting to take that approach. However, it would be so much easier if you use a medication. You have been smoking for a long time and a medication would increase your chance of permanent success.

Patient: Is it ok to take something with all my heart problems?

Clinician: Absolutely. In fact, individuals using medication have the highest success rates. It should be started 1 to 2 weeks prior to quitting and once there is a therapeutic dose in your system, you should notice a significant decrease in your desire to smoke. I can provide you with more information and arrange for a prescription.

Patient: I have heard that quitting smoking makes you feel depressed.

Clinician: Feeling depressed is very common with quitting! I will show you a list of the most common withdrawal symptoms so you will be aware of what to expect. And give you information on the FDA-approved medications so you will have all the options. Think about setting a quit date within a couple of weeks and let me know how I can best assist you through the process. I will keep working with you until you are able to quit.

Patient: OK I will try but this is just between you and me.

Clinician: I understand and will respect that. At some point people in your life will be aware that you are trying to quit and it might help you to let them know what they can do to help you—including NOT nag you! I am very proud of you for making the attempt. You will not regret it!

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Chapter Summary Statement

To be clear, there is no safe form of smoking: all forms of smoking contain hazardous ingredients (e.g., nicotine for combustible tobacco, noncombustible tobacco, and noncombustible nicotine products; and delta-9-tetrahydrocannabinol in marijuana) that can lead to addiction and dependence. Moreover, all forms generate harmful and potentially harmful chemicals that can be hazardous to an individual's overall health. Although newer tobacco/nicotine delivery systems, such as noncombustible tobacco products and noncombustible nicotine products, are being marketed as a safe alternative to conventional combustible products, this is presently an open question as there is still insufficient data to substantiate the manufacturers' purported claims. Further the FDA and Health Canada do not deem these newer tobacco/nicotine delivery systems to be any safer than combustible products. What is certain, however, is that there is sufficient evidence that points to the benefits of smoking cessation on a wide variety of oral health outcomes, including periodontal treatment. Advice and assistance on smoking cessation is therefore an integral part in the management of all patients seeking periodontal care. The benefits in motivating a smoker to be enrolled in a smoking cessation program cannot be overstated. Over time, individuals who cease their smoking habits are less likely to suffer from smoking-associated complications, such as chronic obstructive pulmonary disorders, cancer, and periodontal disease, and are more likely to see positive benefits to their systemic and oral well-being.

Section 8

Focus on Patients

Clinical Patient Care

CASE 1

A new patient with Stage III periodontitis has a history of smoking one to two packs of cigarettes each day. The patient would like to have a healthy mouth but fears that any attempt as quitting smoking will end in failure. What counsel would you provide this patient about the effect of the smoking habit on the likelihood of long-term control of his periodontitis?

Clinical Evidence in Action

Clinical Relevance

The current smoking prevalence in the United States is under 15%—why is the focus on helping smokers quit so important? Fifty years after a momentous report on its hazards, smoking remains the most significant public health predicament. Cigarette smoking is responsible for an estimated 443,000 deaths annually in the United States. Almost 45 million American adults smoke.[1] The most vulnerable of our population are most likely to be affected—the less educated, youth, LGBTQ persons, and minorities. In addition, people who live below the poverty line are 30% more likely to smoke than those who live above it. The prevalence is even higher for those with psychiatric comorbidities.

In its 2014 report, "The Health Consequences of Smoking—50 Years of Progress,"[2] the U.S. Surgeon General concluded that, while significant improvements have been made since the publication of its landmark 1964 report, cigarette smoking remains a major public health problem. It is the leading cause of preventable death, increasing risks of such common causes of mortality as cardiovascular disease, pulmonary disease, and malignancy. Half of those who continue to smoke will die prematurely from a tobacco-related cause.

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Bottom Line on Smoking Cessation: What Should You Recommend?

The most recent comprehensive smoking cessation guideline, sponsored by the U.S. Public Health Service, was published in 2008.[1] The U.S. Preventive Services Task Force (USPSTF) recommendation that "clinicians ask all adults about tobacco use and provide tobacco cessation interventions" for those who smoke was issued 1 year later.[2] Since then, countless investigations have assessed the merits of the various cessation medications and counseling designed to help smokers achieve and maintain tobacco abstinence. The most successful practice recommendations are to prescribe FDA approved medications or nicotine replacement as first-line pharmacotherapy for smoking cessation (advising a first-line medication—bupropion, varenicline, nicotine gum, nicotine inhaler, nicotine lozenge, nicotine nasal spray, or nicotine patch—for every patient who smokes is a key component of the 2008 guideline) and provide counseling along with the medication—the combination of medication and counseling has proven to be more effective than either option alone. Despite this evidence, most smokers are still trying to stop on their own, and more than 95% of smokers who try to quit without treatment assistance will fail, the majority relapsing within 1 week. Members of the dental team should counsel all tobacco users, even those reluctant to quit, about the benefits of stopping smoking and educate about how to best achieve permanent cessation. As one of the largest groups of health care professionals, dental hygienists, can have an enormous impact. All hygienists have multiple clinical opportunities to encourage smoking cessation and are therefore in a unique position to reduce cigarette consumption and consequently the burden of illness, death, and economic costs resulting from tobacco use.

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CHAPTER 18

Nutrition, Inflammation, and Periodontal Disease

- Section 1 Association Between Obesity and Periodontal Disease
- Section 2 Micronutrients, Antioxidants, and Periodontal Disease
- Section 3 Macronutrients and Periodontal Disease
- Section 4 Focus on Patients
  - Clinical Patient Care
  - Ethical Dilemma

CLINICAL APPLICATION

This chapter spotlights the emerging evidence on the association between nutrition and periodontal disease and provides some valuable recommendations for applying this information to a clinical setting. All members of the dental team must be aware of what is known about this relationship and understand how to apply this information to guide patients with nutritional issues that may have an impact upon periodontal health.

LEARNING OBJECTIVES

- Discuss the link between obesity and periodontal disease.
- Discuss the role of polymorphonuclear leukocytes in the production of reactive oxygen species in response to plaque biofilm.
- Discuss how antioxidants modify periodontal disease onset and progression.
- Describe the proposed roles of micronutrients and macronutrients in periodontal disease.
- List some oral symptoms associated with ascorbic acid deficiency gingivitis.
- Explain the role of dental health care providers in addressing obesity and nutrition in the management of periodontal disease.

KEY TERMS

Obesity
Body mass index (BMI)
Reactive oxygen species (ROS)
Micronutrients
Macronutrients
Antioxidants
Ascorbic acid deficiency gingivitis

CLINICAL APPLICATION

Historically, periodontal disease was conceptualized to be a bacterial biofilm-induced inflammatory disease with limited associations to nutrition. This was because unlike the well-established link between sugar and dental caries, many early periodontal studies investigating the relationship between nutrition and periodontal disease failed to

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show associations between nutritional status and periodontal disease. These preliminary studies, however, used poor methodologies, tended to study nutrients in isolation, and failed to control for confounding variables (such as smoking). Improved understanding of periodontal disease at the cellular level and more stringent nutritional methodologies have created a renewed interest in the relationship between nutrition and periodontal disease. Current research is focused on (1) the association between obesity and periodontal inflammation and (2) the role of antioxidants in the prevention and treatment of periodontal disease.

Section 1

Association Between Obesity and Periodontal Disease

1. **Obesity Overview**
    a. **Obesity** is an excess amount of body fat in proportion to lean body mass, to the extent that health is impaired.[1]
        1. The most commonly used screening assessment to measure body fat is the **body mass index (BMI)**. The BMI is a simple index that takes account of a person's weight and height to classify the individual as underweight, normal weight, overweight, or obese. Outside the United States, the BMI is calculated by taking the person's weight in kilograms divided by the square of his or her height in meters ($Weight\ (kg)/[height\ (m)]^2$). In the United States, however, the BMI is calculated by taking the patient's weight in pounds divided by the square of his or her height in inches multiplied by 703 ($Weight\ (lb)/[height\ (in)])^2\ x703$). The World Health Organization (WHO) and the National Heart, Lung, and Blood Institute define underweight as a BMI of less than 18.5, normal weight as a BMI of 18.5 to 24.9, overweight as a BMI of 25 to 29.9, and obesity as a BMI of equal to or greater than 30.
        2. According to the U.S. Centers for Disease Control, the BMI is not intended to directly measure body fat or diagnose an individual's body fitness. Rather, the BMI is an important screening tool that correlates an individual's weight to potential health problems, such as metabolic syndrome (For a review of metabolic syndrome and its potential impact on systemic health and oral health, please refer to Chapter 14, Section 2).[2]
        3. Using BMI, over 1.9 billion adults worldwide were classified as overweight in 2016, and 39 million children under the age of 5 were overweight or obese in 2020.[3]
        4. More than 65% of the U.S. adult population, 15.8% of children aged 6 to 11 years, and 16.1% of adolescents aged 12 to 19 years are overweight.[4,5]
        5. More than a third of adults (42%) in 2017–2018 and 17% of youth (ages 2 to 19) were obese in 2011–2014. From 1999–2000 to 2017–2018, adult obesity in the United States rose from 30% to 42%, respectively, with severe obesity rising from 5% to 9%, respectively. The Centers for Disease Control and Prevention (CDC) data comes from an analysis of the National Health and Nutrition Examination Survey (NHANES) 2011–2014 in the United States.[4,6] NHANES is a research study started in the 1960s to assess the health and nutritional status of children and adults in the United States. International trends are similar to those in the United States. The International Obesity Task Force estimates that worldwide over 1 billion adults are overweight.[7]
    b. Adipose tissue is a complex and metabolically active endocrine organ that secretes numerous immunomodulatory factors and plays a major role in regulating metabolic and vascular biology.[8] Obese individuals are reported to have elevated levels of circulating pro-inflammatory cytokines, such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), compared to normal-weight controls.[9]
2. **The Obesity–Periodontal Disease Link**
    a. Recent research has identified a possible link between obesity and periodontal disease.[10–15]
        1. An analysis of data from the NHANES III involving over 13,000 individuals having one or more sites with clinical attachment loss found a positive correlation between BMI and the severity of periodontal attachment loss.[16]
        2. Al-Zahrani and colleagues conducted a study of a representative sample of participants in the NHANES III survey who were 18 years or younger and had undergone a periodontal examination.

a. The purpose of the study was to examine the relationship between body weight and periodontal disease in a representative U.S. sample. BMI and waist circumference were used as criteria of overall and abdominal fat content, respectively.

b. Study results indicate that, even in a younger population, both overall and abdominal obesity are associated with increased prevalence of periodontal disease, whereas underweight (BMI <18.5) is associated with decreased prevalence.[17]

3. Recent systemic reviews indicate that individuals who are overweight or obese tended to suffer from periodontitis compared to individuals of a normal weight.[15,18] In a separate systemic review focusing on younger individuals, 17 papers were identified that showed an association between obesity and periodontitis in adolescents and young adults.[19]

4. In the United States, a case study by the Forsyth Institute reports that obesity is related to a marked increase in plaque biofilm accumulation, attachment loss, deep pockets, bleeding on probing, and an increase in the proportion of *Tannerella forsythia.*[20]

b. Among lifestyle-related risk factors, smoking has the greatest impact on periodontitis.[21] Both smoking and obesity are independent risk indicators for periodontitis. Nishida and colleagues suggest that obesity is second only to smoking as the strongest lifestyle-related factor for inflammatory periodontal disease destruction.[22]

3. **Biological Mechanisms Linking Obesity with Periodontal Disease.** The mechanism of how obesity modifies the pathogenesis of periodontal disease at the molecular level is currently under review. But what is known is that obesity triggers several harmful biological effects that might overlap with the pathogenesis of periodontitis.[23]

a. **Release of Proinflammatory Cytokines from Adipose Tissue.** Further studies are needed to determine if obesity is a true risk factor for periodontal disease. Nevertheless, several well-known researchers in the field of periodontal medicine have suggested that the association most likely lies in the commonality of their inflammatory pathways. Recently, Genco and colleagues postulated that the continuous release of proinflammatory cytokines into the systemic circulation from adipose tissue in obese individuals leads to a "systemic inflammatory overload."[16] This relentless release of cytokines provides a possible explanation of how obesity intensifies infections, including periodontal disease.

1. Adipose tissue secretes several cytokines and hormones that are involved in inflammatory processes, suggesting that these pathways are involved in the pathogenesis of obesity and periodontitis. The adverse effect of obesity on the periodontium may be mediated through proinflammatory cytokines such as interleukins (IL-1, IL-6, and TNF-α), adipokines (also known as adipocytokines—cell-signaling proteins; i.e., leptin and adiponectin), and reactive oxygen species (ROS) that may affect the periodontal tissues directly.[24]

a. Prior to the 1990s, adipose tissue was generally thought to be dormant and inactive tissue that primarily served to store fats, which were available as a reserve source of energy. Research in the 1990s, however, sparked a reevaluation of how dormant and inactive adipose tissue really is. Adipose tissue is now viewed as a complex, dynamic, metabolically active endocrine organ composed of a variety of cell types. In addition to energy storage, it is now recognized as being involved with metabolic regulation and in immune function.[8,25]

b. Adipose tissue is a complex of adipocytes (fat cells) with stromal and vascular elements infiltrated with resident lymphocytes and macrophages. It has an extensive distribution throughout the body and may be considered the largest endocrine tissue in the body. In obesity, there is an increase in both adipocyte numbers and adipocyte size.[8]

c. Adipocytes secrete hormones, growth factors, cytokines, and enzymes. These signaling proteins can affect the overall systemic inflammation and the body's sensitivity to insulin, in turn affecting the whole-body metabolism. Adipose tissue also differs in its ability to produce cytokines based upon its location in the body.

d. Both harmful and beneficial cytokines can be released from adipose tissue. On the one hand, adipocytes release adiponectin (which is a well-known homeostatic factor that regulates glucose levels and lipid metabolism and has an anti-inflammatory effect). On the other hand, adipocytes can produce leptin and proinflammatory cytokines, such as TNF-α and plasminogen activator inhibitor-1 (PAI-1) which propagate the inflammatory response. In addition, the resident macrophages also secrete TNF-α and IL-6 into the systemic circulation.

2. Adiponectin is the most abundant circulating adipokine secreted exclusively by adipose tissue. While it primarily regulates glucose and lipid metabolism, it also plays important roles in 1) stimulating hunger and appetite by acting on the hypothalamus in the brain and 2) attenuating inflammation by acting upon macrophages to suppress TNF-α production while stimulating these very same immune cells to release the anti-inflammatory cytokine IL-10. This is why adiponectin is widely recognized to have anti-diabetic, anti-inflammatory, anti-atherogenic, and cardioprotective and neuroprotective effects.
   a. While the exact reason has yet to be clearly determined, adiponectin levels are *decreased* in obese individuals. This could be a reason why lower levels of adiponectin are associated with obesity-related diseases, such as type 2 diabetes and coronary artery disease.[26]
   b. Additionally, low levels of adiponectin are associated with increased systemic inflammation.[27] This is because prolonged obesity-associated inflammation suppresses production of adiponectin which perpetuates inflammation.
3. Leptin is another circulating adipokine. However, unlike adiponectin, leptin plays a role in 1) suppressing the appetite and 2) enhancing inflammation. With respect to appetite, leptin serves as the "signal" that is relayed to an individual's brain when he or she is full, thereby suppressing appetite. For this reason, leptin is also called a satiety hormone (satiety means the sensation of feeling full.)
   a. Physiologically, as an individual eats, leptin levels gradually increase. Pathologically however, unlike adiponectin, leptin production is *elevated* in obese individuals due to the greater number of adipocytes which actively produce greater levels of leptin (known as hyperleptinemia). While this may seem counterintuitive, obese individuals are actually resistant to the effects of leptin (known as leptin resistance), thereby inhibiting the suppression of their appetite.[8] Many researchers consider leptin resistance to be one of the features contributing to pathological complications associated with obesity.[27]
   b. On the other hand, compared to obese individuals, lower levels of leptin are observed in lean individuals and in individuals practicing fasting.
   c. In a recent study, serum leptin levels were significantly higher in obese patients with chronic periodontitis compared to nonobese individuals with periodontitis. Further, the same study showed that serum adiponectin levels were significantly lower in obese individuals with periodontitis compared to nonobese individuals with periodontitis, though both the parameters were independent of each other.[28]
   d. Along with playing an appetite suppressive role, leptin has important multiple proinflammatory roles such as activating monocytes and macrophages, promoting immune cell chemotaxis, and regulating the activities of immune cells to phagocytes and produce cytokines that perpetuate the inflammatory response.
4. Obesity-associated TNF-α is secreted from both the adipocytes and the macrophages present within the adipose tissue. As people gain weight, there is an increase in the number of adipocytes and macrophages within their adipose tissue, which leads to elevated release of proinflammatory mediators.
   a. This secretion from the adipose tissue is thought to be the greatest contributor of TNF-α to the systemic circulation. It is thought that increased circulating TNF-α from adipose tissue contributes to general systemic inflammation.[10]
   b. TNF-α has been highly implicated in insulin resistance. It is also seen at elevated levels in periodontitis.[10]
5. About 30% of circulating IL-6 is thought to be derived from adipose tissue. Increasing levels of obesity stimulates IL-6 secretion.[29]
   a. An increased risk of developing coronary artery disease is observed with elevated levels of IL-6.[29]
   b. Inflamed periodontal tissues also demonstrate higher levels of IL-6.[30] IL-6 has been shown to stimulate osteoclasts and thus contribute to the alveolar bone loss seen in periodontitis.
6. PAI-1 has been shown to increase in the systemic circulation as adipose tissue increases. It is a protein involved in fibrinolysis and contributes to atherosclerosis. It has been shown to be elevated in periodontal disease.[8]

b. **Increased Levels of Reactive Oxygen Species**

1. **Reactive oxygen species (ROS)** are chemically active oxygen-containing molecules. ROS can have either physiologic or pathologic properties based on their concentration. ROS have an important role in normal cellular processes and are produced during normal mitochondrial metabolism. At low

concentrations, ROS function in cell signaling and allow for proper tissue homeostasis, helping to direct normal cellular proliferation, differentiation, and metabolism. At lower levels, ROS can stimulate the growth of epithelial cells and fibroblasts present in the periodontal tissues.[31]

2. Higher concentrations of ROS, however, can lead to tissue damage. Higher levels of ROS can be generated by various cells through exposure to cytokines, bacteria or bacterial products, and tobacco products, resulting in elevated levels of ROS with resultant tissue destruction. ROS are also released upon platelet activation and from phagocytic cells such as neutrophils and macrophages. This elevation of ROS can lead to periodontal tissue damage.[31]
3. Damage to the periodontal tissues can be prevented or reduced by having adequate stores of antioxidant molecules within the tissues. These antioxidants include vitamins C and E, β-carotene, selenium, uric acid, and glutathione. These antioxidants can help to prevent the formation of ROS or can help convert them to less reactive forms.[32]
4. An imbalance between ROS production and the antioxidant capacity of the periodontal tissue can lead to the development of periodontal disease. It is possible that periodontal inflammation results from the body's stores of antioxidants being depleted. The levels of antioxidants in gingival crevicular fluid were determined to be statistically lower in patients with periodontal disease compared to age- and sex-matched controls with periodontal health.[33] In a survey of over 1,200 Western European men aged 60 to 70, a strong association was evident between low levels of antioxidants (such as β-carotene and β-cryptoxanthin) and severe periodontitis.[34]
5. Emerging scientific evidence indicates that obesity appears to contribute to the multifactorial etiology of periodontitis through the increased production of ROS and adipokines. **Figure 18-1** shows a suggested model for the link between obesity and periodontal disease.

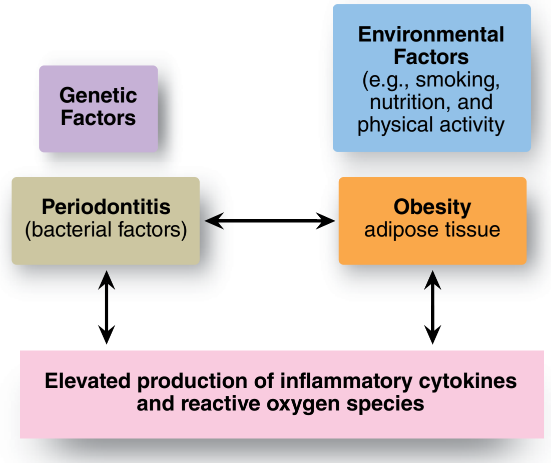
Genetic Factors
Environmental Factors (e.g., smoking, nutrition, and physical activity
Periodontitis (bacterial factors)
Obesity adipose tissue
Elevated production of inflammatory cytokines and reactive oxygen species

Figure 18-1 Hypothesis Linking Obesity and Periodontal Disease. An emerging hypothesis linking obesity with periodontal disease is that both conditions are associated with an elevated production of both inflammatory cytokines and ROS. Of course, both conditions also are affected by other factors such as genetic factors and environmental factors, which makes identifying a precise link between them difficult.

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4. **The Role of the Dental Team in Managing Obesity and Periodontal Disease**
   a. All members of the dental team should be aware of the rising rate of obesity and the significance of obesity as a risk factor for periodontal disease. Addressing obesity in the management of periodontal disease is clearly important in patients who have both conditions. This necessitates the cooperation and collaboration of all health care professionals to educate patients regarding the implications of obesity and periodontitis and to encourage counseling, weight reduction, and treatment.

Section 2

Micronutrients, Antioxidants, and Periodontal Disease

Nutrients are divided into six categories: vitamins, minerals, proteins, lipids, carbohydrates, and water. **Micronutrients** are nutrients that are needed in very small amounts (in microgram to milligram quantities). Vitamins and minerals (like calcium) are micronutrients. Proteins, lipids, and carbohydrates are **macronutrients**, which provide energy or calories and are ingested in larger (in grams) quantities.

1. **Micronutrients and Oxidation**
   a. **Oxidation.** As oxygen interacts with cells of any type—such as the cells of a ripening banana or a cell in the body—oxidation occurs. Oxidation produces some type of change in the cell. The banana may rot; dead body cells are replaced with new cells.
      1. Oxidative stress occurs with the production of harmful molecules called free radicals.
      2. Free radicals containing oxygen, known as reactive oxygen species (ROS), are the most biologically significant free radicals.[32]
      3. It is impossible for the cells of the body to avoid damage by free radicals. Free radicals arise from sources both inside and outside the body. Oxidants develop from processes within the body as the result of normal metabolism and inflammation. Outside the body, free radicals form from environmental factors such as pollution, sunlight, smoking, and radiation.
      4. In response to plaque biofilm, polymorphonuclear leukocytes (PMNs) produce ROS during phagocytosis as part of the host response to infection.[35] These ROS can cause damage to proteins causing fragmentation, peroxidation of lipids, and strand breakage of DNA.
         a. Individuals with periodontal disease have increased numbers of hyperactive PMNs.
         b. It has been suggested that this proliferation results in a high degree of ROS release, culminating in heightened damage to gingival tissue, periodontal ligaments, and alveolar bone.[36]
         c. Studies also have suggested the ROS stimulate osteoclast activation.[37] Compared with healthy controls, patients with periodontitis generate higher levels of ROS. Several studies have demonstrated a correlation between ROS and periodontal disease activity. It is likely that both the periodontopathic bacteria and the host response are responsible for damage mediated by ROS.[32,38–40]
         d. The molecule nuclear factor κB (NF-κB) is a transcription factor that regulates the genes involved in the immune response to infection. It has been shown to be elevated in patients with periodontitis compared with healthy controls.[41]
      5. The damage mediated by ROS can be counteracted by antioxidants.[42]
   b. **Antioxidants. Antioxidants** are substances that counteract the damaging effects of oxidation in a living organism.
      1. Nature offers thousands of natural antioxidants in fruits, vegetables, nuts, whole grains, and legumes to help protect the body from oxidation.
      2. Dietary antioxidants include vitamins and minerals as well as enzymes (proteins in the body that assist in chemical reactions). Some of the common antioxidants include β-carotene, vitamin C, vitamin E, and selenium.
      3. Data from research suggests that there are mechanisms in which nutrition, particularly antioxidants, can influence periodontal disease onset, progression, and wound healing.[43] Antioxidants are thought to be important in the downregulation of proinflammatory responses. Although the research is still ongoing, there is growing evidence that nutrients can act as antioxidants that may modulate gingival inflammation.[42,43]
2. **Vitamins and Minerals**
   a. **Vitamins.** Vitamins are organic compounds that are not synthesized by the body and are necessary for normal metabolism. Vitamin C is the most common water-soluble antioxidant; vitamin E is one of the most common fat-soluble antioxidants.
      1. **Vitamin C**
         a. Vitamin C is a water-soluble vitamin that cannot be stored by the body.
            1. All plants and most animals can synthesize vitamin C from glucose. However, humans, nonhuman primates, and a few other animals do not have the appropriate enzymes to synthesize

vitamin C. Because the human body lacks the ability to synthesize and store vitamin C, we depend on dietary sources to meet our vitamin C needs.

  2. Vitamin C is found in fresh fruits and vegetables such as oranges, berries, tomatoes, leafy greens, and kiwifruit. Consumption of fruits and vegetables or fortifying diets with vitamin C supplements is essential to avoid ascorbic acid deficiency.

b. Vitamin C is also known as ascorbic acid. Vitamin C is well-known for having a curative effect on scurvy. Scurvy is a vitamin C deficiency disease that can lead to muscle weakness, painful and swollen joints, an increased risk of bone fractures, poor wound healing, gingival inflammation, and a weakened periodontal ligament. Oral supplementation or IV supplementation of vitamin C can reverse the detrimental effects of scurvy.

c. The recommended dietary allowance (RDA) for vitamin C is 90 mg for men and 75 mg for non-pregnant/non-lactating women. In certain subsets of the population, the vitamin C RDA increases. For instance, smokers are advised to take an additional 35 mg of vitamin C to offset the oxidative damage caused by smoking. In pregnant women above the age of 18, the vitamin C RDA increases to 85 mg to bolster the general well-being of the pregnant mother and to aid in the neural and bone development of the fetus. In lactating mothers, the RDA increases to 120 mg so that vitamin C can be supplied via breast milk to the newborn to support the continued development of skin, bone, cartilage, and neurological system.

d. Dr. James Lind, a British Royal Navy surgeon, discovered in 1753 that through the addition of citrus fruits to the normal shipboard diet he could prevent the development of scurvy in the sailors. Due to the presence of citrus fruits carried by British ships, British sailors received the name of "limey."[44]

e. Vitamin C has effective antioxidant properties that are important for maintaining the integrity of cell membranes and protection against the ROS generated during inflammatory responses.[45]

f. In addition, vitamin C can enhance the overall immune system by priming neutrophil, monocyte, and natural-killer (NK) cell functions.[46]

g. Data from NHANES III showed that a lower level of intake of vitamin C leads to an increased risk of having periodontal disease in both current and former smokers.[47] Further analysis demonstrated that by having an increased serum concentration of vitamin C, there was a reduced risk of developing periodontitis.[48] Dietary intake of vitamin C showed a weak, but statistically significant, relationship with periodontal disease in current and former smokers as measured by clinical attachment. Cross-sectional studies show that low serum levels of vitamin C led to a higher relative risk for periodontal disease and to the number of teeth with periodontal disease progression.[49,50]

h. In scurvy, mature collagen fibers fail to develop, thereby resulting in the predominance of immature collagen in the bodily tissues. Vitamin C is needed to modify the amino acids, proline and lysine, for collagen maturation to occur. Collagen is a major component of the periodontium. In ascorbic acid deficiency gingivitis, patients present with tooth mobility (due to the presence of weakened, immature periodontal ligament fibers).

i. **Ascorbic acid deficiency gingivitis** is an inflammatory response of the gingiva caused by plaque biofilm that is aggravated by chronically low vitamin C levels.

  1. Ascorbic acid deficiency gingivitis manifests clinically as bright red, swollen, ulcerated gingival tissue that bleeds with the slightest provocation.[51]
  2. **Figures 18-2** and **18-3** show a 39-year-old woman who presented with severe signs of oral scurvy. Her typical diet consisted of macaroni and cheese, peanut butter, and soda. She could not recall when she had consumed a fruit or vegetable. Her serum vitamin C level was about 10 times below average.[52]

j. Rebound scurvy is an extremely rare condition that may potentially develop in individuals who had routinely taken excessive dosages of vitamin C for a prolonged period of time, but suddenly discontinued them. Due to the abrupt cessation of vitamin C intake, scurvy may develop because the body cannot physiologic adapt to the sudden reduction in vitamin C. Reinstituting the regular dietary allowance of vitamin C can reverse this, and the gingival tissues can return to normal.[53] Newborns whose mothers had taken heavy doses of vitamin C during their pregnancy may be at high risk of rebound scurvy. Following birth, the baby can develop scurvy due to a reduction in vitamin C blood levels because they are no longer getting the higher levels of vitamin C from the maternal circulation.[54]

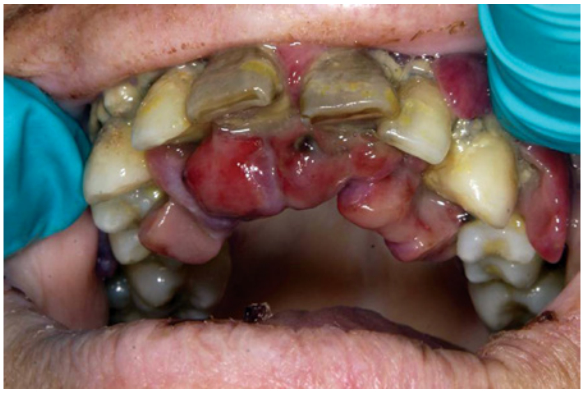

Figure 18-2 Adult Patient with Scurvy. Facial view of the gingival tissues on a 39-year-old woman with scurvy.

Courtesy of Dr. William J. Dunn, Col., USAF, DC, Military Consultant to the Surgeon General for Medical Ethics, Keesler Air Force Base, MS 39434.

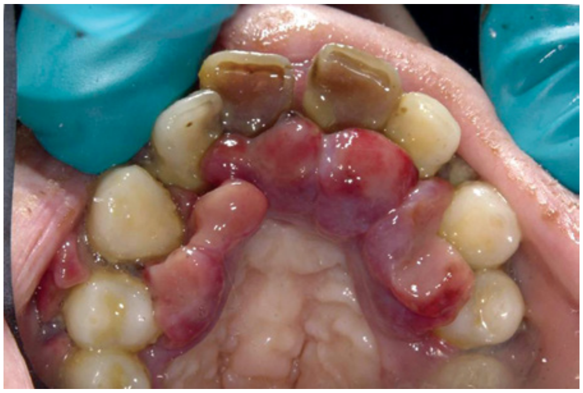

Figure 18-3 Adult Patient with Scurvy. Lingual view of the gingival tissues on a 39-year-old woman with scurvy.

Courtesy of Dr. William J. Dunn, Col., USAF, DC, Military Consultant to the Surgeon General for Medical Ethics, Keesler Air Force Base, MS 39434.

2. **Vitamin A**
   a. Vitamin A is a fat-soluble vitamin needed for epithelial cell differentiation (especially mucus-secreting glands), bone remodeling, cell-mediated immunity and T-helper cell differentiation, and anti-inflammatory response.[55]
   b. Vitamin A has a key role in epithelial maintenance. A lack of vitamin A can lead to a reduction in mucous-containing salivary and lacrimal secretions. The reduction in lacrimal secretions is a leading cause of blindness in third world countries.[56]
   c. Severe vitamin A deficiency, however, is rare in industrialized nations. The NHANES III study found no significant relationship between serum vitamin A and risk for periodontitis.[57]
   d. Vitamin A is found primarily in animal fats and fish oils as retinoids, which can be toxic when taken in high doses. β-carotene is present in fruits and vegetables and can be converted in the body into vitamin A. β-carotene also is an antioxidant, but vitamin A is not. Foods high in vitamin A or β-carotene include milk, eggs, liver, carrots, pumpkin, and mangos.
   e. In a case report, a 20-year-old woman exhibited gingival ulcerations, bleeding, swelling, and xerostomia. Headache and hair loss were also noted. The woman reported taking 200,000 IU of vitamin A daily for 6 months. When the vitamin supplementation was stopped, improvement in the gingival tissues was noted within 1 week. At 2 months, the appearance of the oral tissue was normal.[58]
3. **Vitamin D**
   a. Vitamin D can be obtained from diet and also be synthesized in the body via sunlight exposure. In countries furthest away from the equator (like Nordic countries), sunlight is often inadequate, especially during the winter months, necessitating the intake of vitamin D–rich food/drinks. Vitamin D helps to enhance calcium and phosphate absorption, maintain blood calcium levels, aids in the calcification of teeth and osseous tissues, and acts as an anti-inflammatory agent. Good sources of dietary vitamin D include egg yolk, liver, and oily fish. In some countries, cow's milk is a rich source as it is fortified with vitamin D.
   b. Vitamin D promotes the differentiation of monocytes to macrophages.[59]
   c. NHANES data on 6,700 never smokers were analyzed. The subjects with the highest vitamin D levels presented with less gingival inflammation and less bleeding on probing.[60] Vitamin D may reduce the susceptibility to gingival inflammation through its anti-inflammatory effects.[61]
   d. A 3-year clinical study of subjects aged 65 or older evaluating the use of supplemental vitamin D and calcium to prevent hip fracture, also looked at tooth loss. The researchers noted that when taking calcium and vitamin D to prevent osteoporosis and hip fracture that subjects also lost fewer teeth. Twenty-seven percent of subjects taking a placebo lost at least one tooth, while only 13% of subjects taking the supplements lost teeth.[62]

4. **Vitamin E**
    a. Vitamin E is considered the most important fat-soluble antioxidant. It protects cell membranes from oxidative damage and reduces the production of prostaglandin E2 in macrophages.[63] Vitamin E is present in the lipid membrane of the cells helping to quench free radicals that can cause oxidative damage to the fatty acids in the membrane.
    b. Recent in vitro studies show vitamin E to have mitigating effects on gingival inflammation.[36]
    c. Several clinical studies have shown positive effects of supplementation of vitamin E on the immune response, particularly in aged populations.[64]
    d. Good dietary sources rich in vitamin E include nuts, seeds, and wheat germ.
5. **Folic Acid**
    a. Folic acid is a water-soluble vitamin belonging to the B-complex group of vitamins. Except for folic acid, there is little evidence that B vitamins are vital to periodontal health.
    b. Folic acid is involved with DNA synthesis. Cells with rapid turnover, such as those in the gingival epithelium, have high folic acid requirements.
    c. A deficiency of folic acid may reduce the ability of the gingival tissue to act as a barrier to bacteria.
    d. NHANES data on serum folate levels and periodontal disease were evaluated for 844 subjects. There was a statistically significant negative association between folate level and the presence of periodontal disease. A low serum foliate was independently associated with periodontal disease in older adults.[65]
    e. During the milling process, folic acid is removed from the grain. In Canada and the United States, grains marketed as enriched have been fortified with folic acid since 1998. It has been suggested that if all women entered pregnancy with optimal levels of folic acid, up to 70% of neural tube birth defects in the United States could be prevented.[66] Other good dietary sources include broccoli, asparagus, brussels sprouts, peas, and brown rice.

b. **Minerals**
1. **Calcium**
    a. The micronutrient calcium is intricately involved in the calcification process and has an important role in the formation and maintenance of alveolar bone. Nishida and colleagues found a 54% increased risk of periodontal disease in females who ingested less than 499 mg per day of calcium and a 27% increased risk in those with intakes below 800 mg per day.[67] The RDA for calcium is 1,000 mg for adults aged 19 to 50.
    b. In another interesting longitudinal study, elderly adults with higher daily calcium intake had a decreased risk for tooth loss than adults who consumed lower calcium levels.[62]
    c. Consumption of milk, yogurt, and cheese (preferably lower-fat varieties) is important in ensuring adequate intakes of calcium. Data from NHANES III showed that individuals who consumed higher levels of dairy products had a lower level of periodontal disease.[68,69]
2. **Magnesium**
    a. Magnesium is found in all cells and is concentrated in the mitochondria given that it is involved in energy transfer reactions.
    b. The periodontal health of 60 subjects taking magnesium-containing medications was compared to 120 controls. It was noted that the subjects taking the magnesium-containing drugs had a greater number of teeth, shallower pockets depths, and less loss of periodontal attachment than the controls.[70] These results suggest that nutritional magnesium supplementation may improve periodontal health in individuals aged 40 years and older.

Section 3

Macronutrients and Periodontal Disease

Proteins, lipids, and carbohydrates are macronutrients, which provide energy or calories. These are consumed in gram quantities versus the micronutrients, which are consumed in milligram to microgram quantities. Many of the pathways to inflammation are related to high caloric intake from refined carbohydrates and oxidative stress, which is stimulated by changes in the metabolism of adipose tissue.[71] While diet plays a *primary role* in the development of dental caries, diet primarily plays a *modifying role* in the progression of periodontal disease. Further, although bacteria are the primary etiologic factors for periodontal disease, a susceptible host is also necessary for disease initiation. Nutrition may be a modifying factor that impacts the host's immune response and the integrity of the tissues of the periodontium.[72]

1. **Proteins**
    a. Protein deficiency can have an effect on the host defenses that modulate the progress of periodontitis. Proteins are the building blocks to many of the host's defensive barriers, such as the junctional and crevicular epithelium. They also contribute to other host immune responses, such as cell-meditated immunity, humoral or antibody mediated immunity, cytokines, and complement molecules. These defenses are impaired with a protein deficiency.
    b. The effect of protein-energy malnutrition on periodontal disease risk was extensively reviewed by Enwonwu and colleagues, who observed that periodontal disease was more prevalent and severe in undernourished individuals than in well-nourished ones.[73] The immune depression that occurs in protein-energy malnutrition promotes vulnerability of the periodontium to inflammatory stimuli from the plaque biofilm.
2. **Refined Carbohydrates and Lipids.** Diets rich in refined carbohydrates and saturated fats may play a role in increasing the risk for periodontal disease. Excessive energy intake causes obesity, which has been shown to be associated with increased risk of periodontitis.
    a. Excess consumption of refined carbohydrates can affect the immune response and may lead to continued destruction of the periodontium in patients with existing periodontitis through mechanisms such as the action of enzymes (i.e., collagenase) and proinflammatory mediators (i.e., IL-1 and IL-6).[71] These destructive mechanisms have been discussed in other chapters of this book.
    b. Diets high in refined carbohydrates and saturated fats cause rapid release of glucose into the bloodstream. High blood glucose levels increase triglyceride levels and stimulate the release of insulin, which decreases the ability of the body to break down fat stored in adipose depots.[71]
    c. As discussed earlier, obesity increases the circulation of ROS, which in turn, causes oxidative damage, and progression of periodontal disease.[23]
    d. Unlike saturated fats (fats commonly found in animal products such as cheese, whole milk, fatty meats, and butter and oils such as coconut oil palm oil, and palm kernel oil), which are associated with a higher risk of inflammation, some polyunsaturated lipids, specifically omega-3 fatty acids, exhibit anti-inflammatory properties and, as a result, have health benefits. Omega-3 fatty acids are classified as essential fats, which means that the body cannot produce them from scratch and must obtain them from food. They are found at high levels in fish, vegetable oils, flaxseed oils, and nuts. The two main type of omega-3 fatty acids are eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA).
        1. In a recent study, rats were infected with *Porphyromonas gingivalis* and received a diet containing either fish oil having omega-3 fatty acids or corn oil. Rats receiving the omega-3 fatty acid diet showed less alveolar bone loss and reduced expression of IL-1 and TNF-α. Thus, in the rat model, omega-3 fatty acids reduced the inflammation associated with infection with *P. gingivalis*.[76,77]
        2. NHANES data was evaluated to see if there was a relationship between periodontal disease and the intake of omega-3 fatty acids in 9,182 adults. The data showed that having a higher level of dietary omega-3 fatty acids was associated with a lower level of periodontitis.[74]
        3. Fifty-five adults with moderate periodontitis were enrolled in a study and received capsules containing the omega-3 fatty acid, DHA, or a capsule with a mixture of soybean and corn oil. Both groups also took 81 mg of aspirin daily and received oral hygiene instruction at the start of the study. No periodontal treatment was provided. At the end of 3 months, the group receiving the omega-3 fatty acid capsules had lower pockets depths and a lower gingival index.[75]
3. **The Role of the Dental Team in Promoting Good Nutrition**
    a. Making an appropriate connection between diet, nutrients, and periodontal disease can allow clinicians to make specific suggestions to improve an individual's diet. When indicated, nutritional counseling, with an emphasis on periodontal health and root caries prevention, should be routinely offered to all patients.

Educating patients about good dietary habits can be accomplished using standard diet forms, counseling techniques, and analysis procedures.

b. **Table 18-1** summarizes the possible association between diet/nutrition and periodontal disease.

Table 18-1 Summary of the Association Between Nutrition and Periodontal Disease

Dietary Component	Association with Periodontal Disease
Dietary antioxidants	Antioxidant status is compromised in periodontal disease.[32,33]
Vitamin C	An epidemiologic study demonstrated a direct association between low vitamin C intake and periodontal disease, especially among smokers.[47]
Vitamin D	An epidemiologic study demonstrated a positive association between low vitamin D intake and gingival inflammation. The anti-inflammatory effects of vitamin D may contribute to a reduction in gingival inflammation.[60,61]
Vitamin E	In vitro studies show that vitamin E can dampen gingival inflammation.[36,64]
Folic acid	Supplementation with folic acid reduces gingival inflammation.[65]
Calcium	Lower intake of calcium has been shown to be independently associated with increased risk of periodontitis.[62,67,68]
Magnesium	Subjects taking magnesium-containing medications have shallower pocket depths and less gingival inflammation.[70]
Protein	Protein malnutrition increases risk and enhances progression of periodontal disease.[73]
Refined carbohydrates and lipids	Excessive energy intake causes obesity, which has been shown to be associated with increased risk of periodontitis. Obesity increases the circulation of ROS, which, in turn, causes oxidative damage and progression of periodontal disease.[23,24,31]
Omega-3 fatty acids	Epidemiologic data shows that a higher intake of omega-3 fatty acids results in a reduced risk of having periodontitis. Subjects taking omega-3 fatty acid in a research study had lower pocket depths and less inflammation after 3 months.[74,75]

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Chapter Summary Statement

While nutritional deficiencies do not cause periodontal disease, some nutritional deficiencies do indeed appear to modify the severity and extent of the periodontal disease. Research has linked specific nutrient deficiencies and foods high in refined carbohydrates to increased inflammation—the very kind that triggers the host-mediated inflammatory response seen in periodontal disease.

Reports of the association between obesity and periodontal disease are strengthening. At this time, it is not possible to determine whether obesity predisposes an individual to periodontal disease or periodontal disease affects lipid metabolism, or both. Future longitudinal studies with more precise measures of adiposity on large populations are needed to clarify whether obesity is one of the risk factors for periodontal disease or simply a risk indicator.

Research suggests that there are mechanisms in which nutrition, particularly antioxidants, can influence periodontal disease onset, progression, and wound healing.

It is wise to counsel patients with periodontal disease to be thoughtful food shoppers and to prepare meals and snacks that are rich in specific nutrients. A nutritionally adequate diet will help maintain host immunity and preserve the integrity of the periodontal tissues. At present, there is insufficient evidence to justify treatment with vitamin and mineral supplementation in the adequately nourished, healthy individual. Nevertheless, a continued focus on an individual's diet, with the consumption of foods rich in essential macronutrients and micronutrients, will continue to be an essential component of any dental evaluation.

Section 4

Focus on Patients

Clinical Patient Care

Directions for Clinical Patient Cases: Analyze the information and 24-hour diet recall (**Tables 18-2** and **18-3**) for the two fictitious periodontal patients to answer the following questions:

1. Can you identify any eating patterns (healthy or unhealthy)? If so, what are they?
2. List diet practices that have the potential to be most detrimental to the individual's periodontal health.
3. List two diet behaviors that the individual could modify to benefit his or her periodontal health.

CASE 1

Fictitious Periodontal Patient A

Patient A is 39 years old. The patient is 5′8″ (173 cm) in height and weighs 280 pounds (127 kg). The periodontal diagnosis is generalized Stage I, Grade A periodontitis.

Table 18-2 **Patient A: 24-Hour Diet Recall**

Time	Food/Beverage	Amount	Reason
6:00 AM	2 cups of coffee with nondairy creamer and 2 packets of sugar Honey Nut Cheerios cereal and milk	2 medium cups Medium-sized bowl	Breakfast
10:00 AM	Apple danish pastry with icing		Snack
12:00 /PM	Double hamburger with cheese, lettuce, tomato, and onion French fries Regular Coke	Supersize Large Large	Lunch
2:00 PM	Regular Coke Bag of potato chips	1 can Small bag	Snack
7:00 PM	Steak French fries Tossed salad with Italian dressing Garlic toast Water	Large Large serving Small bowl 2 pieces	Dinner
9:00 PM	Ice cream	Medium bowl	Snack while watching TV

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CASE 2

Fictitious Periodontal Patient B

Patient B is 70 years old. The patient is 5′1″ (155 cm) in height and weighs 160 pounds (73 kg). The periodontal diagnosis is generalized Stage III, Grade B periodontitis.

Table 18-3 **Patient B: 24-Hour Diet Recall**

Time	Food/Beverage	Amount	Reason
7:00 AM	Coffee with organic nondairy cream Whole-grain English muffin Peanut butter and jelly	1 large mug 1	Breakfast
10:30 AM	Green grapes	25	Hungry
11:00 AM	M&Ms	3 handfuls	Stressed (arguing with daughter on the phone)
12:30 PM	Philly cheese steak (fried shaved beef, onions, and green peppers, covered with cheese sauce in a large white "hoagie" bun) Potato chips Iced tea sweetened with sugar	 One bag One glass	Lunch
2:00 PM	Caramel-coated popcorn mix	2 handfuls	Stressed/hungry
6:00 PM	Fried hamburger patty with brown gravy Baked potato with sour cream and real bacon bits Peas Coffee with sugar		Dinner
7:00 PM	Glass of water		Thirsty
8:00 PM	Popcorn with butter	Medium bowl	Watching a movie

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Ethical Dilemma

Stuart Fisher is a 48-year-old patient who is new to the practice. He owns a successful restaurant, where he works as the head chef. As he is self-employed, he doesn't carry dental insurance.

A review of his medical history reveals the following:

- Vitals at today's appointment: 160/96 mm, HR: 82 bpm
- Height: 5′10″ (177.8 cm); Weight: 265 lbs (120 kg), BMI:38.0
- History of hypertension, hyperlipidemia, Type II diabetes mellitus
- Current medications: Hyzaar® (losartan/hydrocholorothiazide) for hypertension, Vytorin® (ezetimibe-simvastatin) for hyperlipidemia, Januvia® (sitagliptin) for Type II diabetes mellitus
- He states that he is scheduled for lap-band surgery next month to address his abdominal obesity

Due to his busy lifestyle, Stuart admits that he has neglected his general and oral health for many years. He also notes that although he knows better, his diet is poor. After cooking in the restaurant all day and night, the last thing he wants to do is prepare food for himself. So, both before and after work, he stops for "fast food."

Your radiographs and examination reveal generalized horizonal bone loss throughout his mouth, as well as Glickman Grade II furcation involvement on all molar teeth which is consistent with generalized stage III periodontitis.

You review the treatment plan with Stuart, which includes periodontal instrumentation with anesthesia, nutritional counseling, and referral to a periodontist. Stuart refuses to agree with your suggestions, as he states that he doesn't carry dental insurance. He only scheduled this appointment, as his surgeon required it prior to his surgery. He wants to receive the minimal dental treatment and will not "go for" anything more.

1. What is the best way for you to handle this ethical dilemma?
2. What is the best way to address/discuss Stuart's treatment plan with him?
3. What ethical principles are in conflict in this dilemma?

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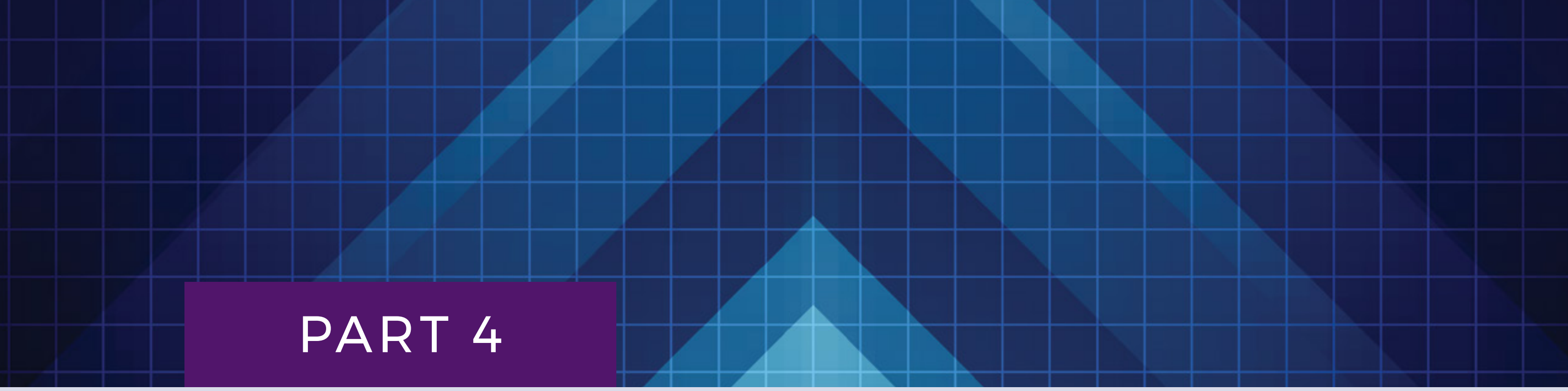

PART 4

Assessment and Planning for Patients with Periodontal Diseases and Peri-Implant Diseases/Conditions

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CHAPTER 19

Clinical Periodontal Assessment

- Section 1 Overview of the Periodontal Assessment Process
- Section 2 The Comprehensive Periodontal Assessment
  - Components of the Comprehensive Periodontal Assessment
  - Components of the Comprehensive Periodontal Assessment That Require Calculations
- Section 3 Periodontal Screening Examination
- Section 4 Supplemental Diagnostic Tests
- Section 5 Focus on Patients
  - Clinical Patient Care
  - Ethical Dilemma

CLINICAL APPLICATION

Clinical periodontal assessment is a critical step in the care of all patients with periodontal diseases. A careful and meticulous clinical periodontal assessment serves as the foundation for assigning a periodontal diagnosis, developing plans for treating patients, and monitoring the success or failure of periodontal treatment. The rigorous standards of care in practice today require every practitioner to be familiar with the details of performing and documenting a clinical periodontal assessment. This chapter describes how to perform a clinical periodontal assessment, how to document the findings of the assessment, and how to perform calculations needed during the assessment.

LEARNING OBJECTIVES

- List the components of a comprehensive periodontal assessment.
- Describe how to evaluate each component of a comprehensive periodontal assessment.
- Be able to calculate the width of attached gingiva.
- Be able to calculate clinical attachment level given several different clinical scenarios.
- Compare and contrast a periodontal screening examination and a comprehensive periodontal assessment.
- Given a clinical scenario, calculate and document the clinical attachment levels for a patient with periodontitis.

KEY TERMS

Clinical periodontal assessment
Comprehensive periodontal assessment
Baseline data
Exudate
Horizontal tooth mobility
Vertical tooth mobility
Fremitus
Furcation probes
Attached gingiva
Mucogingival defects
Clinical attachment level (CAL)
Periodontal screening examination
Periodontal Screening and Recording (PSR)
World Health Organization probe
Gingival crevicular fluid

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Section 1

Overview of the Periodontal Assessment Process

1. **Clinical periodontal assessment** is a fact-gathering process designed to provide a comprehensive picture of the patient's periodontal health status.
    a. **Importance of Periodontal Assessment**
        1. This assessment is one of the most important duties performed by any member of the dental team.
            a. The dental team is obligated to perform and document findings of a periodontal assessment for every patient when he or she first enters the dental practice and periodically thereafter.[1–5]
            b. Periodontal assessment requires meticulous attention to detail because successful patient care is dependent on a thorough and accurate periodontal evaluation.
        2. The information gathered from the clinical periodontal assessment forms the basis of both a periodontal diagnosis and an individualized treatment plan for the patient.
    b. **Objectives of Periodontal Assessment.** The objectives of the clinical periodontal assessment process include the following:
        1. Detect clinical signs of inflammation in the periodontium.
        2. Identify damage to the periodontium already caused by disease or trauma.
        3. Provide the dental team with data used to assign a periodontal diagnosis and a tooth-by-tooth prognosis.
        4. Document features of the periodontium to serve as baseline data for long-term patient monitoring.
    c. **Two Types of Periodontal Assessment.** Two commonly used periodontal assessments are the periodontal screening examination and the comprehensive periodontal assessment.
        1. **Periodontal Screening Examination.** A periodontal screening examination is a quick information-gathering process that may be used to determine if a more thorough comprehensive periodontal assessment (see below) is needed to formulate a diagnosis.
        2. **Comprehensive Periodontal Assessment.** A **comprehensive periodontal assessment** is an in-depth information-gathering process used to gather the detailed data needed to document the complete periodontal health status of a patient.
2. **Standard of Care**
    a. The standard of care for dentists and dental hygienists is to complete an accurate and thorough periodontal assessment on every patient.
    b. Without a thorough clinical periodontal assessment, periodontal diseases are often overlooked or misdiagnosed, inevitably leading to either undertreatment or overtreatment of the patient.
3. **Documentation of Assessment Findings**
    a. The clinical periodontal assessment is not complete until all of the information gathered during the assessment has been accurately recorded in the patient's dental chart.
    b. The importance of the accuracy of the documentation cannot be overstated.
        1. Findings documented during the clinical periodontal assessment serve as baseline data used to evaluate the success or failure of an episode of periodontal therapy. **Baseline data** refers to clinical information gathered at the initial appointment that can be used for comparison to clinical information gathered at a subsequent appointment.
        2. Documented findings also provide the baseline data used in the long-term monitoring of the patient's periodontal health status. An example of when patient monitoring may occur is at periodontal maintenance visits following successful treatment.

Section 2

The Comprehensive Periodontal Assessment

A comprehensive periodontal assessment is an intensive clinical periodontal evaluation used to gather information about the periodontium. This section outlines important clinical features that should be noted and documented during a comprehensive periodontal assessment. Note that special precautions are necessary when examining dental implants. A guideline to conducting a proper implant assessment is provided in Chapter 8.

The comprehensive periodontal assessment normally involves the recording and interpretation of (1) clinical features such as probing depth measurements, bleeding on probing, presence of exudate, level of the free gingival margin, level of the mucogingival junction, tooth mobility, furcation involvement, presence of calculus, presence of plaque biofilms, gingival inflammation, and presence of local contributing factors and (2) radiographic features such as the level of the alveolar crest, pattern of the trabecular bone, width of the periodontal ligament (PDL) space, and presence or absence of the lamina dura. A more in-depth description of radiographic assessment is discussed in Chapter 20. Findings obtained from a comprehensive periodontal assessment will provide vital information of the overall health of the periodontium and enable proper periodontal diagnosis.

Note that there are a number of excellent electronic tools that can be used while performing a comprehensive periodontal assessment; for example, electronic periodontal probes are available that can record probing depths directly in computer software. The discussion in this chapter focuses only on the basic concepts underlying each of the clinical factors being assessed with the knowledge that a clinician who understands the basic concepts can easily apply any of the available electronic tools appropriately.

Components of the Comprehensive Periodontal Assessment

1. **The Gingiva.** The comprehensive periodontal assessment includes evaluation of the health of the gingival tissues, including assessment of tissue color, contour, consistency, and texture. These clinical features are discussed in Chapter 5. The landmarks of the gingival tissues are pictured in **Figure 19-1**.

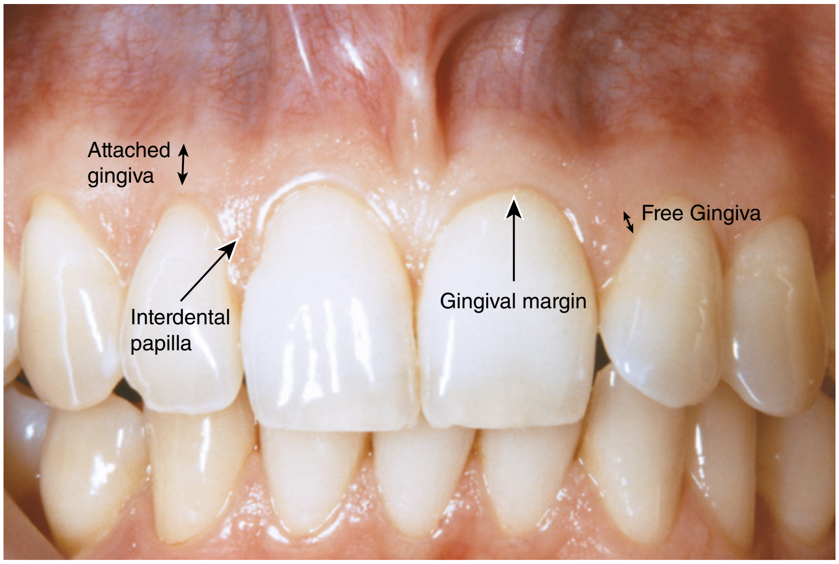

Figure 19-1 Landmarks of the Gingival Tissues. Important gingival landmarks include the gingival margin, the interdental papilla, free gingiva, attached gingiva, and mucogingival junction.

   a. **Gingival Inflammation**
      1. A thorough periodontal assessment includes recording the overt clinical signs of inflammation. The overt clinical signs of inflammation of the gingiva include erythema (redness) and edema (swelling) of the gingiva resulting in readily identifiable changes in gingival color and contour.
      2. It is always important to be aware that inflammation can be present in the deeper structures of the periodontium without necessarily involving any obvious visual signs of inflammation of the gingival margin.
         a. When assessing the presence of inflammation, it is important to remember that bleeding on probing also can be a sign of inflammation.

   b. Thus, when a clinician is identifying gingival inflammation for purposes of planning treatment, the visible signs such as color, contour, and consistency changes in the gingiva must be correlated with the other signs such as bleeding on probing or the presence of exudate.

b. **Level of the Free Gingival Margin.** The level of the free gingival margin in relationship to the cementoenamel junction (CEJ) should be recorded on the dental chart. This level can simply be drawn on the facial and lingual surfaces of the dental chart. The photographs in **Figure 19-2** show examples of the three possible relationships that may exist between the level of the free gingival margin and the CEJ.

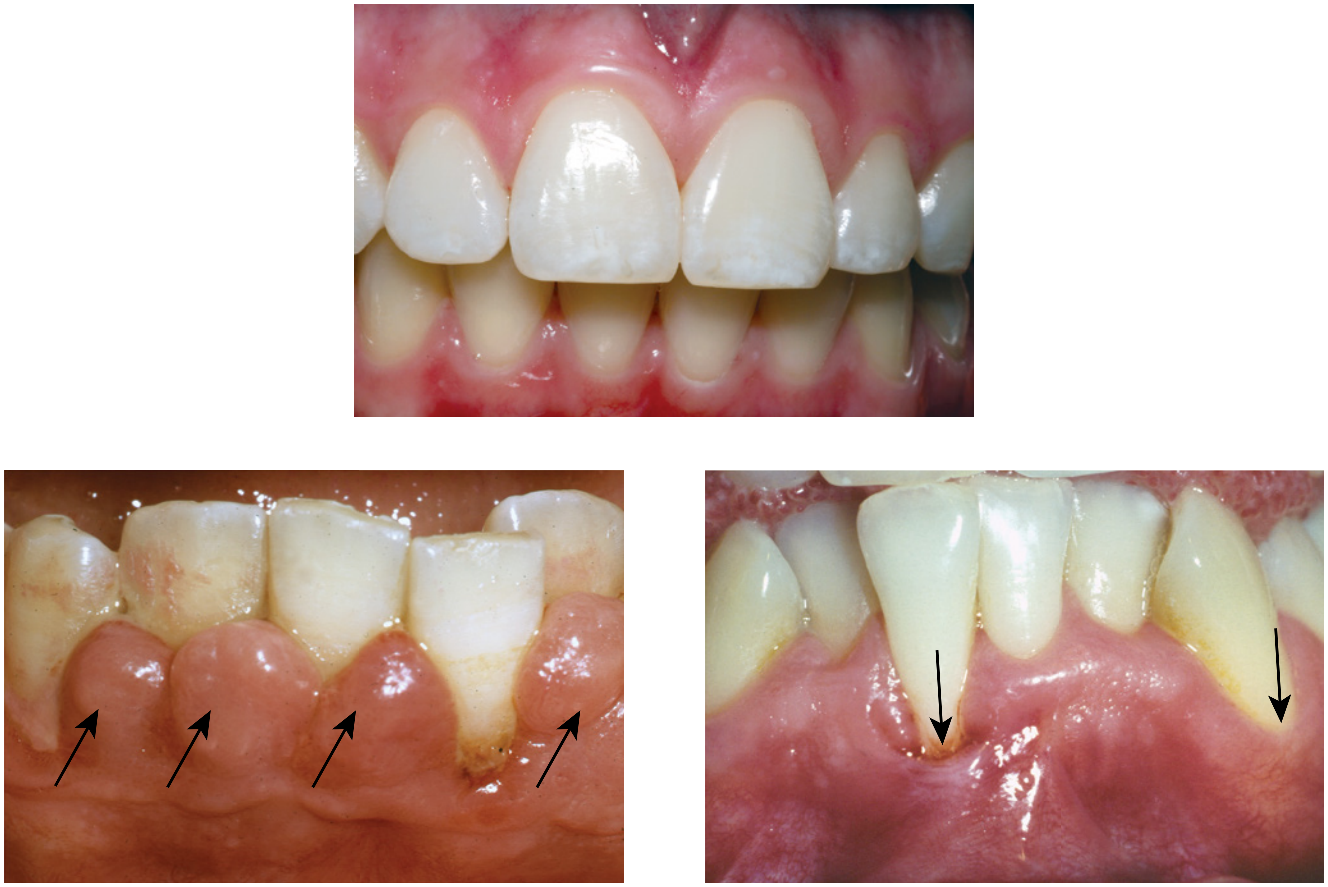

Figure 19-2 Level of the Free Gingival Margin. The photo on the left is an example of the gingival margin in health—slightly coronal to the CEJ. The center photo shows swollen gingiva where the gingival margin is coronal to the CEJ. The photo on the right shows the gingival margin apical to the CEJ on the affected canine and central incisor (recession of the gingival margin).

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1. Free gingival margin can be slightly coronal to (above) the CEJ. This is the natural level of the gingival margin and represents the expected position of the gingival margin in the absence of disease or trauma.
2. Free gingival margin can be significantly coronal to the CEJ. The gingival margin can be significantly coronal to the CEJ due to (1) swelling (edema), (2) overgrowth (as seen in patients taking certain medications), or (3) increase in fibrous connective tissue (as seen in long-standing inflammation of tissue).
3. Free gingival margin can be apical to the CEJ. This relationship, known as gingival recession, results in exposure of the root surface.

c. **Recession of the Gingival Margin**

1. Gingival recession is the displacement of the gingival soft tissue margin apical to the CEJ that results in exposure of the root surface.[6] This is a common clinical condition, and research indicates it presents in at least one or more tooth surfaces in 23% of U.S. adults between 30 and 90 years of age.[6–8]

2. The prevalence of gingival recession has been shown to increase with age and can occur in patients with good standards of oral hygiene as well as those with poor oral hygiene and periodontal disease.[8]
3. The etiology of gingival recession is often multifactorial. Chan and colleagues[9] categorize the etiological factors of gingival recession under predisposing factors and precipitating factors.
    a. Predisposing factors include anatomical factors, inflammation, high frenal attachments, and a lack of adequate keratinized gingiva.
    b. Precipitating factors include traumatic forces (i.e., excessive brushing), habits (i.e., oral piercings), plaque-induced inflammation, and certain dental procedures (such as subgingival restorations).
4. The severity of gingival recession can be classified using the Miller classification system for gingival recession.[10] This classification system is outlined in **Figure 19-3**.

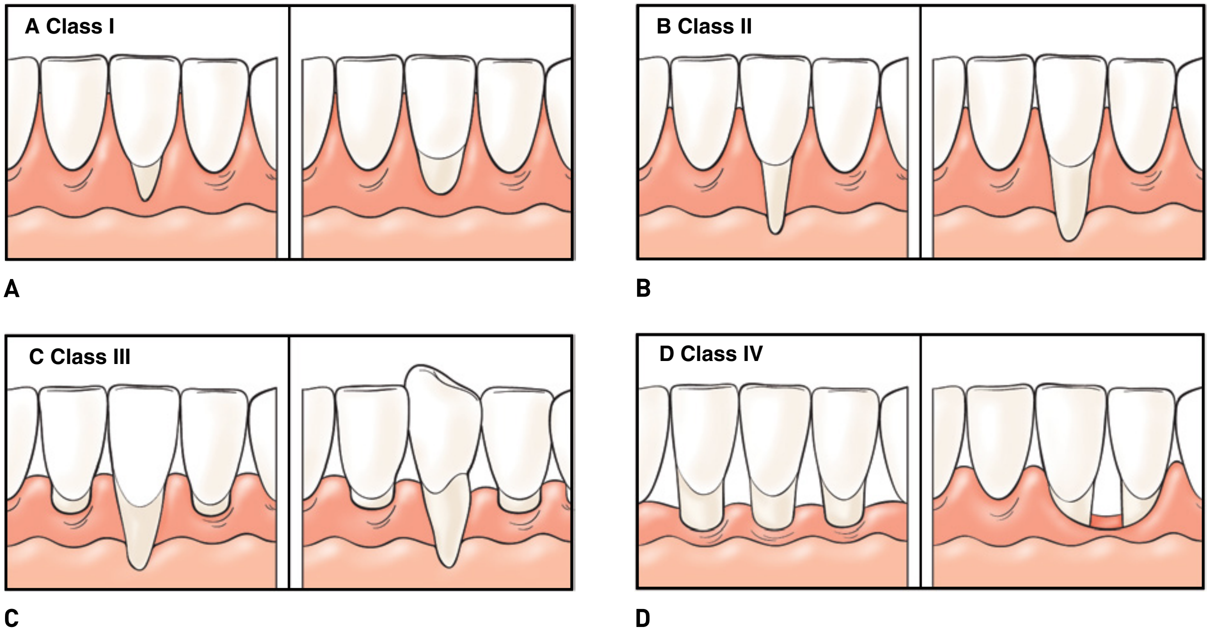

Figure 19-3 The Miller Classification System for Gingival Recession. A. Miller Class I Defect. In a Miller Class I gingival defect, the recession is isolated to the facial surface and the interdental papillae completely fill the adjacent interdental spaces. Class I recession does not extend to the mucogingival line. **B.** Miller Class II Defect. In a Miller Class II gingival defect, the recession is isolated to the facial surface and the papillae remain intact and fill the interdental spaces. Class II recession does extend to or beyond the mucogingival line into the mucosa. **C.** Miller Class III Defect. In a Miller Class III gingival defect, the recession is quite broad with partial loss of the interdental papillae. The Class III defect extends to or beyond the mucogingival line into the mucosa. **D.** Miller Class IV Defect. In a Miller Class IV gingival defect, the recession extends to or beyond the mucogingival junction with severe loss of interproximal alveolar bone resulting in open interdental areas.

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d. Level of Mucogingival Junction

1. The level of the mucogingival junction represents the junction between the keratinized gingiva and the nonkeratinized mucosa. The level of the mucogingival junction is used in calculation of the width of the keratinized gingiva, as will be described later in this section.
2. The mucogingival junction is usually readily visible because the keratinized gingiva is normally pale pink and opaque, whereas the surface of the mucosa appears thin and translucent with small visible capillaries (**Figure 19-4**).

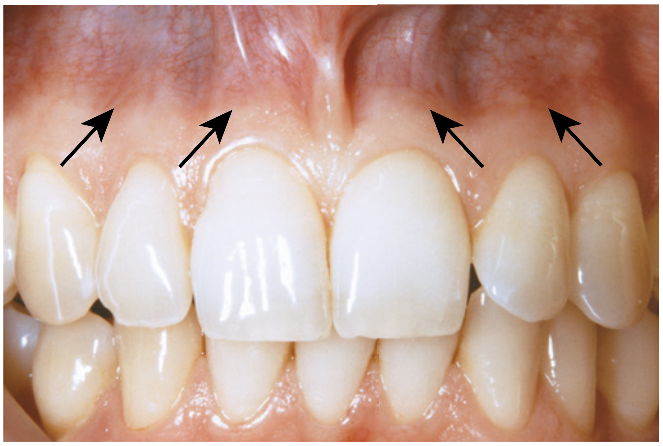

Figure 19-4 Mucogingival Junction. The mucogingival junction represents the junction between the keratinized gingiva and the nonkeratinized mucosa and is usually readily visible.

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3. Occasionally, the mucogingival junction can be difficult to detect visually. In this case, the tissue can be manipulated by pulling on the patient's lip or pushing on the tissue with a blunt instrument to distinguish the moveable mucosa from the more firmly keratinized gingiva.

e. **Probing Depth Measurements.** Probing depth measurements are made from the free gingival margin to the base of the pocket (or base of the sulcus).

   1. Probing depths are recorded to the nearest full millimeter. Measurements are normally rounded up to the next higher whole number (e.g., a reading of 3.5 mm is recorded as 4 mm, and a 5.5 mm reading is recorded as 6 mm).
   2. For every tooth, probing depth measurements are recorded at six specific sites: (i) distofacial, (ii) middle facial, (iii) mesiofacial, (iv) distolingual, (v) middle lingual, and (vi) mesiolingual.

f. **Bleeding on Probing**

   1. Bleeding on gentle probing represents bleeding from the soft tissue wall of a periodontal pocket where the wall of the pocket is ulcerated (i.e., where portions of the epithelium have been destroyed) (**Figure 19-5**).

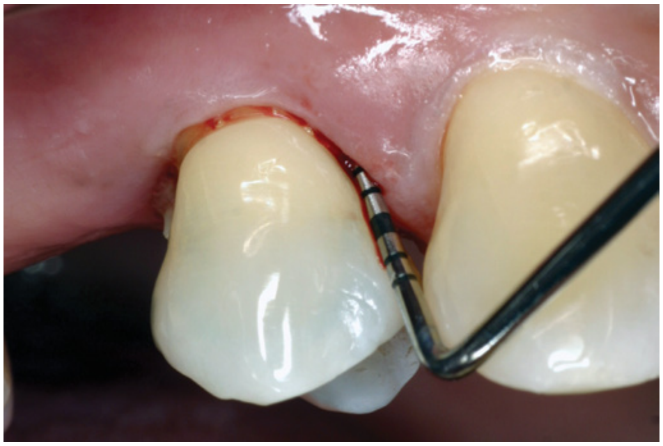

Figure 19-5 Bleeding Site. Bleeding from the soft tissue wall is a sign of disease. This bleeding was evident upon gentle probing.

Courtesy of Dr. Richard J. Foster, Educational Solutions and Design, Greensboro, NC.

   2. Bleeding can occur immediately after the site is probed or can be slightly delayed after probing. An alert clinician will observe each site for a few seconds before moving on to the next site.

g. Presence of Exudate

1. **Exudate** (sometimes referred to as suppuration) is pus that can be expressed from a periodontal pocket. Pus is composed mainly of dead white blood cells and can occur in response to any infection, including periodontal disease.
2. Exudate can be recognized as a pale-yellow discharge oozing from the orifice of a pocket. It is usually easiest to detect when the gingiva is manipulated in some manner. **Figure 19-6** illustrates the use of light finger pressure on the gingiva to express exudate. **Figure 19-7** shows exudate being expressed from the periodontal tissues.

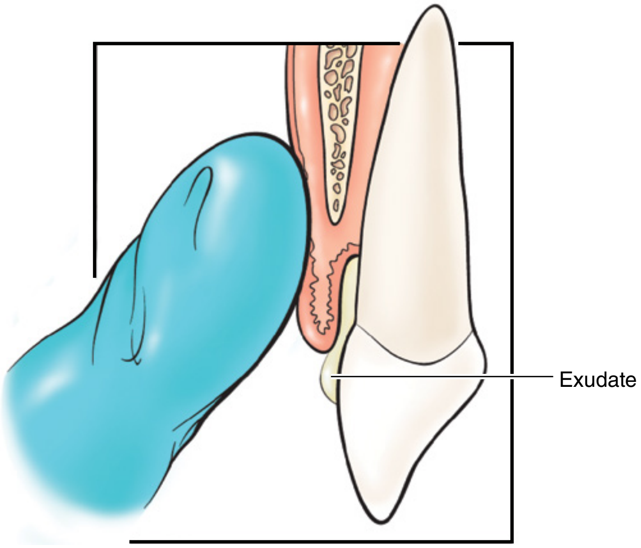

Figure 19-6 Using Finger Pressure to Detect Exudate. Exudate can be detected in a periodontal pocket by placing an index finger on the soft tissue in the area of the pocket and exerting light pressure. This light pressure can force the exudate out of the pocket, making it readily visible to the clinician.

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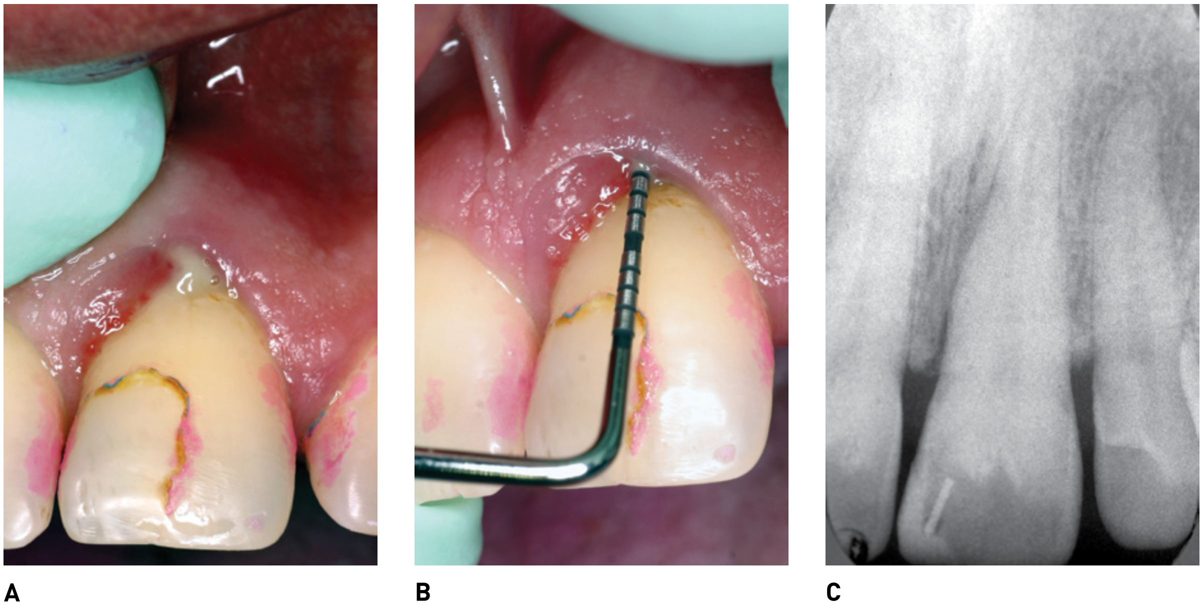

Figure 19-7 Exudate. A. Pressure with the clinician's finger on the gingiva reveals exudate from the gingival tissue adjacent to the central incisor. **B.** Exudate also is visible during probing. **C.** Radiograph of the same incisor.

Courtesy of Dr. Richard J. Foster, Educational Solutions and Design, Greensboro, NC.

2. **The Teeth**
   a. **Tooth Mobility and Fremitus**
      1. **Horizontal tooth mobility**, movement of a tooth in a facial-to-lingual direction, is assessed using the blunt ends of two dental instrument handles (like the end of a mirror) on either side of the tooth (**Figure 19-8**).

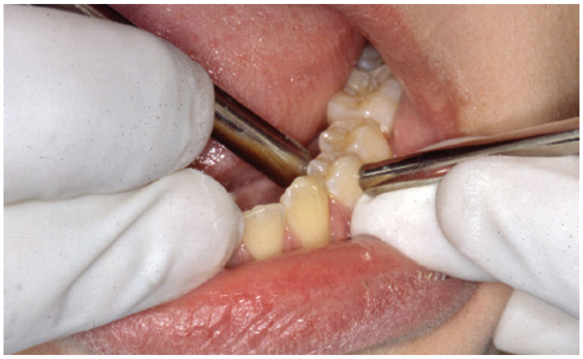

Figure 19-8 Determining Horizontal Tooth Mobility. To determine facial-to-lingual (or buccal-to-lingual mobility), the blunt ends of two instrument handles are applied to the tooth to see if it can be displaced horizontally. Light, alternating pressure is applied with the instrument handles to detect movement relative to the adjacent teeth.

Used by permission from Scheid RC, Woelfel JB. *Woelfel's Dental Anatomy Its Relevance to Dentistry*. 7th ed. Lippincott Williams & Wilkins; 2007. © Wolters Kluwer.

         a. Light, alternating pressure is applied in the facial-lingual direction against the tooth first with one, then the other instrument handle.
         b. Mobility can be observed by using an adjacent tooth as a stationary point of visual reference during attempts to move the tooth being examined.
      2. **Vertical tooth mobility**, the ability to depress the tooth in its socket, can be assessed using the blunt end of an instrument handle to exert pressure against the occlusal or incisal surface of the tooth (**Figure 19-9**).

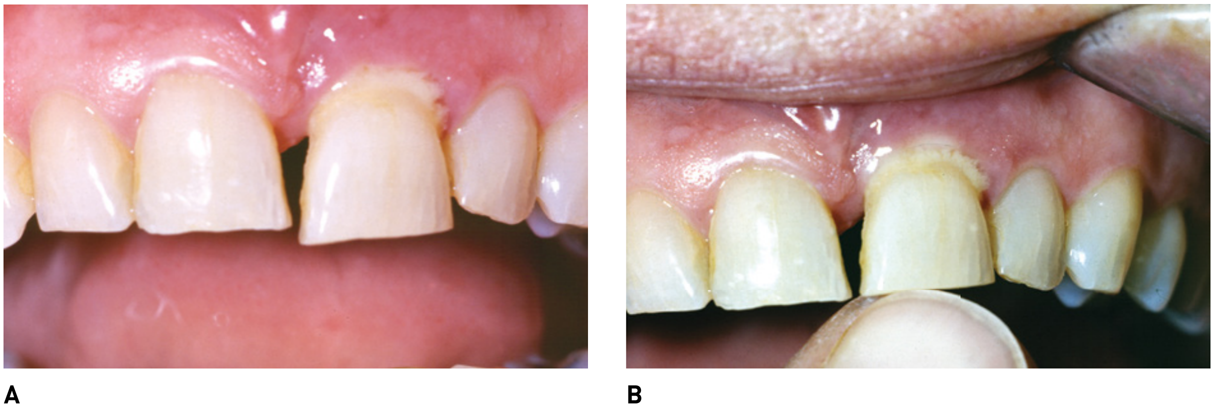

Figure 19-9 Vertical Tooth Mobility. A. The patient came to the dental office complaining of a loose tooth. Note the supraeruption of the maxillary left central incisor. **B.** The patient then demonstrated how he could push this tooth upward by applying pressure with his index finger against the incisal edge. This central incisor has vertical mobility.

Courtesy of Dr. Don Rolfs, Periodontal Foundations, Wenatchee, WA.

      3. Even though the periodontal ligament allows some slight movement of the tooth in its socket, the amount of this natural tooth movement is so slight that it cannot normally be seen with the naked eye. Thus, when visually assessing mobility, the clinician should expect to find no visible movement in a periodontally healthy tooth.

    4. Many rating scales are available for recording clinically visible tooth mobility. One useful scale is presented in **Table 19-1**.

Table 19-1 Scale for Rating Visible Tooth Mobility

Classification	Description
Class 1	Slight mobility that is visually discernible; up to 1 mm of horizontal displacement in a facial-lingual direction.
Class 2	Moderate mobility that is visually discernible; greater than 1 mm but less than 2 mm of horizontal displacement in a facial-lingual direction.
Class 3	Severe mobility that is visually discernible; greater than 2 mm of displacement in a facial-lingual direction or vertical displacement (tooth depressible in the socket).

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    5. In some dental offices, the dentist may also wish to assess fremitus.
        a. **Fremitus** is a palpable or visible movement of a tooth when in function.
        b. Fremitus can be assessed by gently placing a gloved index finger against the facial aspect of the tooth as the patient either taps the teeth together or simulates chewing movements. Fremitus is easy to detect if the finger pressure is gentle. Fremitus may be an indication of occlusal interferences.

b. **Furcation Involvement**
    1. A **furcation probe** is used to assess furcation involvement on multirooted teeth. Most molar teeth and some maxillary first premolars may present with more than 1 root, creating the potential for a furcation involvement.
    2. Furcation probes are curved, blunt-tipped instruments that allow easy access to the furcation areas; straight periodontal probes cannot be relied upon to detect furcation involvements accurately. The most common type of furcation probe is known as the Nabers probe.
    3. Furcation involvement occurs on a multirooted tooth when periodontal infection invades the area between and around the roots, resulting in a loss of attachment and loss of alveolar bone between the roots of the tooth.
        a. Mandibular molars are usually bifurcated (with mesial and distal roots), with potential furcation involvement on both the facial and lingual aspects of the tooth (**Figure 19-10**).

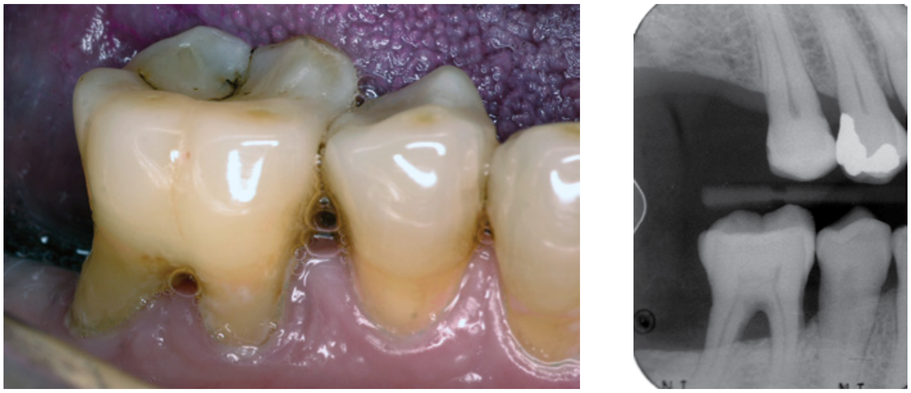

Figure 19-10 Furcation Involvement. The photograph on the left shows a furcation involvement as viewed from the facial aspect of a mandibular first molar. On the right, a radiograph of the same molar tooth shows bone loss between the roots of this molar.

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   b. Maxillary molar teeth are usually trifurcated (with mesiobuccal, distobuccal, and palatal roots) with potential furcation involvement on the facial, mesial, and distal aspects of the tooth.
   c. Maxillary first premolars that have bifurcated roots (buccal and palatal roots) have the potential for furcation involvement on the mesial and distal aspects of the tooth.
4. Furcation involvement frequently signals a need for periodontal surgery after completion of nonsurgical therapy. Thus, detection and documentation of furcation involvement is a critical component of the comprehensive periodontal assessment.
5. Furcation involvement should be recorded using a scale that quantifies the severity (or extent) of the furcation invasion. **Table 19-2** shows a commonly used scale for rating furcation invasions of multi-rooted teeth. This scale is known as the Glickman furcation classification system.

Table 19-2 **Scale for Rating Furcation Involvement**

Classification	Description
Class I	Incipient (early) furcation involvement; the tip of the Nabers probe can detect the curvature of the furcation concavity, but cannot enter inside the furcation.
Class II	A Nabers probe penetrates the furcation greater than 1 mm, but does not pass completely through the furcation. A class II furcation is like a cul-de-sac at the end of a neighborhood.
Class III	The probe passes completely through the furcation, but the furcation entrance is not clinically visible because the soft tissue margin remains coronal to the furcation. In mandibular molars, the probe passes completely through the furcation between the mesial and distal roots. In maxillary molars, the probe passes between the mesiobuccal and distobuccal roots and will touch the palatal root.
Class IV	Same as Class III furcation, except that the entrance to the furcation is clinically visible because of advanced gingival recession.

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c. **Presence of Calculus Deposits on the Teeth**
   1. The presence of dental calculus on the teeth should be noted because these deposits must later be identified and removed as part of the nonsurgical therapy.
   2. Calculus is a local contributing factor in both gingivitis and periodontitis; thus, the identification and removal of these deposits is a critical component of successful patient treatment.
   3. Calculus deposits can be located through several techniques that include the following:
      a. Direct visual examination using a mouth mirror to locate supragingival deposits.
      b. Visual examination while using compressed air to dry the teeth to aid in locating supragingival deposits.
      c. Tactile examination using an explorer to locate subgingival calculus deposits.

d. **Presence of Plaque Biofilm on the Teeth**
   1. The presence of plaque biofilm on the teeth should be noted during a comprehensive periodontal assessment because these deposits contain living periodontal pathogens that can lead to both gingivitis and periodontitis.
   2. Plaque biofilms can be identified using disclosing dyes or by moving the tip of an explorer or a periodontal probe along the tooth surface adjacent to the gingival margin.
   3. The presence of plaque biofilms can be recorded in a number of ways, but most dental offices record plaque levels in terms of the percentage of tooth surfaces with plaque biofilm evident at the gingival margin. A useful formula for recording plaque percentages is shown in **Box 19-1**.

Box 19-1 **Formula for Calculating Plaque Percentages**

$$\frac{\text{Number of tooth surfaces with plaque}}{\text{Total number of tooth surfaces}} \times 100 = \text{percentage score}$$

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   a. Note that in using the calculation shown in Box 19-1, a plaque score of 90% indicates that 90% of the total available tooth surfaces have plaque biofilm at the gingival margin.
   b. One goal of therapy would be for the patient to learn and perform biofilm control measures that would bring the plaque score as close to 0% as possible (or at least to bring the percentage of tooth surfaces with plaque biofilm as low as possible).
4. Plaque biofilm is the primary etiologic factor for both gingivitis and periodontitis. Identification of the presence and distribution of plaque biofilm on the teeth provides a critical piece of information needed when planning appropriate therapy and patient education.

3. **Local Factors and Radiographic Evidence**
   a. **Presence of Local Contributing Factors**
      1. A thorough periodontal assessment will always include identification of local contributing factors. These factors are discussed in Chapter 16.
      2. The treatment plan for any periodontal patient will always include steps to eliminate or to minimize the impact of these local factors.
   b. **Radiographic Evidence of Alveolar Bone Loss**
      1. It is important for the clinician to remember that radiographs play an important role in arriving at the periodontal diagnosis and in developing an appropriate plan for nonsurgical periodontal therapy.
      2. Radiographic evidence of alveolar bone loss is always an important part of a clinical periodontal assessment.

Components of the Comprehensive Periodontal Assessment That Require Calculations

Some judgments that are made as part of a comprehensive periodontal assessment require some calculations. The most common features that may require some calculations are the width of the keratinized gingiva, width of attached gingiva, and clinical attachment level.

1. **Width of Keratinized Gingiva and Width of Attached Gingiva**
   a. **Description.** The keratinized gingiva is composed of the free gingiva and the **attached gingiva**. The attached gingiva is the part of the gingiva that is firm, dense, and tightly connected to the cementum on the cervical-third of the root or to the periosteum (connective tissue cover) of the alveolar bone. The attached gingiva lies between the free gingiva and the alveolar mucosa, extending from the base of the sulcus (or pocket) to the mucogingival junction (**Figure 19-11**). The width of the keratinized gingiva is measured from the free gingival margin to the mucogingival junction. The width of the attached gingiva is calculated by measuring the probing depth, then subtracting this from the total width of the keratinized gingiva.

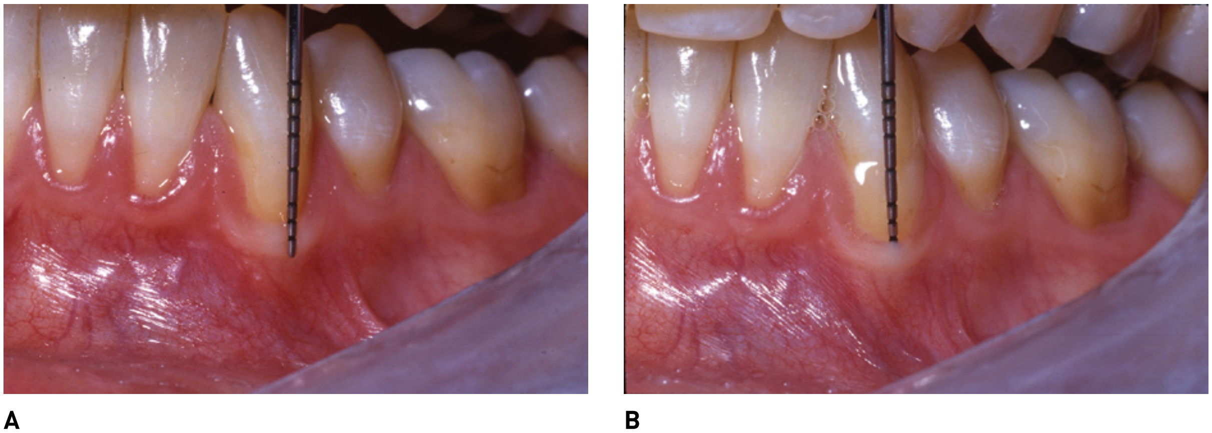

Figure 19-11 Width of Keratinized Gingiva and Width of Attached Gingiva. A. The total width of the *keratinized* gingiva is measured from the gingival margin to the mucogingival junction. In this figure, it is measured as 2 mm. **B.** The probing depth—from the gingival margin to the base of the pocket—is 1 mm. Thus, the width of *attached* gingiva is 1 mm.

Used by permission from Scheid RC, Woelfel JB. *Woelfel's Dental Anatomy Its Relevance to Dentistry*. 7th ed. Lippincott Williams & Wilkins; 2007. © Wolters Kluwer.

1. The functions of the attached gingiva are to keep the free gingiva from being pulled away from the tooth and to protect the gingiva from trauma.
2. On the palate, the width of the attached gingiva is not measured because it is not possible to determine where the attached gingiva ends and the palatal mucosa begins due to the lack of a mucogingival junction on the palate.
3. The attached gingiva does not include any portion of the gingiva that is separated from the tooth by a crevice, sulcus, or periodontal pocket (like the free gingiva).
4. It is worth reiterating that although the attached gingiva is keratinized tissue, the width of the attached gingiva is not synonymous with the width of keratinized tissue. The width of attached gingiva is one of two components that makes up keratinized gingival tissue (the other component is the free gingiva). To measure the width of keratinized tissue, the width of the attached gingiva and the width of the free gingiva are added together, or, more simply, the periodontal probe tip is placed over the soft tissue and measured from the free gingival margin to the mucogingival junction as depicted in Figure 19-11A.

b. **Significance of Inadequate Attached Gingiva and Keratinized Gingiva**

1. The width of the attached gingiva and the width of the keratinized gingiva are important clinical features for the dentist to keep in mind when planning restorative procedures. An adequate band of keratinized gingiva may be a prerequisite to maintain periodontal health and unaltered attachment levels on restored teeth. However, if a submarginal restoration is placed on a tooth with a narrow band of keratinized gingiva, the submarginal restoration may impinge on the tissues, resulting in gingival inflammation and periodontal breakdown. Further, it is conceivable that on a tooth with a narrow band of keratinized tissue the submarginal restoration could act as a plaque-retentive area that is difficult to keep clean. Therefore, it is important to use the information collected during the comprehensive periodontal assessment to calculate this clinical feature.
2. **Mucogingival defects** are deviations from the normal anatomic relationship between the gingival margin and the mucogingival junction.
   a. Common mucogingival conditions are recession and reduction or absence of keratinized tissue.
   b. With increased attachment loss, the base of a pocket may eventually extend beyond the mucogingival junction (thus, this site has no attached gingiva). When the periodontal probe extends apical to the mucogingival junction, a mucogingival defect is present.
   c. Areas with a lack of keratinized gingiva can be difficult to clean, leading to biofilm accumulation.

c. **Calculating the Width of Attached Gingiva.** The method for calculation of the width of attached gingiva is shown in **Box 19-2** (**Figure 19-12**). Note that the information needed to calculate the width of the attached gingiva already would have been recorded during the periodontal assessment.

Box 19-2 Calculating the Width of Attached Gingiva

Formula: To calculate the width of attached gingiva (WAG) at a specific site, measure the width of the keratinized gingiva (WKG) and subtract the probing depth (PD) from the total width using the steps below (WKG − PD = WAG).

Step 1: Place a periodontal probe on the outside of the tissue and measure from the gingival margin to the mucogingival junction. This measurement provides the total width of keratinized tissue.

Step 2: Measure the sulcus or pocket depth (from the gingival margin to the base of the sulcus or pocket).

Step 3: The width of the attached gingiva is calculated by subtracting the probing depth from the total width of the keratinized gingiva.

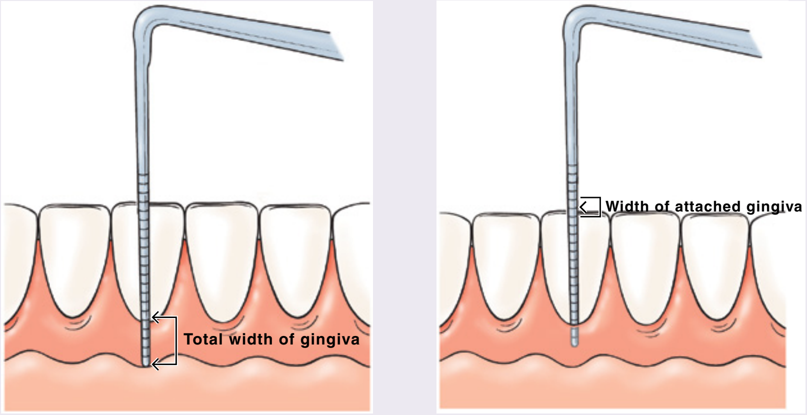

Figure 19-12 Calculating the Width of Attached Gingiva.

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2. **Clinical Attachment Level (CAL)**
   a. **Definition.** The **clinical attachment level (CAL)** is a clinical measurement of the true periodontal support around the tooth as measured with a periodontal probe. It is the gold standard by which clinicians measure periodontal health. **Box 19-3** outlines a comparison of probing depths and CALs. **Box 19-4** compares and contrasts *clinical attachment level* versus *clinical attachment loss* (which is an estimated measurement of periodontal destruction). These two concepts sound the same but are notably separate and distinct from one another.

Box 19-3 Comparison of Probing Depths and Clinical Attachment Levels

Monitoring the periodontal support of teeth over time is a vital component of long-term care of patients with periodontal disease. Two measurements are used to describe the amount of periodontal support for teeth: (1) probing depths and (2) clinical attachment levels (CALs). Probing depths are frequently used, whereas CALs are less commonly used. However, clinicians should be aware that using probing depths alone is not a reliable means to monitor the patient's periodontal status; for example, when there is gingival recession, probing depths measurements alone lead to an underestimation of true attachment levels, whereas in cases of negative recession, probing depth measurements alone lead to an overestimation of true attachment levels.

Probing depths alone are not the most reliable indicator of the amount of periodontal support for a tooth. Probing depths are measured from the gingival margin, and the position of the gingival margin often changes over time. Changes in the level of the gingival margin occur with gingival swelling, gingival overgrowth, or gingival recession. Thus, a change in probing depth over time may indicate a change in the amount of periodontal support for a tooth, but it may also only indicate that there has been some change in the level of the gingival margin that may well be unrelated to the actual periodontal support of the tooth.

CALs are the preferred and more accurate indicator of the actual amount of periodontal support for a tooth. CAL measurements are made from a fixed point that does not change. By convention, the CEJ of the tooth serves as the fixed reference point to measure the CAL. Therefore, when there is a change in the CAL over time, this change reflects an accurate measurement of a true change in the periodontal support of a tooth.

When the CEJ is not present due to a restoration or is difficult to detect, a different fixed reference point other the CEJ can be used. In such a case, the attachment level is measured by using a different fixed reference point, such as the margin of the restoration or the occlusal surface of a tooth. This is known as relative attachment level.

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Box 19-4 Clinical Attachment Level Versus Clinical Attachment Loss

Some have suggested that *clinical attachment level* and *clinical attachment loss* are synonymous terms that describe interchangeable concepts.

From a mathematical standpoint, this is true, as both clinical attachment level and clinical attachment loss are calculated using the same equation (probing depth + distance of the gingival margin to the CEJ = clinical attachment level or clinical attachment loss).

However, from a clinical standpoint, this is confusing and not completely accurate. For example, in a *periodontally healthy, noninflamed site* where the free gingival margin coincides with the CEJ (free gingival margin level = CEJ level), a given sulcus may probe 2 mm. If one were to think that the clinical attachment level is synonymous with clinical attachment loss, then the clinical attachment loss measurement at that given site would be 2 mm as well (probing depth + distance from the gingival margin to the CEJ = 2 mm + 0 mm). However, this would not make much sense because, in actuality, nothing has really been lost.

Hence, a more straightforward way to think of clinical attachment level is that it reflects the true attachment level measurement of a periodontally healthy, intact site that has not previously lost attachment, whereas clinical attachment loss reflects the remaining attachment level following irreversible periodontal destruction.

b. **Significance of Clinical Attachment Levels**
   1. An attachment level measurement is a more accurate indicator of the periodontal support around a tooth than is a probing depth measurement.
      a. Probing depths are measured from the free gingival margin to the base of the sulcus or pocket. The position of the gingival margin may change with tissue swelling, overgrowth of tissue, or recession of tissue. Because the position of the gingival margin can change (move coronally or apically), probing depths do not provide an accurate means to monitor changes in periodontal support over time in a patient.
      b. In contrast, CAL provides an accurate means to monitor changes in periodontal support over time. The CAL is calculated from measurements made from a fixed point on the tooth that does not change (i.e., the CEJ of the tooth).
   2. The presence of loss of attachment is a critical factor in distinguishing between gingivitis and periodontitis.
      a. Inflammation with no attachment loss is characteristic of gingivitis.
      b. Inflammation with attachment loss is characteristic of periodontitis.

c. **Calculating the Clinical Attachment Level**
   1. **When the gingival margin is near the CEJ.** When the gingival margin is at its natural location (i.e., slightly coronal to the CEJ of the tooth),[11] the probing depth and the CAL readings are the same for all practical purposes (**Figure 19-13A**).
   2. **When the gingival margin is significantly coronal to the CEJ (i.e., there is gingival enlargement).** When gingival enlargement is present, a straight calibrated periodontal probe is used to measure the distance that the gingival margin is coronal to the CEJ (**Figure 19-13B**). Remember that the natural position for the gingival margin is either at or slightly coronal to the CEJ, but there are many instances when the gingival margin will be found to be significantly (several millimeters) coronal to the CEJ. If the gingival margin is significantly coronal to the CEJ, the distance between the margin and the CEJ is estimated using the following technique:
      a. Position the tip of the straight calibrated probe at a 45-degree angle to the tooth surface.
      b. With light force, slowly advance the probe beneath the gingival margin until the junction between the enamel and cementum is detected.
      c. Measure the distance between the gingival margin and the CEJ, then measure the probing depth at this site.
      d. Subtract the distance from the gingival margin to the CEJ from the probing depth to determine the CAL at the site.
   3. **When the gingival margin is apical to the CEJ (i.e., the gingival margin is recessed with root exposure).** When gingival recession is present, a straight calibrated periodontal probe is used to measure the distance the gingival margin is apical to the CEJ (**Figure 19-13C**), and the same probe is used to measure the probing depth at the site. Both of these measurements will have been made and recorded as part of the comprehensive periodontal assessment. To calculate the CAL, simply add these two measurements (i.e., the amount of gingival recession plus the amount of the probing depth at the site).

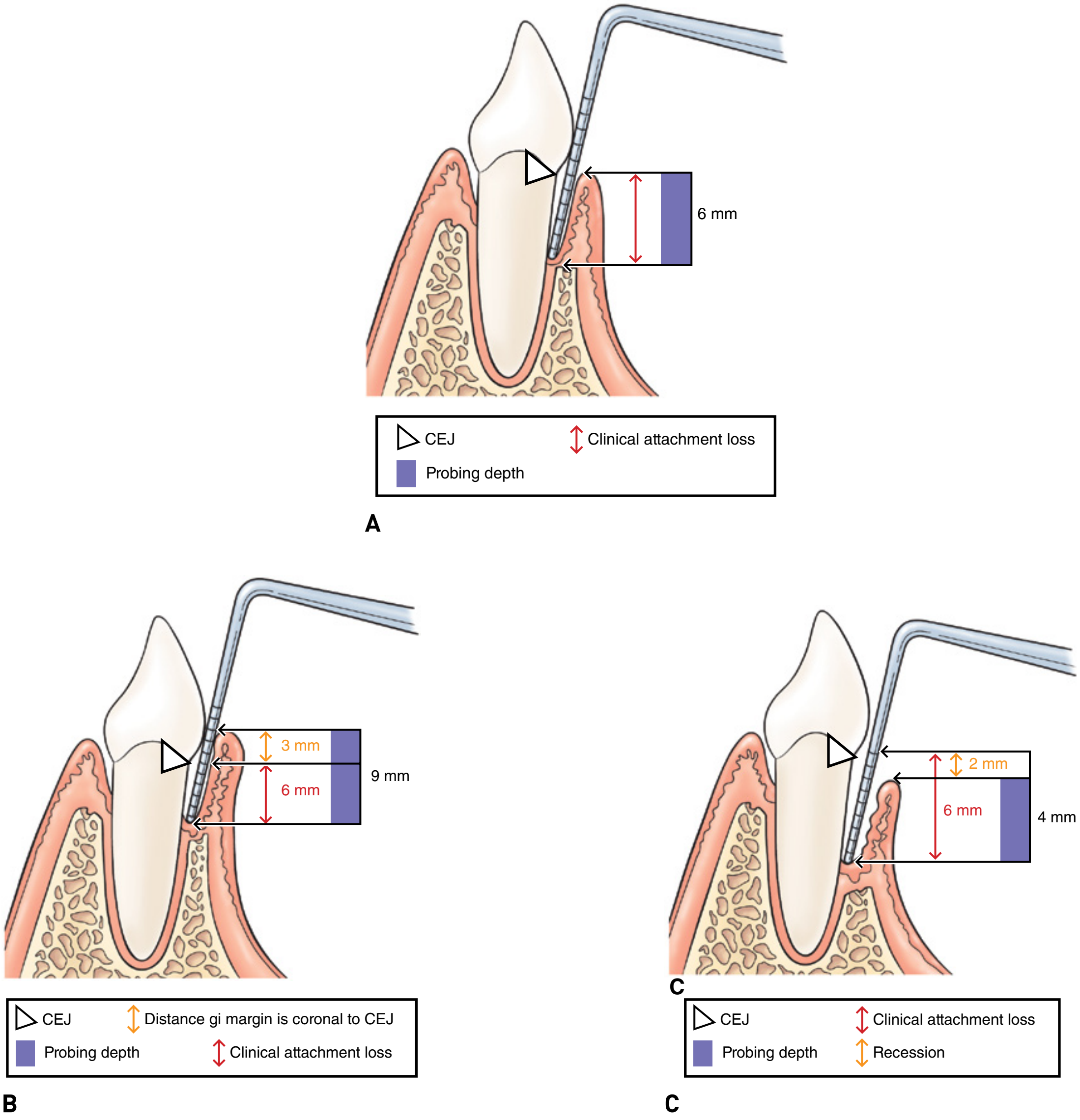
6 mm
CEJ
Clinical attachment loss
Probing depth
A
3 mm
6 mm
9 mm
CEJ
Distance gi margin is coronal to CEJ
Probing depth
Clinical attachment loss
B
2 mm
6 mm
4 mm
C
CEJ
Clinical attachment loss
Probing depth
Recession
C

Figure 19-13 Calculating Clinical Attachment Levels. A. Calculating the clinical attachment level when the gingival margin is slightly coronal to the CEJ. When the gingival margin is slightly coronal to the CEJ (as in a healthy, noninflamed site), no calculations are needed because the probing depth and the clinical attachment level are equal. This figure depicts a previous periodontitis site that is currently stable with the gingival margin coinciding with the CEJ. For example:

Probing depth measurement: 6 mm
Gingival margin level: 0 mm
Clinical attachment loss: 6 mm

B. Calculating the clinical attachment level when the gingival margin is significantly coronal to the CEJ. When the gingival margin is significantly coronal to the CEJ, the CAL is calculated by *subtracting* the gingival margin level from the probing depth. For example:

Probing depth measurement: 9 mm
Gingival margin level: −3 mm
Clinical attachment loss: 6 mm

C. Calculating the clinical attachment level in the presence of gingival recession. When recession is present, the CAL is calculated by *adding* the probing depth to the gingival margin level. For example:

Probing depth measurement: 4 mm
Gingival margin level: +2 mm
Clinical attachment loss: 6 mm

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4. **Recording the Gingival Margin on a Periodontal Chart.** Customarily, the notations 0, −, or + are used to indicate the position of the gingival margin with respect to the CEJ on a periodontal chart (**Box 19-5**).

Box 19-5 Notations That Indicate the Position of the Free Gingival Margin

- A zero (0) indicates the free gingival margin is slightly coronal to the CEJ. In health, the free gingival margin should be at this level.
- A negative number (−) indicates that the free gingival margin significantly covers the CEJ. This is also known as negative recession.
- A positive number (+) indicates that the free gingival margin is apical to the CEJ. This is also known as positive recession.

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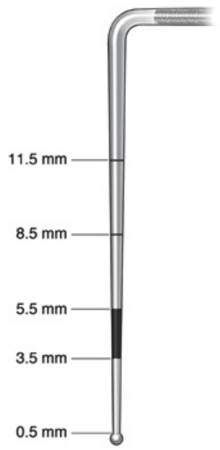
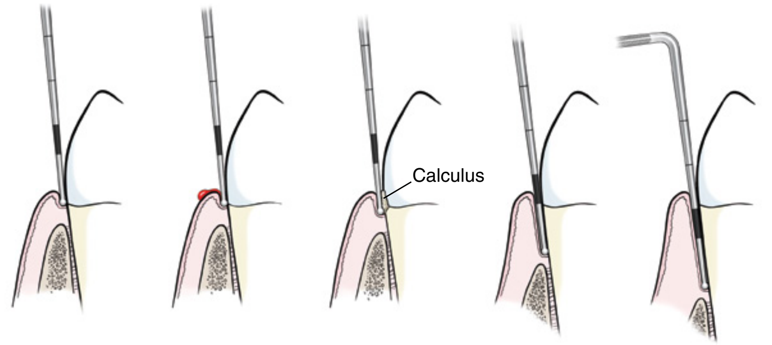
Section 3

Periodontal Screening Examination

In some dental offices, a periodontal screening examination is used as one of the first steps in evaluating the periodontal status of a patient. A **periodontal screening examination** is a rapid, inexpensive screening process that may be used to determine if a more comprehensive periodontal assessment is necessary. The **Periodontal Screening and Recording (PSR)** examination is one example of an easy-to-use screening system that can aid in the detection of periodontal disease.

1. **The PSR Examination**
    a. **Characteristics of the PSR Examination**
        1. The PSR can help separate patients into three broad categories: those that seem to have (1) periodontal health, (2) gingivitis, or (3) periodontitis.
        2. When the PSR screening examination indicates the presence of periodontal health or gingivitis, except in a very few instances, no further clinical periodontal assessment may be needed beyond the PSR. It is important to note that individual states have different rules related to the use of this type of screening examination.
    b. **Techniques for Performing the PSR Screening Examination**
        1. **Special Probe.** A **World Health Organization (WHO) probe** is used for this examination. The WHO probe is easily identifiable by a 0.5 mm diameter ball at the tip, wide black band (called the reference mark) located 3.5 to 5.5 mm from the ball tip, and markings further up the probe which represent 8.5 mm and 11.5 mm from the ball tip (**Figure 19-14**). The reference mark is used when performing the PSR screening examination. The purpose of the ball tip is to provide the clinician with greater tactile feel when probing the sulcus and identifying sites with subgingival calculus. Additionally, the ball tip may cause less damage to the periodontal tissue compared to a straight, thin, slender pointed probe tip, like a UNC-15 probe or a Williams probe. Another name for the WHO probe is the CPITN (Community Periodontal Index of Treatment Needs) probe.





PSR and CPI sextant scores	Code 0	Code 1	Code 2	Code 3	Code 4
CPI description	• Entire black band of the probe is visible	• Entire black band of the probe is visible, but bleeding is present after gentle probing	• Entire black band is visible, but calculus is present • Bleeding may or may not be present.	• 4 to 5 mm pocket depth • Black band on probe partially hidden by gingival margin	• 6 mm or greater pocket depth • Black band of probe completely hidden by gingival margin
PSR sextant code description	• Colored area of probe completely visible • No calculus, defective restoration margins or bleeding	• Colored area of probe completely visible • No calculus or defective restoration margins • Bleeding after gentle probing	• Colored area of probe completely visible • Supra or subgingival rough surface or calculus • Defective restoration margins	• Colored area of probe only partially visible • Calculus, defective restorations, and bleeding may or may not be present	• Colored area of probe completely disappears (probing depth of 5.5 mm or greater)
PSR management guidelines	• Biofilm control instruction • Preventive care	• Biofilm control instruction • Preventive care	• Biofilm control instruction • Complete preventive care • Calculus removal • Correction of defective restoration margins	• Comprehensive periodontal assessment and treatment plan is indicated	• Comprehensive periodontal assessment and treatment plan is indicated

Figure 19-14 WHO Probe and Criteria for Assigning PSR Codes.

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a. Instead of reading and recording six precise measurements per tooth, the clinician only needs to observe the position of the reference mark in relation to the gingival margin and a few other clinical features such as the presence of bleeding on probing, the presence of calculus, or the presence of an overhang on a restoration.
b. Each of the sextants is examined as a separate unit during the PSR screening (i.e., only one PSR code number will be assigned to the entire sextant).
c. Only one PSR code is recorded for each sextant in the mouth. Each sextant is assigned a single PSR code; the highest code obtained for the sextant is recorded.
d. An "X" is recorded instead of a PSR code if the sextant is edentulous.

2. **One Code Per Sextant.** Using the PSR, the mouth is divided into the six sextants (segments) listed below. Each sextant of the mouth is examined and assigned an individual PSR code. The unique aspects of the PSR screening system are the manner in which the probe is read and the minimal amount of information that needs to be recorded.
    a. Maxillary right posterior sextant: terminal maxillary right molar to maxillary right first premolar.
    b. Maxillary anterior sextant: maxillary right canine to maxillary left canine.
    c. Maxillary left posterior sextant: maxillary left first premolar to terminal maxillary left molar.
    d. Mandibular left posterior sextant: terminal mandibular left molar to mandibular left first premolar.
    e. Mandibular anterior sextant: mandibular left canine to mandibular right canine.
    f. Mandibular right posterior sextant: mandibular right first premolar to terminal mandibular right molar.
3. **Probing Technique**
    a. The probe is "walked" circumferentially around each tooth in the sextant being examined. Walking a periodontal probe refers to moving the probe in small increments circumferentially around a tooth.
    b. The reference mark is monitored continuously as the probe is walked around each tooth. At each site probed, the reference mark will be (a) completely visible, (b) partially visible, or (c) not visible at all.

2. **The PSR Codes**
    a. **Use of PSR Codes**
        1. A PSR code is assigned to each sextant according to the criteria shown in Figure 19-14. The code assigned to a sextant should represent the most advanced periodontal finding on any tooth in that sextant.
        2. The PSR codes are used to guide further clinical documentation.
            a. For some patients with low PSR codes in all sextants (codes 0, 1, or 2), the PSR screening may be adequate documentation of the patient's periodontal health status. Note, however, that the dentist may request a comprehensive periodontal assessment even when low PSR codes are found, because many periodontal conditions must be monitored in more detail than that included in the PSR. In other words, the benefits of the PSR system do not supplant the wealth of additional information garnered from a thorough comprehensive periodontal assessment.
            b. For patients with higher PSR codes in one or more sextants (codes 3 or 4), a comprehensive periodontal examination should be performed as outlined previously in Section 2 of this chapter.
    b. **Cautions for Interpreting PSR Codes.** The PSR codes can be misleading.
        1. As already pointed out, lower codes usually mean periodontal health or gingivitis, and higher codes usually mean periodontitis. When interpreting the results of the PSR, the clinician must be alert for teeth with gingival enlargement or gingival recession. In the presence of either of these conditions, the PSR can give misleading results.
        2. Another shortcoming is that the partial recording approach of the PSR index may grossly underestimate the severity of periodontal destruction because it only relies on probing depth measurements but fails to quantify the amount of clinical attachment loss.

Section 4

Supplemental Diagnostic Tests

1. **Overview of Supplemental Diagnostic Tests**
    a. Clinical periodontal assessment using the parameters discussed in Section 2 will result in an accurate periodontal diagnosis and can serve as a sound basis for designing an appropriate plan for therapy for the patient with periodontal disease. However, a number of supplemental diagnostic tests are available that can be used for certain patients.
    b. Clinicians might consider using some of these supplemental tests for patients who have refractory forms of periodontitis.
    c. A number of supplemental tests have been suggested for use, and much research is continuing related to these types of tests. Most of these tests fall into three general types:
        1. Tests related to bacteria.
        2. Tests that analyze gingival crevicular fluid content.
        3. Tests for genetic susceptibility to periodontal disease.

d. It is critical for the clinician to realize that, based on current research, currently no supplemental diagnostic test stands out as the gold standard. Thus, supplemental diagnostic tests should not be routinely ordered for all patients with periodontal disease unless the disease is refractory or the disease is considered to be too complex to diagnose with conventional methods alone.

2. **Tests Related to Bacteria**

a. **Table 19-3** presents an overview of the tests related to bacteria. It is important to keep in mind that conventional periodontal therapy brings periodontal pathogens to low enough levels that disease progression can be halted without the need for identifying specific periodontal pathogens in most patients.

Table 19-3 Tests Related to Bacteria

Test Name	Purpose of Test	Special Considerations
Phase contrast microscopic study of plaque sample	Used for patient education and motivation. Can discriminate motile from nonmotile bacterial species.	Test cannot discriminate individual bacterial species (e.g., *Treponema* spp. vs. *Aggregatibacter* spp.).
Culture and sensitivity	Used to determine the sensitivity of bacteria to specific antibiotics.	Sampling techniques for this test and the transport of bacterial samples to the laboratory are difficult.
Deoxyribonucleic acid (DNA) probe analysis	Used to identify specific periodontal pathogens in a patient's mouth.	Only a few bacterial species can be identified by this test.
Polymerase chain reaction	Very sensitive testing assay in identifying specific microorganisms. Provides rapid, "real-time" information of the specific microorganisms from plaque samples.	Requires expensive instrumentation.

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b. **Patient Education Tool: Phase Contrast Microscope**

1. The advantages of the phase contrast microscope are that it enables patients to view live microorganisms and to actually see the motion of the motile microorganisms (**Figure 19-15**).

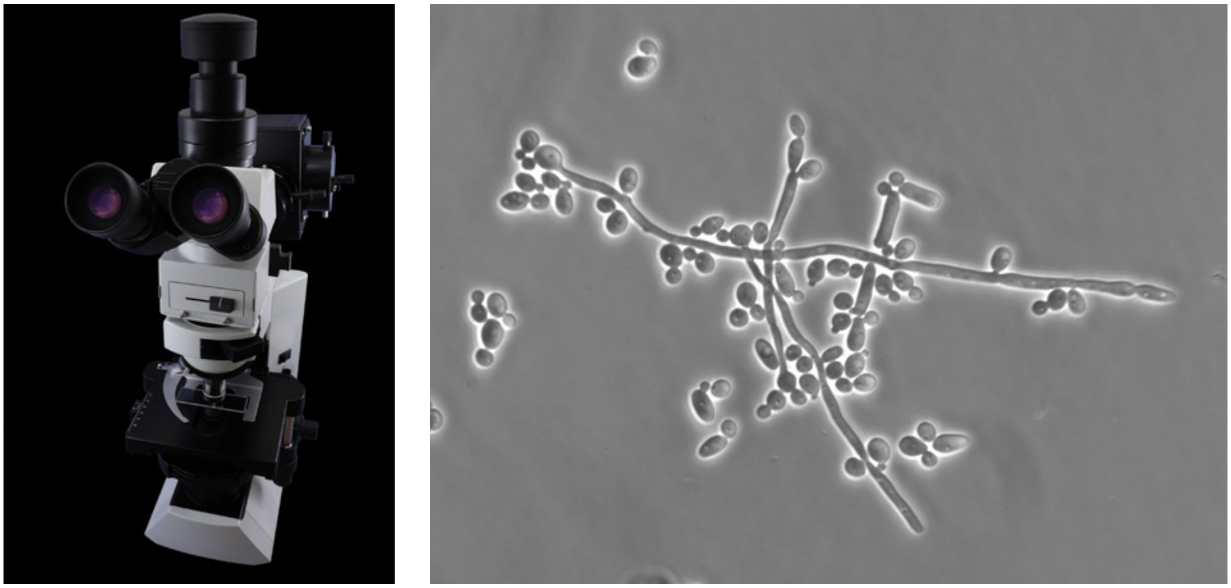

Figure 19-15 Microscope and Microorganisms. On the left, an example of a phase contrast microscope. The photograph on the right shows the opportunistic fungus *Candida albicans*. Oral candidiasis is a fungal infection caused by the overgrowth of *C. albicans*, which causes creamy white lesions on the tongue and lining of mucous membranes of the oral cavity.

Left: © Satirus/Shutterstock; **Right:** © Andre Nantel/Shutterstock

2. Effective communication is an essential skill for the dental hygienist. Viewing live microorganisms is an effective tool to educate patients by allowing them to view the live microorganisms that are contributing to their periodontal disease.
3. Phase contrast microscopy is an effective means of microscopically monitoring motile (movable) bacteria versus nonmotile microorganisms. However, the utility of phase contrast microscope is limited because it cannot be used to discriminate a specific microorganism from another. Thus, to date, although phase contrast microscopy has a positive effect in patient education, the diagnostic capabilities of the phase contrast microscope are limited, and it is not recommended for routine use in clinical settings.

3. **Tests That Analyze Gingival Crevicular Fluid Content**
    a. **Gingival Crevicular Fluid**
        1. **Gingival crevicular fluid** is the fluid that flows into the sulcus from the adjacent gingival connective tissue; the flow is slight in health and increases in the presence of inflammatory disease. The advantage of analyzing gingival crevicular fluid is that it provides site-specific information about the inflammatory status of a specific periodontal site.
        2. Gingival crevicular fluid originates in connective tissue and flows into periodontal pockets. It has long been believed that this gingival crevicular fluid can contain markers for periodontal disease progression, and quite a bit of research time has been devoted to the study of this fluid.
        3. In a clinical setting, obtaining gingival crevicular fluid from all sites in an individual's mouth may be prohibitive (too time-consuming and too costly).
    b. **Examples of Gingival Crevicular Contents That Have Been Studied**
        1. Collagenase (an enzyme that breaks down collagen) is an example of one of the gingival crevicular fluid contents that has been studied, though no test for this is currently in widespread use.
        2. Prostaglandin E2 is another such gingival crevicular fluid constituent that has been studied. Prostaglandin E2 is a cyclooxygenase product that is a major mediator that contributes to inflammatory reactions such as those seen in periodontal disease.
    c. **Tests for Genetic Susceptibility to Periodontal Disease**
        1. **Genetic Susceptibility**
            a. It is obvious that a patient's genetic makeup affects susceptibility to many diseases including periodontal disease.
            b. This genetic makeup is inherited and cannot normally be altered.
        2. **Tests for Interleukin-1**
            a. One test for genetic susceptibility to periodontal disease has been studied extensively and has resulted in a test that has been marketed to clinicians. The first version of this test was the PST Genetic Susceptibility Test from Interleukin Genetics Incorporated, Waltham, MA. In 2013, the company released a new version of this genetic susceptibility test, called Interleukin's PerioPredict Genetic Risk Test.
            b. Both of these tests identify patients who are genetically predisposed to produce high levels of interleukin-1, which is an inflammatory mediator produced in response to the presence of periodontal pathogens.
                1. Higher levels of interleukin-1 in patients tend to predispose the patients to more inflammation in the periodontium and has been associated with increased risk for severe and progressive periodontal disease.[12]
                2. It has been reported that 30% of the people in the United States may be genetically predisposed to producing high levels of interleukin-1 in response to periodontal pathogens.

4. **The Future.** An ideal diagnostic test would reliably predict which individuals are susceptible for future periodontal breakdown. It would also have the capability to quickly and accurately monitor the current status of disease activity. As technology improves and our understanding of periodontal disease broadens, we will undoubtedly have more practical, more accurate, and more reliable diagnostic tests available to utilize as part of our routine periodontal assessment.

Chapter Summary Statement

The information gathered by the members of the dental team during the clinical periodontal assessment forms the basis for an individualized treatment plan for the patient. This chapter discusses two types of clinical periodontal assessment: a periodontal screening examination and the comprehensive periodontal assessment. Periodontal Screening and Recording (PSR) is an example of a quick periodontal screening system for the detection of periodontal disease.

The comprehensive periodontal assessment is a complete, more thorough clinical periodontal assessment used to gather information about the periodontium. The information collected in a comprehensive periodontal assessment includes probing depth measurements, bleeding on probing, presence of exudate, level of the free gingival margin, level of the mucogingival junction, tooth mobility, furcation involvement, presence of calculus, presence of plaque biofilm, gingival inflammation, radiographic evidence of alveolar bone loss, and presence of local contributing factors. In addition, as part of the comprehensive periodontal assessment, several measurements—namely, the width of the keratinized gingiva, the width of the attached gingiva, and the clinical attachment levels—must also be calculated. Measuring clinical attachment level is the gold standard in determining whether gingivitis or periodontitis is present at a site of inflammation.

Supplemental diagnostic tests are indicated for certain patients and may provide critical information that will aid in formulating a diagnosis. However, at this time, there is no established, standardized protocol to justify the routine use of supplemental diagnostic test for all individuals.

To summarize, performing a careful and thorough periodontal assessment is necessary to evaluate the health status of the periodontium, identify the type of periodontal disease, and monitor disease activity. All members of the dental team must be capable of performing a meticulous periodontal assessment. Furthermore, as new supplemental diagnostic assessments emerge, clinicians must be able to think critically about the practicality of these emerging tests, understand the strength and limitations of each test, and analyze the outcomes of these tests based on sound scientific evidence.

Section 5

Focus on Patients

Clinical Patient Care

CASE 1

While visiting a dental office, you observe a member of the dental team performing a periodontal assessment. You note that while searching for furcation invasion, the clinician is using a straight calibrated periodontal probe. What critical information might be lost because of instrument selection for this step in a periodontal assessment?

CASE 2

During a comprehensive periodontal assessment, you note severe inflammation of the gingiva over the facial surface of a lower right molar tooth. On the dental chart you are using, there is no obvious mechanism to record this important piece of periodontal information. How should you proceed?

CASE 3

During a periodontal assessment of a periodontitis patient, you are trying to determine the clinical attachment level on the facial surface of a canine tooth. On the facial surface of the canine tooth, you have measured 3 mm of gingival recession and a probing depth of 6 mm. How much attachment has been lost on the facial surface of this canine tooth?

Ethical Dilemma

Your next patient is Marlene Perkins, who is a new patient to your practice. She is a 37-year-old married mother of two small daughters, who works part-time as a lawyer. Her chief complaint is "bleeding gums." She left her last dental practice because she questioned if she was receiving quality dental services.

The radiographs you have taken today show that there is very slight horizontal bone loss on her anterior teeth. Marlene's pocket readings range from 1 to 4 mm throughout her mouth, and there is generalized bleeding on probing. She presents with heavy plaque biofilm and moderate supragingival and subgingival calculus, although states that she had her teeth cleaned 4 months ago. She states that she "isn't a very good flosser, and was always reprimanded by the hygienist at her former dental practice, to do a better job."

She notes that she has a history of periodontal disease in her family, as both her mother and father have had "extensive gum surgeries." She asked the dentist and hygienist in her last practice if periodontal disease was hereditary and if there was a way to predict if she also would have periodontal disease. She was told that each person is an individual, and that if she "took care of her teeth and gums," she would be just fine.

1. What is the best way for you to handle this ethical dilemma?
2. What is the best way to address/discuss Marlene's treatment plan?
3. What ethical principles are in conflict in this dilemma?

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CHAPTER 20

Radiographic Analysis of the Periodontium

- Section 1 Radiographic Appearance of the Periodontium
  - Radiolucent Versus Radiopaque Structures
  - Radiographic Features of Parts of the Periodontium
- Section 2 Use of Radiographic Images During Periodontal Evaluation
  - Basic Techniques for Obtaining Diagnostic Radiographs
  - Limitations of Dental Radiographs During Periodontal Evaluation
  - Benefits of Dental Radiographs During Periodontal Evaluation
  - Beyond Conventional Dental Radiography
- Section 3 Focus on Patients
  - Clinical Patient Care
  - Ethical Dilemma

CLINICAL APPLICATION

Interpretation of dental radiographic images is an integral part of the diagnosis and treatment planning process. All members of the dental team should be competent and confident in interpreting radiographic images on a routine basis. This chapter outlines basic information related to the interpretation of dental radiographic images that members of the dental team can use as a springboard for further study of this topic.

LEARNING OBJECTIVES

- Describe the dental radiographic features of the healthy periodontium.
- Describe early dental radiographic evidence of periodontal disease.
- Name some techniques that can be employed with periodontal patients to obtain high-quality dental radiographs.
- Explain the basic principles of the vertical bitewing technique.
- Describe the limitations of dental radiographs that all clinicians should keep in mind when viewing radiographs.
- Explain the difference between vertical and horizontal alveolar bone loss as seen on dental radiographs.
- Given a selection of sample dental radiographs, apply the information from this chapter when analyzing those radiographs.

KEY TERMS

Radiolucent
Radiopaque
Cortical bone
Lamina dura
Crestal irregularities
Triangulation
Horizontal bone loss
Vertical bone loss
Cone beam computed tomography (CBCT)

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Section 1

Radiographic Appearance of the Periodontium

No comprehensive periodontal evaluation is complete without supplementing the information obtained from the clinical evaluation with information obtained from high-quality dental radiographs.[1,2] All dental clinicians must be able to recognize certain radiographic signs associated with periodontal diseases. The level of the alveolar bone is usually considered among the most important of the features revealed by dental radiographs, but there are many other, sometimes subtle, features that can be equally important for the clinician to note when viewing dental radiographs. Not all structures of the periodontium are even visible on dental radiographic images; some periodontal structures, such as the gingiva, cannot be viewed on a dental radiograph. Nevertheless, radiographs provide crucial information that is needed during the routine data-gathering process, such as the level of the interdental alveolar bone crests; the size and shape of the tooth roots; the relationship between adjacent tooth roots; the condition of the alveolar bone in furcation areas; and the presence of certain local contributing factors, such as overhanging margins or open margins.

1. **Radiolucent Versus Radiopaque Structures.** Structures (or materials) that can be viewed on a dental radiograph are classified as either **radiolucent** or **radiopaque** depending upon the degree of x-ray beam penetration through these structures.
   a. Radiolucent structures, such as the gingiva and periodontal ligament space, are less dense and not as mineralized compared to radiopaque structures. As such, radiolucent structures permit the x-ray beam to readily pass.
      1. Most x-ray beams will be able to pass through these structures to expose the radiographic film. Thus, on the completed radiograph, radiolucent structures have a dark gray to black appearance.
      2. Examples of radiolucent structures that can be seen on dental radiographs are the tooth pulp, the periodontal ligament space, bone loss due to a periapical abscess, marrow spaces in the bone, and bony defects due to periodontitis.
   b. In contrast, radiopaque structures are more dense and more mineralized than radiolucent structures. As such, radiopaque structures either absorb or resist the passage of x-rays.
      1. Most x-ray beams will not be able to pass through these structures to expose the radiographic film. Thus, radiopaque areas appear light gray to white on the radiograph.
      2. Examples of radiopaque structures are tooth enamel, dentin, metallic tooth restorations (such as amalgam restorations or gold restorations), some composite restorations, pulp stones, enamel pearls, and alveolar bone supporting the tooth roots.
   c. Note that many periodontal and tooth-associated structures allow only limited (but not total) penetration of the x-rays. These structures will appear as various subtle shades of gray on the completed radiograph. At times, these subtle differences can be confusing to clinicians.
2. **Radiographic Features of Parts of the Periodontium.** One of the components of the periodontium that can be identified on radiographic images includes the alveolar bone itself. Thus, it is important for clinicians to be very familiar with the radiographic appearance of this tooth-supporting bone (**Figure 20-1**).
   a. **Cortical Bone**
      1. **Cortical bone** is the outer surface of bone and is composed of layers of bone closely packed together.
         a. In the maxilla, the cortical bone is usually a thin shell covering the outer surface of the maxilla.
         b. In the mandible, the cortical bone is usually a denser and thicker layer than the maxilla.
      2. **Radiographic Appearance of Cortical and Cancellous Bone**
         a. Both cortical bone and cancellous bone can be identified on dental radiographs.
            1. Some examples of cortical bone found in the oral cavity include, but are not limited to, the inferior border of the mandible, external oblique ridge, and intraoral exostosis. Radiographically, cortical bone appears much more radiopaque with a thicker white border than the surrounding cancellous bone. **Figure 20-2** is a panoramic radiograph—the white arrow points to the inferior border of the mandible; the black arrow points to the external oblique ridge.

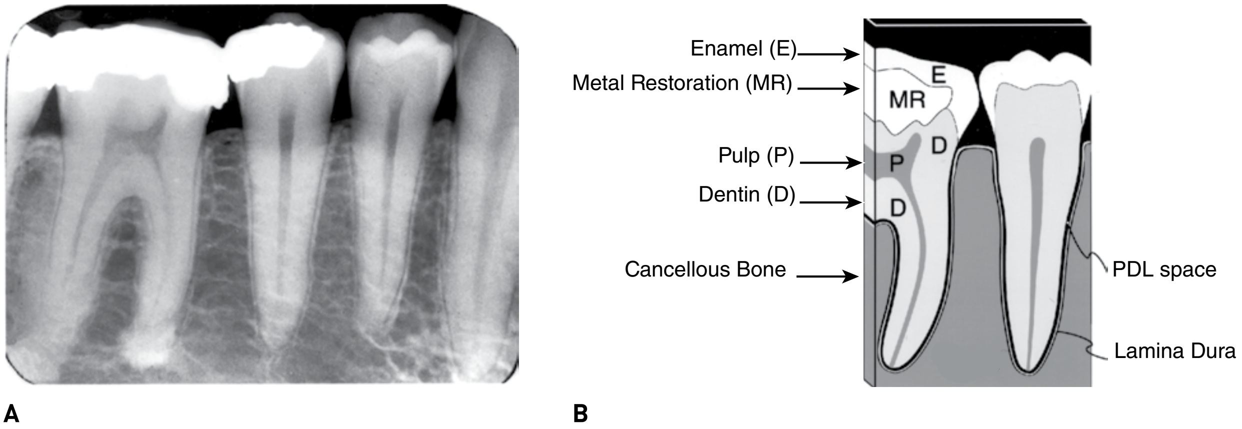

Figure 20-1 **Radiographic Structures of the Periodontium.** The dental radiograph (**A**) and drawing (**B**) provide points of reference for many of the features discussed in this chapter. Radiographs are an essential component of the dental examination because they provide the clinician an opportunity to detect any abnormalities that may not be apparent to the naked eye solely when conducting a clinical exam. For instance, on this radiograph, note the presence of an iatrogenic restoration (the overhanging restoration margin) on the first molar which may not be apparent to the naked eye during a routine clinical exam. Also, radiographs may be helpful in identifying variations of the normal anatomy. From this radiograph, note the ovoid, dense radiopacities at the apices of the first molar which are variations of the normal bony anatomy. These radiopacities may be indicative of condensing osteitis, sclerosing osteomyelitis, hypercementosis, idiopathic osteosclerosis, or cementoblastoma.

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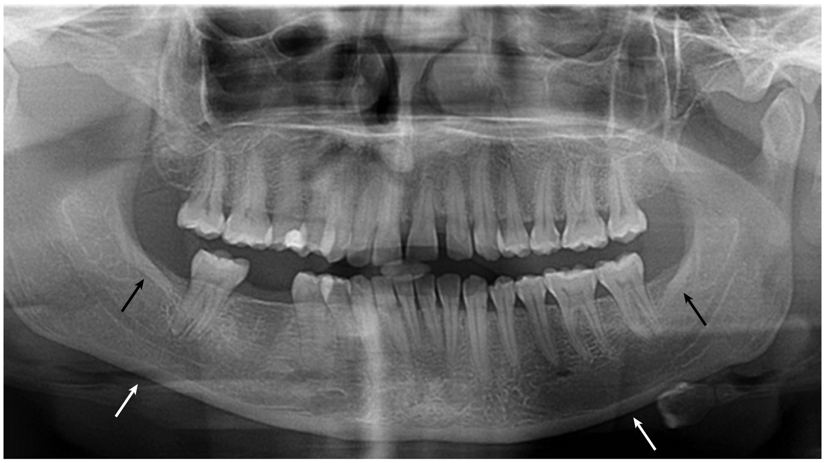
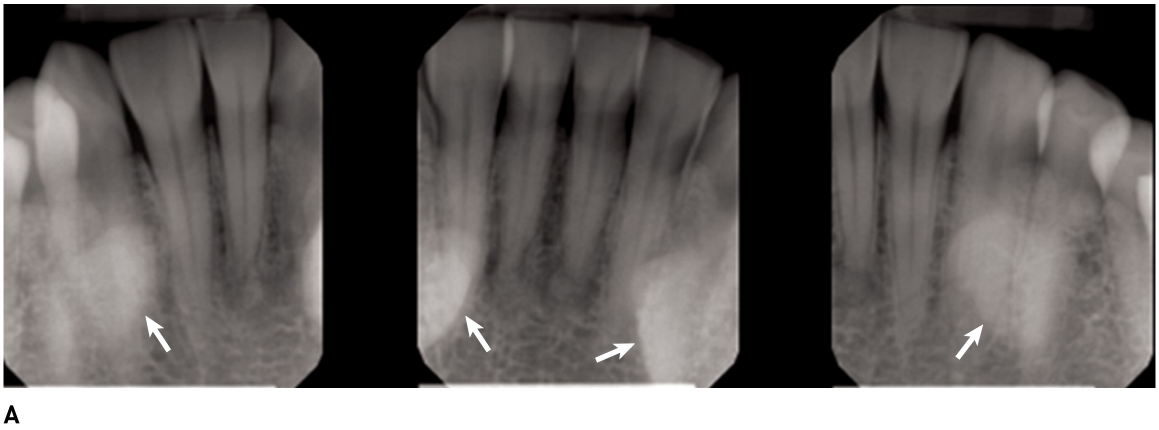


Figure 20-2 **Radiograph of Cortical Bone.** Radiographic appearance of the inferior border of the ramus (white arrows) and the external oblique ridge (black arrows).

Figure 20-3 is a different case showing the radiographic appearance of a bilateral mandibular lingual exostoses along with an accompanying clinical photograph. Note that in the above figures, the cortical bone appears much denser and thicker compared to the surrounding cancellous bone.

2. Another notable cortical bony structure is the interdental alveolar crest. In health, the interdental alveolar crest appears on the dental radiographs as a thin radiopaque (white) line that runs along the outer surface of the crestal bone. Radiographically, this is known as the crestal lamina dura.
3. In contrast, cancellous bone appears less radiopaque than cortical bone and has a lattice-like pattern that fills the interior portion of the alveolar process. The radiographic pattern of the cancellous bone can be thought of as delicate white interlaced tracings within the bone.



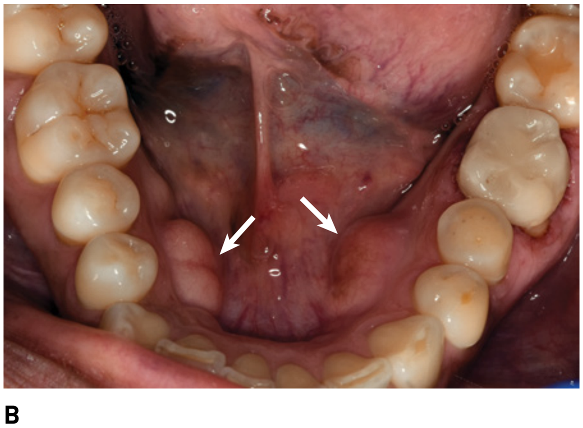


Figure 20-3 **Radiograph of Cancellous Bone. A.** Radiographic appearance of the bilateral mandibular lingual exostoses (white arrows). **B.** Accompanying clinical photograph of bilateral exostoses of the same case.

b. **Level of the Alveolar Crest in Health**
   1. The normal level of the alveolar bone crest (as it appears on a radiograph) is located approximately 2 mm apical to (or below) the cementoenamel junction (CEJ).
   2. The level of the alveolar bone crest in relationship to the CEJ is one indicator of periodontal health that should always be noted when analyzing a dental radiograph.

c. **Contour of the Alveolar Crest in Health**
   1. The contour of the interproximal alveolar crest in health is parallel to an imaginary line drawn between the CEJs of adjacent teeth. The contour of the crest of the interproximal bone is another indicator of periodontal health that should always be noted when analyzing a dental radiograph.
   2. In posterior sextants, the normal contour of the interproximal crest can vary depending upon the actual levels of the CEJs of the adjacent teeth.
      a. **Horizontal Crest Contour.** The crest of the interproximal bone will have a horizontal contour when the CEJs of the adjacent teeth are at the same level. **Figure 20-4** shows an example of posterior teeth with flat horizontal crest contours with flat horizontal crest contours between tooth #29 and tooth #30.
      b. **Vertical (or Angular) Crest Contour**
         1. The crest of the interproximal bone will have a vertical or angular contour when one of the adjacent teeth is tilted or erupted to a different height. **Figure 20-5** shows an example of posterior teeth with an angular contour when one of the adjacent teeth is tilted or erupted to a different height.
         2. When interpreting radiographs, it should be kept in mind that normal vertical crestal contour is not the same as a vertical (angular) bony defect (to be discussed later in this chapter).

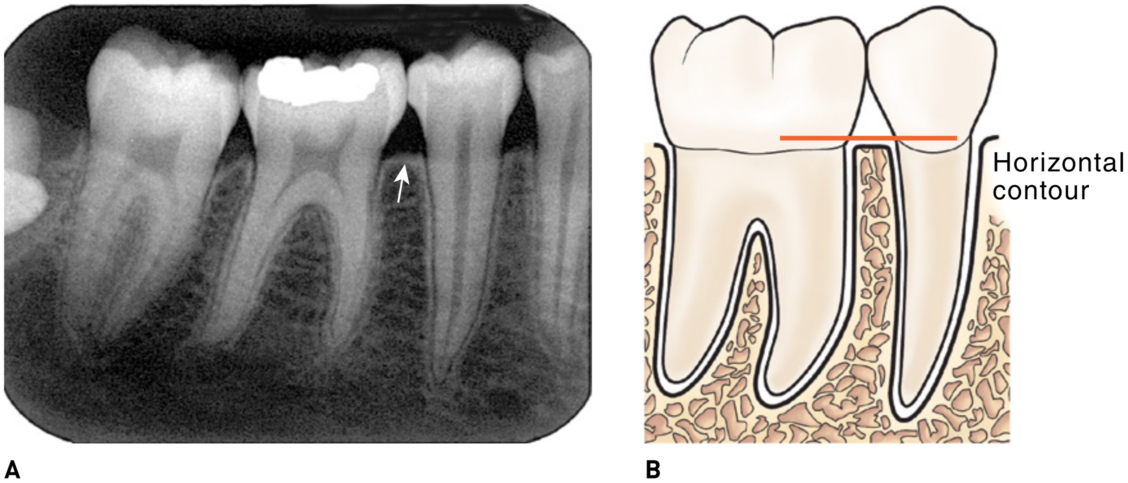


Figure 20-4 **A. Normal Alveolar Bone Height.** This radiograph shows a normal alveolar bone height that is 1.5 to 2 mm below and parallel to the CEJ. In this example, the alveolar crest appears as a dense radiopaque line as it would normally appear in health (white arrow). **B. Horizontal Crest Contour.** The crest of the interproximal bone will have a horizontal contour when the CEJs of the adjacent teeth are at the same level. Note that the crest parallels an imaginary line drawn between adjacent CEJs.

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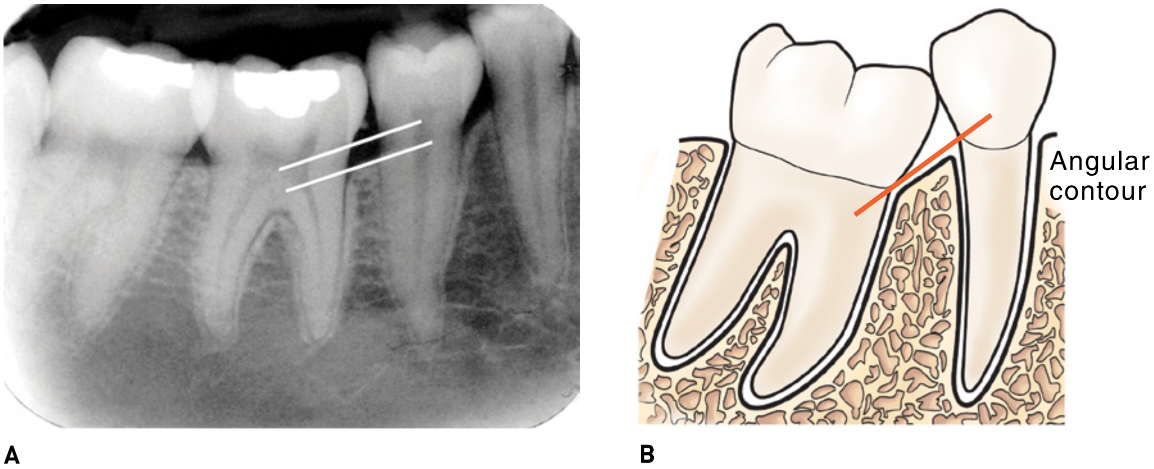

Figure 20-5 **Vertical (or Angular) Crest Contour. A.** The crest of the interproximal bone will have a vertical or angular contour when one of the adjacent teeth is tilted or erupted to a different height. **B.** Note that this crest also parallels an imaginary line drawn between adjacent CEJs, though the adjacent CEJs are at different levels. Also note that the interproximal bone level is still within 2 mm of the CEJ level of the approximating teeth.

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3. In an area with normal vertical crestal contour, the interproximal bone level remains within 2 mm from the level of the CEJ of the approximating teeth. Normal vertical crestal contour should be considered as a variation of the normal. A vertical (angular) bony defect, on the other hand, should be considered suspicious and may be an indicator of pathology.

d. **Shape and Character of the Alveolar Crestal Bone Crest in Health**
   1. The surfaces of the interdental bone crests are smooth and covered by a thin layer of cortical bone that radiographically appears as a fine, white line, known as the lamina dura (to be discussed in the next section).

2. One of the most important radiographic features of the alveolar crest is that it forms a smooth intact surface between adjacent teeth with only the width of the periodontal ligament space separating it from the adjacent root surface.
   a. The crest of the interdental bone between incisor teeth is usually thin and somewhat pointed in appearance because of the relatively close proximity of the adjacent roots of anterior teeth.
   b. The crest of the interdental bone between the posterior teeth is usually flat or slightly rounded because of the relatively greater distance between adjacent tooth roots (**Figure 20-6**).

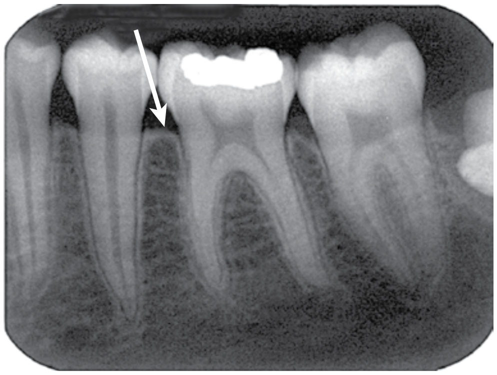

Figure 20-6 Alveolar Crest. The alveolar crest (white arrow) forms a smooth intact surface between adjacent teeth and displays a relatively flat contour. In health, the interdental alveolar crest is lined by a fine radiopaque (white) line that lines the socket walls, known as the lamina dura. On the tooth side of the lamina dura, a thin radiolucent shadow runs down the entire length of the root. This represents the periodontal ligament space. On the bone side of the lamina dura lies the cancellous bone of the alveolar process.

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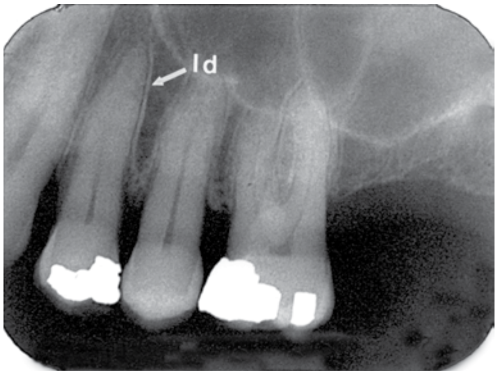

Figure 20-7 Lamina Dura and Periodontal Ligament Space. The lamina dura (ld) appears as a continuous white line around the tooth root. The periodontal ligament space appears as a thin black line that runs along the entire root. The periodontal ligament fibers reside within the periodontal ligament space.

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3. The shape and orientation of the alveolar bone crest are other indicators of periodontal health that should always be noted when analyzing a dental radiograph.

e. **Lamina Dura**
   1. The alveolar bone proper is the thin layer of dense bone that lines a normal tooth socket. In radiographic images, the alveolar bone proper is referred to as the **lamina dura**.
   2. The presence of the lamina dura is another indicator of periodontal health that should always be noted when analyzing a dental radiograph.
   3. On a dental radiograph, the lamina dura appears as a continuous white (radiopaque) line that runs along the entire socket wall (**Figure 20-7**).
   4. Note that on a radiograph, the lamina dura is continuous with the cortical bone layer of the crest of the interdental septa.
   5. The precise appearance of the lamina dura on a dental radiograph can be affected by the complex contours of individual tooth roots or by changing the angulation of the x-ray tube (**Figure 20-8**).

f. **Periodontal Ligament Space**
   1. The space between the tooth root and the lamina dura of the socket is filled with the periodontal ligament tissue. The periodontal ligament tissue functions as the actual attachment of the tooth to the lamina dura of the socket.
   2. The appearance of the periodontal ligament space is another indicator of periodontal health that should always be noted when analyzing a dental radiograph.

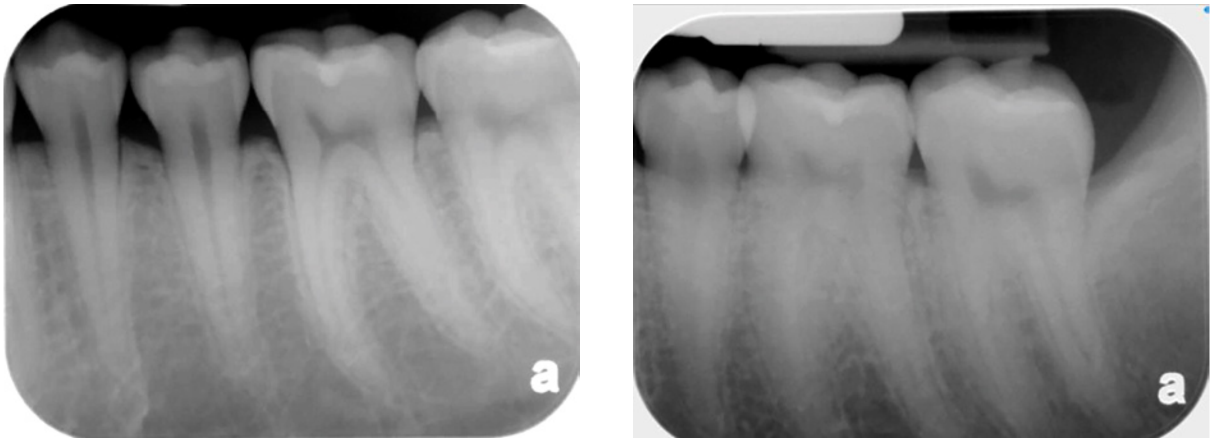

Figure 20-8 Appearance of the Lamina Dura on Two Different Radiographs. These two radiographs were taken at the same appointment from a single patient. In the left radiograph, the lamina dura is distinct and well defined. However, in the right periapical radiograph, the lamina dura appears fuzzy and is difficult to detect in some areas due to differences in x-ray tube angulation.

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3. As already noted, the periodontal ligament tissue does not resist penetration of x-rays and, therefore, appears on the radiograph as a thin radiolucent black line surrounding the tooth root.
4. Extremely widened periodontal ligament (PDL) spaces always need to be evaluated further (**Figure 20-9**).

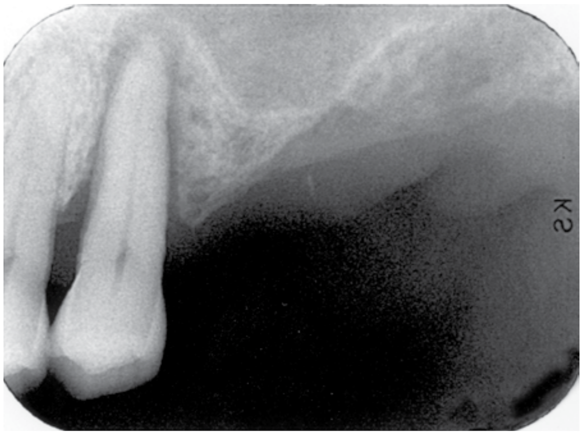

Figure 20-9 Extreme Widening of the PDL Space. This maxillary second premolar has an extremely widened periodontal ligament space which will require further evaluation.

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Section 2

Use of Radiographic Images During Periodontal Evaluation

1. **Review of Some Basic Techniques for Obtaining Good Quality Radiographs.** Many outstanding textbooks are available that explain dental radiology in detail. Such detail is not appropriate for this chapter, but the following are a few straightforward suggestions to consider when dental radiographs are intended for use as adjuncts to clinical periodontal evaluations.

a. **Long-Cone Paralleling Technique for Periapical Radiographs.** The long-cone paralleling technique can provide radiographs that are more anatomically accurate when compared with other intraoral radiographic techniques (such as the bisecting-angle technique).

b. **Long-Cone Paralleling Technique for Bitewing Radiographs.** Combining the long-cone paralleling technique with the use of bitewing radiographs provides excellent diagnostic images of the periodontal structures.[3–5]
   1. Utilizing poor radiographic technique can result in excessive vertical angulation and can obscure alveolar bone loss associated with periodontitis.
   2. If the clinician inadvertently uses poor techniques to position the radiographic film or x-ray beam, then the radiograph may overestimate or underestimate the true outline of the alveolar bone (**Figure 20-10**).

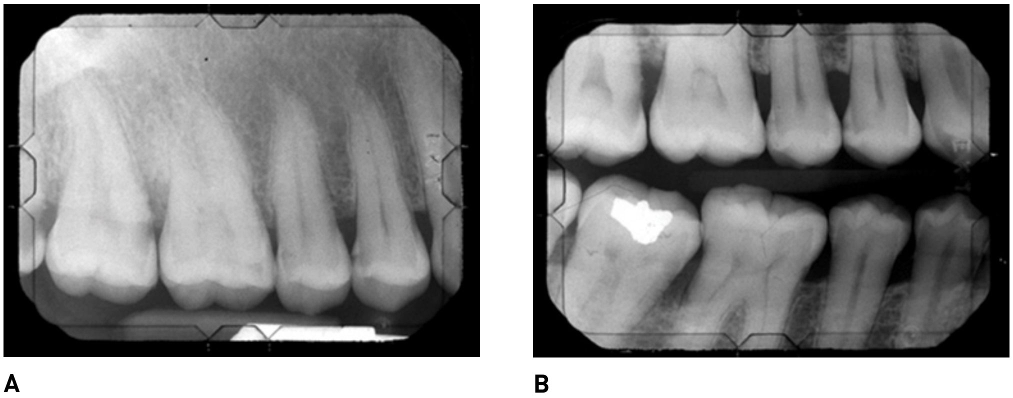

Figure 20-10 Periapical vs. Bitewing Radiograph. A. Excessive Vertical Angulation. Note how the crestal bone height appears distorted in the periapical radiograph shown here as opposed to the bitewing radiograph. **B. Bitewing Radiograph.** This bitewing radiograph shows the same maxillary teeth as the one with excessive vertical angulation above. Note the striking difference between the apparent bone height from these radiographs.

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      a. For this reason, many clinicians utilize bitewing radiographs as the primary radiographic image to evaluate crestal bone height (rather than periapical radiographs).
      b. It should be noted again that proper long-cone paralleling technique can minimize distortion of crestal bone height on periapical radiographic images and improve their usefulness.

c. **Vertical Bitewing Technique for Periodontal Patients.** Extensive alveolar bone loss may not be visible on a horizontal bitewing radiograph. The use of vertical bitewing radiographs, on the other hand, may help offset this problem.
   1. Alveolar bone loss of 5 mm or greater may not be seen at all on a normal horizontal bitewing radiographic image.
   2. Vertically oriented bitewings may be used in these situations to increase visualization of the alveolar bone.
   3. An adaptor is available for most film holders to accomplish this vertical orientation.
   4. Using the vertical bitewing technique, the long axis of the film is rotated 90 degrees to be perpendicular to the occlusal plane (**Figure 20-11**).
   5. Vertical bitewing radiographic images show more of the interproximal bone than horizontal bitewings, especially when the teeth are widely separated by the film holder (**Figure 20-12**). Vertical bitewings also show more of the root trunk and root anatomy compared to horizontal bitewings.

d. **Long-Grayscale/Low-Contrast Images in Periodontal Patients.** Long-grayscale/low-contrast radiographic images have many visible shades of gray that make it easier to see some of the subtle changes such as alveolar bone loss in periodontal disease. These images can be obtained using high-kVp exposures (70 to 100 kVp) or using digital imaging software adjustments to maximize the grayscale of normally exposed images. In addition, many software programs now provide presets to optimize for detection of periodontal disease.

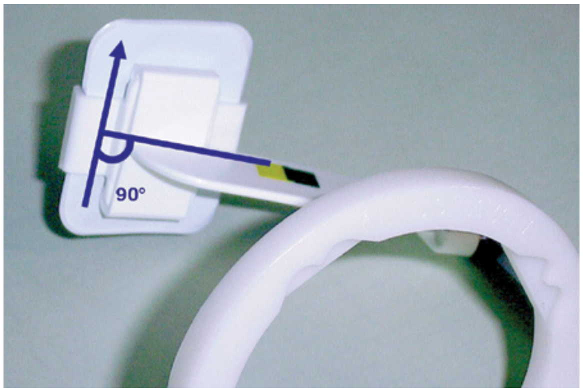

Figure 20-11 Film Placement for Vertical Bitewing. A #2 periapical film positioned for taking a vertical bitewing radiograph. Note how the film is rotated 90 degrees from the usual orientation.

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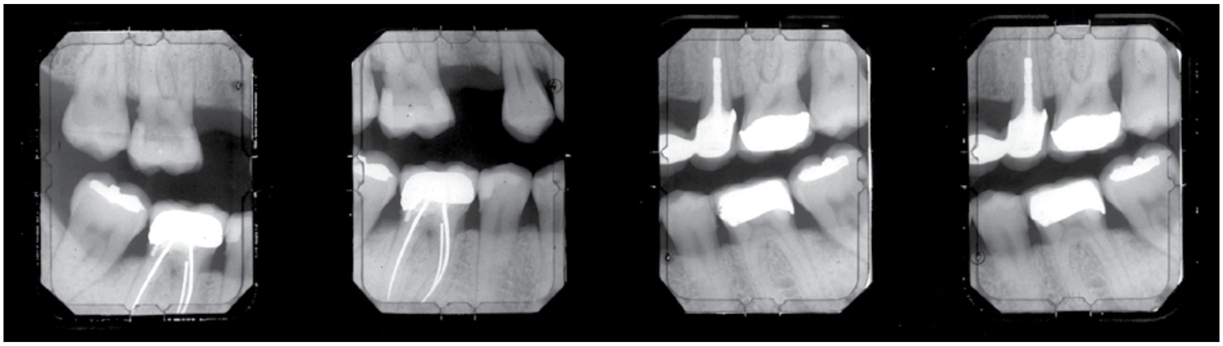

Figure 20-12 Four-Film Vertical Bitewing Series. Note how much interproximal bone is visible on these vertical bitewings despite the separation of the teeth by the positioning device.

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2. **Limitations of Dental Radiographs During Periodontal Evaluation.** Despite the high value of the information obtained from dental radiographs, there are several limitations in the use of these dental radiographs that should always be kept in mind. Some of these limitations are outlined below.
   a. *Radiographs are two-dimensional images of three-dimensional structures.* A radiograph provides a two-dimensional image of a very complex three-dimensional structure (i.e., a tooth and the surrounding periodontal structures). The fact that the radiograph is a two-dimensional image can often be misleading to the viewer. Two examples that frequently cause confusion during radiographic interpretation are (1) that the buccal alveolar bone can obscure bone loss on the lingual aspect of a tooth and (2) that the palatal root makes it difficult to visualize furcation alveolar bone loss on a maxillary molar tooth (**Figure 20-13**).
   b. *Information obtained from a dental radiograph is primarily limited to information about calcified structures.* This fact has already been discussed, but is important enough to state again. Radiographic images do not normally provide information about the noncalcified (nonmineralized) components of the periodontium that, of course, play a leading role in the development and progress of periodontitis.
   c. *Radiographs provide only limited information about certain critical aspects of the periodontium.* Dental radiographic images do not reveal the following: the presence or absence of periodontal pockets, the presence of early stages of alveolar bone loss, the precise morphology of any existing alveolar bone destruction, the presence of tooth mobility and its extent, incipient furcation invasions (Glickman Grade I furcation), the level of the epithelial attachment on the tooth, or information about periodontal disease activity. These limitations are discussed below.
      1. Dental radiographs do not reveal the presence or absence of periodontal pockets.
         a. Because the periodontal pocket is composed of soft tissue, it will not be visible on the radiograph.

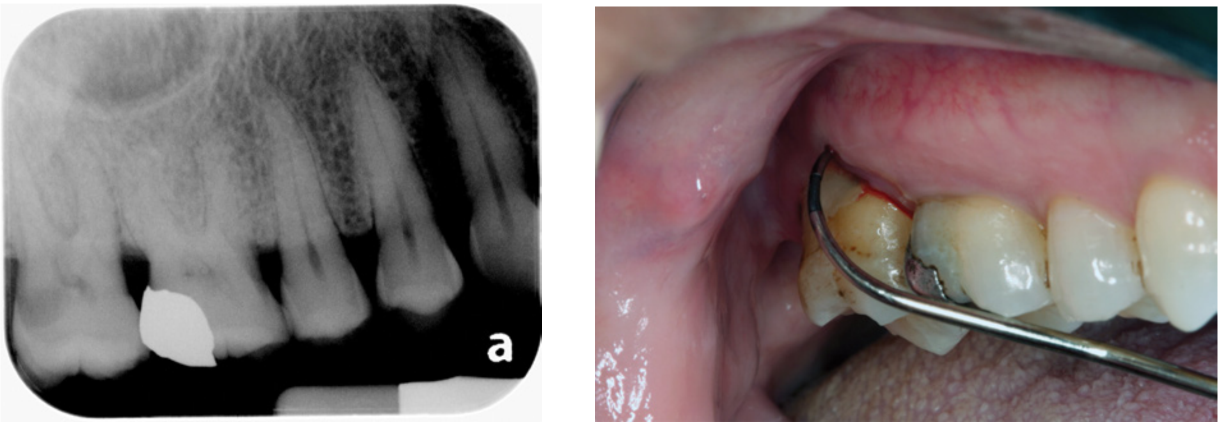

Figure 20-13 **Limitations of Two-Dimensional Conventional Radiographs.** The overlying buccal cortical plate and the underlying palatal root obscure any radiographic sign of mid-buccal furcation involvement on the maxillary molars. As seen in the accompanying clinical photograph, however, clinical evaluation of the area with a curved Nabers probe indicates the presence of a furcation invasion on the maxillary second molar.

Courtesy of Dr. Jennifer Chang, University of Texas Dental School at Houston, TX.

   b. The only reliable method of locating a periodontal pocket and evaluating its extent is by careful periodontal probing.
2. Dental radiographs do not reveal the presence of early stages of alveolar bone loss.
   a. The very earliest signs of periodontitis must be detected clinically, not radiographically.
   b. It should be noted that by the time bone loss due to periodontitis becomes detectable on the dental radiograph, the disease usually has progressed well beyond the earliest stages.
3. Dental radiographs do not reveal the precise morphology of any existing alveolar bone destruction.
   a. Conventional dental radiographs cannot accurately display the precise shape of alveolar bone deformities because these are not three-dimensional images. For instance, any bony changes that occur in the buccal or lingual cortical bony plate cannot be seen on a conventional radiograph because it is obscured by the dense root structure. As such, it is very difficult to detect a bony fenestration or dehiscence from a conventional radiograph (**Figure 20-14**).

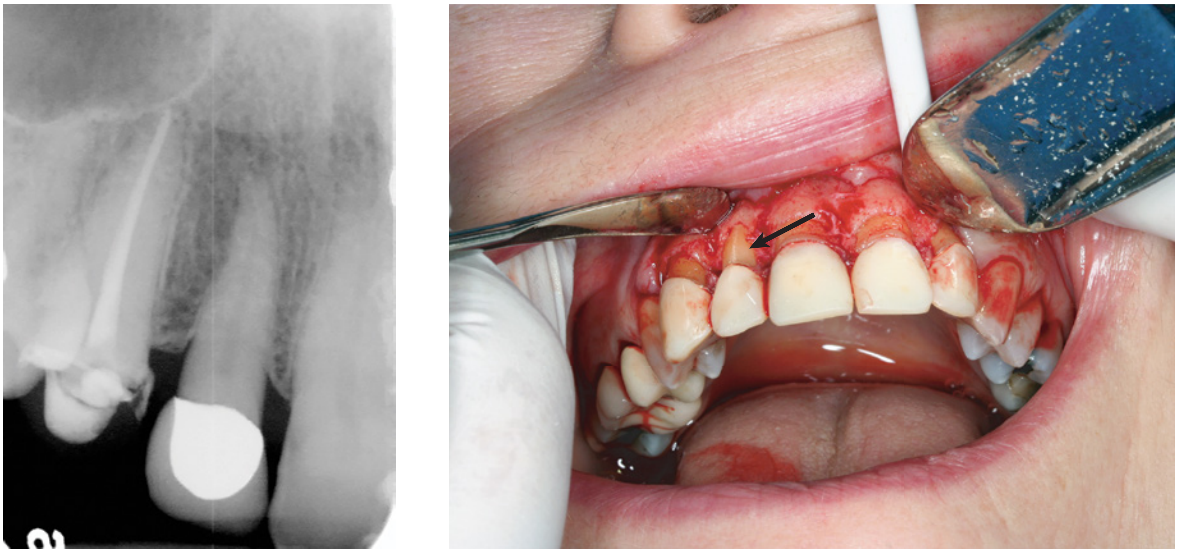

Figure 20-14 **Bony Dehiscence.** From this case, the radiograph fails to reveal the presence of a bony dehiscence. However, following surgical flap reflection, a bony dehiscence on the maxillary lateral incisor is apparent (black arrow). (Note: Crowns were placed on the maxillary canine and central incisor soon after the radiograph was taken, but prior to flap surgery.)

Courtesy of Dr. Jennifer Chang, University of Texas Dental School at Houston, TX.

   b. Alveolar bone loss from periodontitis can result in complex and convoluted contours of the remaining alveolar bone that may not be detectable on a conventional radiograph.
4. Dental radiographs do not always reveal the presence of alveolar bone loss in furcation areas.
   a. Radiographic images frequently appear to show more interradicular bone (alveolar bone between the roots of the teeth) than is actually present. The facial and lingual aspects of the alveolar bone will often be superimposed over the furcation and hide bone loss from view, thereby obscuring the presence and the extent of the furcation invasion (**Figure 20-15**).

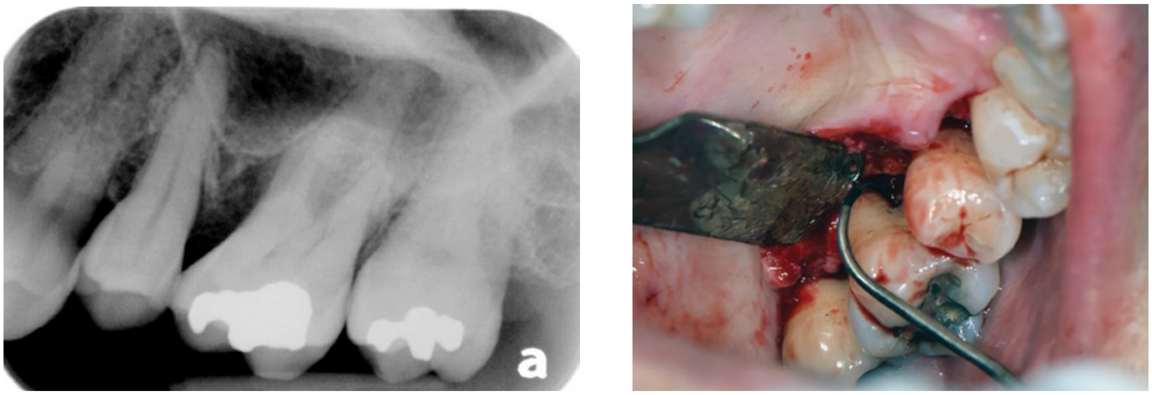

Figure 20-15 Limitation of Radiographs in Detecting Furcation Bone Loss. An angular defect on the mesial of the maxillary first molar is clearly visible on the periapical radiograph. However, following flap reflection, a mesiobuccal furcation invasion that was not radiographically apparent is present.

Courtesy of Dr. Takaaki Kishimoto, Oita, Japan.

   b. Also, slight variations in alignment of the x-ray beam may conceal the presence or extent of furcation alveolar bone loss.
   c. Furcation involvement (attachment and bone loss between the roots) must be evaluated by clinical examination with a curved furcation probe, as discussed in Chapter 19.
5. Dental radiographs do not give any information regarding tooth mobility. Although it is indeed possible to visualize the width of some periodontal ligament spaces on dental radiographs, the only way to accurately determine the presence of tooth mobility is during the clinical evaluation.
6. Dental radiographs do not reveal the level of the epithelial attachment on the tooth.
   a. Because the epithelial attachment to the tooth is soft tissue, this structure will not be apparent on a dental radiograph.
   b. The only way to identify the level of epithelial attachment is through careful periodontal probing.
7. Dental radiographs do not reveal any information about periodontal disease activity.
   a. Radiographic images do not reveal that periodontal disease activity is taking place at the time the radiographs were taken. They only show what destruction has happened at some time in the past (i.e., before the radiograph was taken—in some cases even years before).
   b. It is important to remember that the radiographic examination itself is never a satisfactory substitute for a clinical periodontal assessment.

3. **Benefits of Dental Radiographs During Periodontal Evaluation.** Despite the limitations of dental radiographs, the periodontal evaluation is never complete without supplementing clinical information with accurate radiographic images. Examples of what dental radiographs may reveal are bony changes associated with periodontitis; important anatomical information regarding the tooth root morphology (i.e. root trunk length, number of roots on a multi-rooted tooth, length of the root(s) presence of enamel pearls, etc.); the relationship of the maxillary sinus to the periodontal deformity; widening of periodontal ligament space; advanced furcation bone loss; alveolar bone loss due to periodontal abscesses; and the presence of some local factors, such as overhanging restorations, marginal ridge height discrepancies, open contacts and calculus (**Table 20-1**).
   a. **Assessment of Alveolar Bone Changes.** As already discussed, by the time alveolar bone loss is detectable on the dental radiograph, the disease usually has progressed well beyond the earliest stages. Nevertheless,

Table 20-1 Radiographic Signs Associated with Periodontal Disease

Condition	Radiographic Sign(s)
Early bony changes	Break or fuzziness at the crest of the interdental alveolar bone Widening of the periodontal ligament space at crestal margin
Horizontal bone loss	Can be measured from a plane that is parallel to a tooth-to-tooth line drawn from the CEJs of adjacent teeth
Vertical bone loss	Seen as more bone loss on the interproximal aspect of one tooth than on the adjacent tooth; bone level is at an angle to a line joining the CEJs
Bone defects	Are radiolucent due to bone loss and therefore visible on radiographic images, although three-dimensional structure of the bone defect may be hard to determine from conventional radiographs alone.
Furcation alveolar bone loss	Loss of alveolar bone in furcation area may be detectable as a triangular radiolucency especially on mandibular molars
Unfavorable crown-to-root ratio	Seen as a greater portion of the tooth coronal to the alveolar crest compared to the portion of tooth structure apical to the alveolar crest
Peri-implant bone loss	Bone loss around a dental implant

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it is important to examine radiographs for the earliest radiographic signs of periodontal disease. Two early radiographic signs of periodontitis are (1) crestal irregularities and (2) triangulation of the periodontal ligament space.

1. **Crestal Irregularities.** **Crestal irregularities** are the appearance of breaks or fuzziness at the interdental crest, instead of the normal intact lamina dura (**Figure 20-16**).

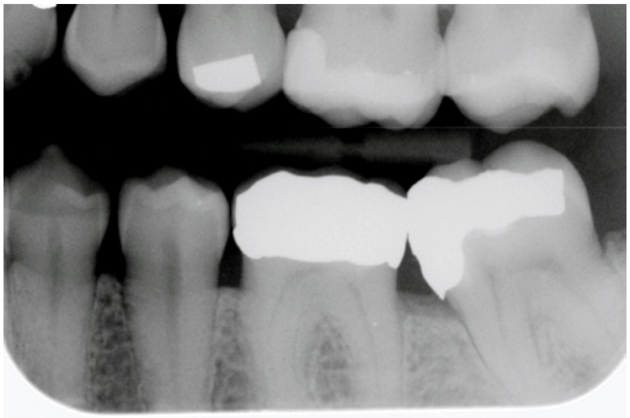

Figure 20-16 Crestal Irregularities. The interproximal crestal bone level between the mandibular first molar and the mandibular second molar appears indistinct and irregular. Additionally, note the overhanging restorative margin on the mesial of the mandibular second molar that is contributing to the localized crestal irregularity.

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2. **Triangulation (Funneling).** **Triangulation** is the widening of the periodontal ligament space caused by the resorption of bone along either the mesial or distal aspect of the interdental (interseptal) crestal bone (**Figure 20-17**).

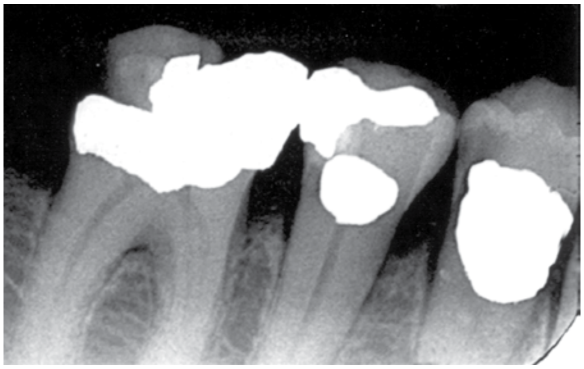

Figure 20-17 Triangulation. The crestal bone between these mandibular teeth demonstrates triangulation, a pointed, triangular appearance.

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b. **Horizontal Versus Vertical Alveolar Bone Loss Due to Periodontitis.** Alveolar bone loss from periodontitis is described as either horizontal bone loss or vertical (or angular) bone loss.
   1. The extent and direction of bone loss is determined using the CEJ of adjacent teeth as the points of reference.
   2. **Horizontal bone loss** is alveolar bone loss that is parallel to an imaginary line drawn between the CEJs of adjacent teeth and is beyond the normal 2 mm distance from the CEJ (**Figure 20-18**). In health, the normal horizontal bone levels are within 2 mm from the CEJ. In contrast, a crestal bone level greater than 2 mm from the CEJ is indicative of horizontal bone loss.
   3. **Vertical bone loss** occurs when there is greater bone destruction on the interproximal aspect of one tooth than on the adjacent tooth so that the bone meets the tooth at an acute angle (**Figure 20-19**). The presence of vertical bone loss is often a radiographic diagnostic sign of severe periodontitis.[2]

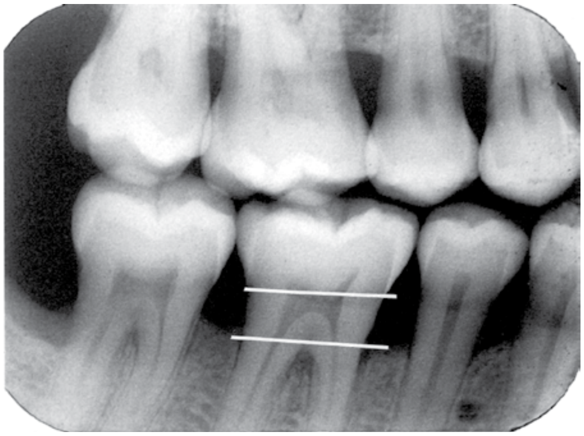

Figure 20-18 Horizontal Bone Loss. Horizontal bone loss is parallel to an imaginary line drawn between the CEJs of adjacent teeth.

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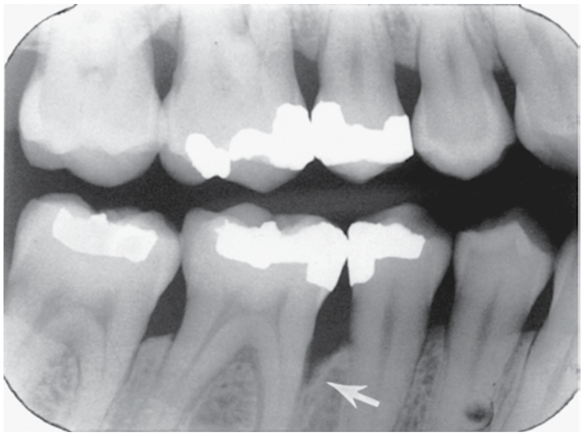

Figure 20-19 Vertical Bone Loss. The arrow points to vertical bone loss associated with the mesial surface of the mandibular first molar.

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c. **Assessment of Alveolar Bone Loss in a Furcation**
   1. As mentioned previously, furcation alveolar bone loss will not usually be seen on the dental radiograph until the bone destruction extends past the furcation area.
      a. Furcation bone loss on mandibular molar teeth is easier to detect on a dental radiograph than furcation bone loss on maxillary molar teeth because mandibular molars have only two roots, a

mesial root and a distal root. On the radiograph, the clinician can usually visualize a diminished radiodensity between these two roots (**Figure 20-20**).

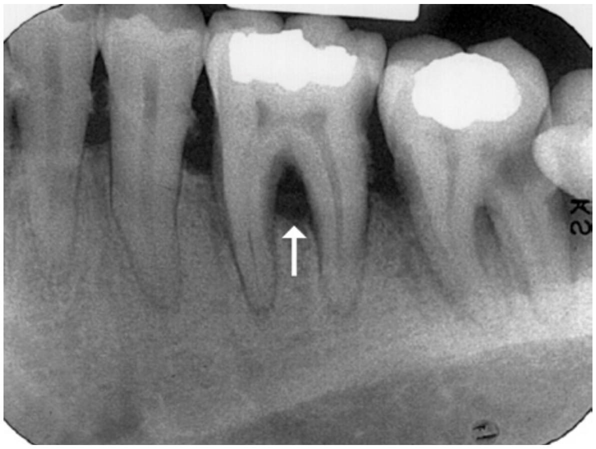

Figure 20-20 **Furcation Involvement.** The furcation alveolar bone loss is easily visible on the mandibular first molar in this radiograph. Furcal bone loss to this degree can be easily confirmed by using a Nabers probe.

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   b. Furcation bone loss on maxillary molar teeth is more difficult to detect on a radiograph than furcation bone loss on mandibular molars because maxillary molars have three roots: a mesiobuccal root, a distobuccal root, and a palatal root. The palatal root is often superimposed over some of the furcation areas of the tooth on the radiograph and masks (hides) any radiolucency there.

2. It is a general rule that actual furcation bone loss is often greater than what the radiograph reveals. Hence, furcation areas should always be probed with a curved Nabers probe during a clinical periodontal evaluation.
3. If severe alveolar bone loss is evident on the mesial or distal surface of any multirooted tooth (especially maxillary molars), furcation involvement should be suspected.
   a. On a radiograph, an interproximal furcation invasion appears as a small, triangular radiographic shadow across the mesial or distal root of a maxillary molar. This radiographic finding is known as a furcation arrow (**Figure 20-21**).

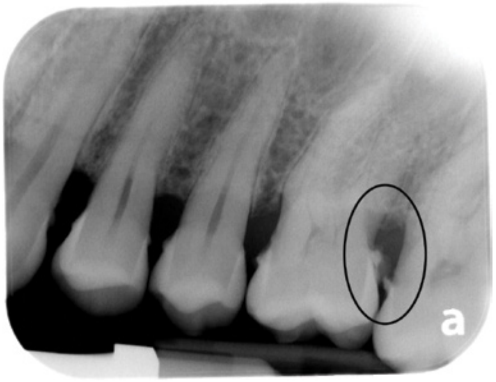

Figure 20-21 **Furcation Arrow.** Note the small, triangular radiolucent shadow (circled) that falls across the distobuccal root of the maxillary first molar. This shadow is indicative of an interproximal furcation invasion. This is known as a furcation arrow.

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   b. However, as previously discussed, the furcation arrow may be difficult to visualize from a radiograph due to the complex root anatomy or thickness of the overlaying buccal cortical bone. In addition, it is important to keep in mind that the absence of a furcation arrow is not indicative of an absence of a bony furcation involvement.
   c. As in all cases, the presence of furcation bone loss must be confirmed clinically with a curved furcation probe.

d. **Assessment of Peri-Implant Bone Loss**
   1. Two-dimensional radiographs are useful in detecting peri-implant bone loss (bone loss around a dental implant) (**Figure 20-22**).

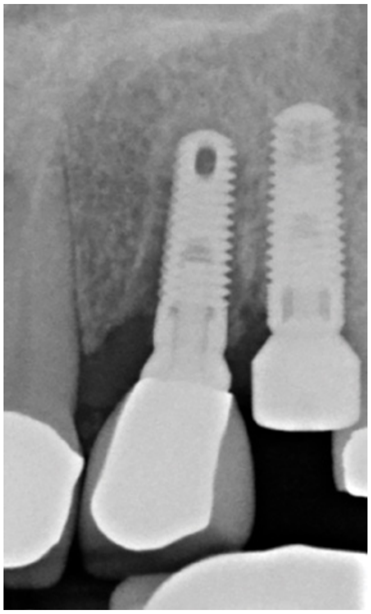

Figure 20-22 **Radiographic Sign of Peri-Implantitis.** Note the loss of radiodensity around this osseointegrated implant. This is indicative of peri-implant bone loss.

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   2. Peri-implant bone loss is one of the major signs of peri-implantitis. In its simplistic terms, peri-implantitis is defined as an oral inflammatory process that affects the soft tissue and the hard tissue around an osseointegrated implant.

e. **Special Considerations in the Assessment of Alveolar Bone Loss**
   1. The clinician should keep in mind that the radiograph reveals the bone remaining rather than the amount of bone lost. The amount of bone loss needs to be estimated as the difference between the level of the remaining bone and the expected bone height.
   2. Alveolar bone loss can occur on any or all surfaces of a tooth; however, tooth roots tend to mask (or hide) bone loss on the facial and lingual sides of the teeth.

f. **Assessment of the Crown-to-Root Ratio**
   1. The crown-to-root ratio is a measure of the length of the tooth coronal to the alveolar crest of bone compared with the length of the root embedded in the bone, as determined using a periapical radiograph.
      a. The crown-to-root ratio determines the ability of a tooth to be maintained without becoming mobile and eventually lost.
      b. The crown-to-root ratio is determined using a radiograph of the tooth.
      c. The ideal crown-to-root ratio is approximately 1:2 (**Figure 20-23**).
   2. The crown-to-root ratio plays an important role in determining the periodontal prognosis of an individual tooth. It should also be a key consideration when evaluating whether a tooth is suitable to act as an abutment for a fixed or removal denture prosthesis.

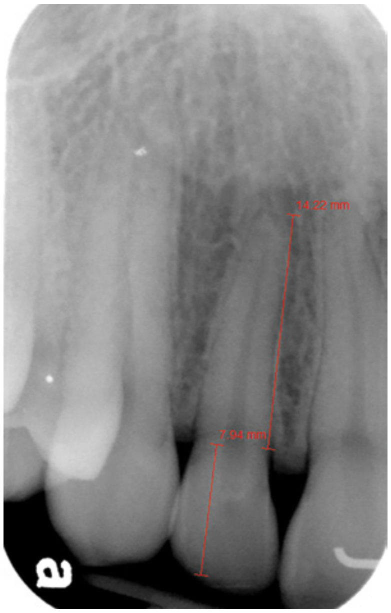

Figure 20-23 Favorable Crown-to-Root Ratio. On the maxillary lateral incisor, note the favorable crown-to-root ratio of approximately 1:2.

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g. **Recognition of Local Contributing Risk Factors on Dental Radiographs.** In some instances, it is possible to identify local contributing risk factors for periodontitis on dental radiographs. Examples of local contributing risk factors that may be apparent on the dental radiograph are calculus deposits and faulty restorations.
   1. **Calculus Deposits.** The only accurate way to detect calculus deposits is with an explorer; however, large calculus deposits may indeed be visible on a dental radiograph.
   2. **Faulty Restorations.** Faulty dental restorations are common contributing factors in patients with periodontal disease. In many cases, faulty restorations can be detected on a radiograph (**Figure 20-24**).

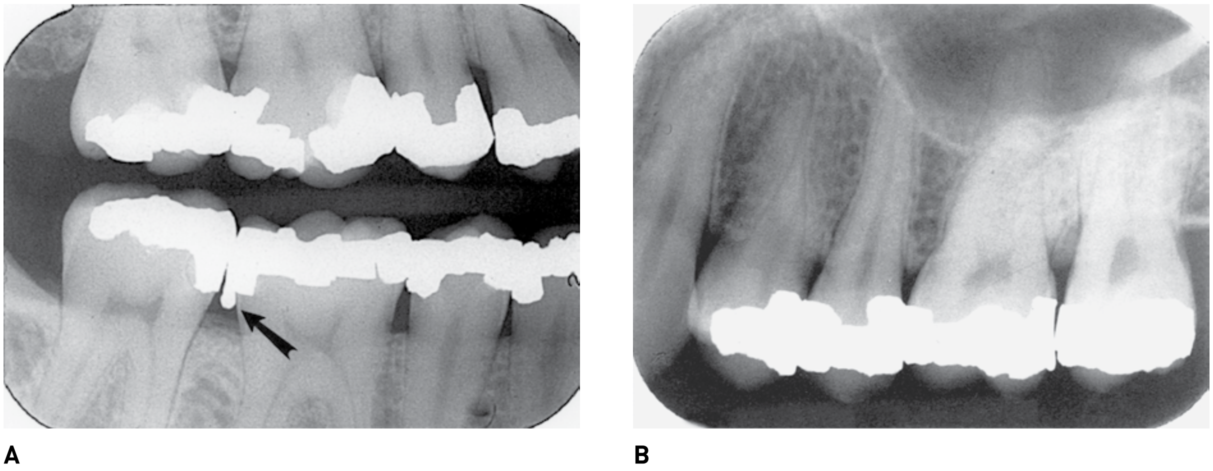

Figure 20-24 Faulty Restorations. A. The distal surface of the mandibular first molar, indicated by the arrow, has a faulty restoration that can harbor plaque biofilm and prevents thorough daily self-care. **B.** The distal proximal tooth surface of the maxillary first molar and the mesial tooth surface of the second molar have been restored but the restorations do not reproduce their original shape and contour. These faulty contours can allow food impaction at the site, which, in turn, may cause direct damage to the periodontium at the site.

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4. **Beyond Conventional Dental Radiography.** Modern radiology offers an interesting array of imaging methods that have been suggested for possible use in place of conventional dental radiography. One of these imaging methods is the use of **cone beam computed tomography (CBCT)** (**Figure 20-25**). Dental cone beam computed tomography generates detailed three-dimensional images of the periodontium as well as of other anatomic structures in the head and neck region (**Figure 20-26**).

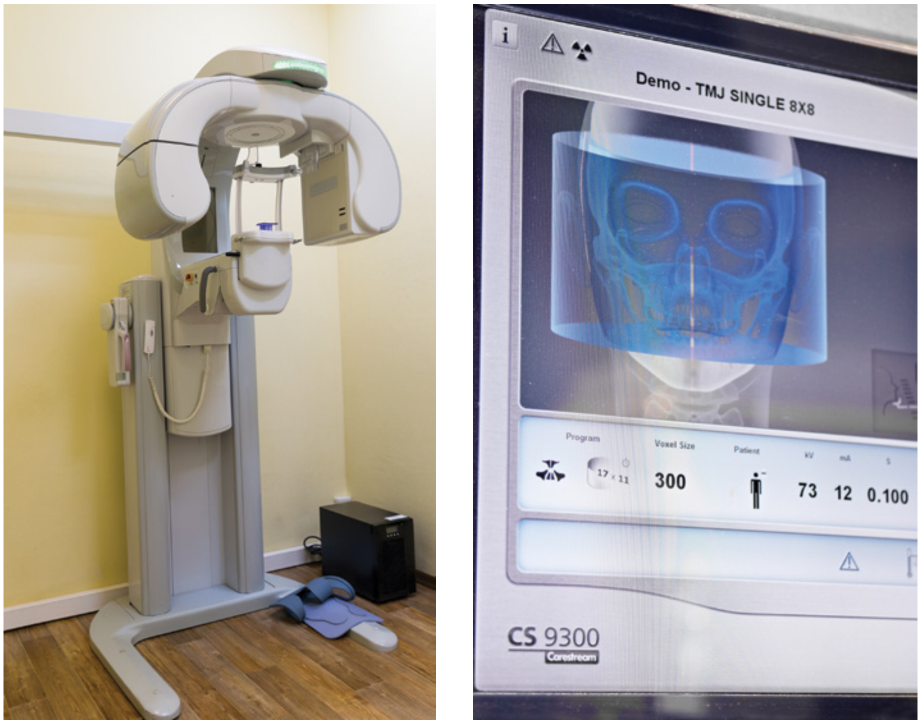

Figure 20-25 **Dental Cone Beam Computed Tomography.** On the left, an example of a digital dental CBCT machine. The photograph on the right shows an example of the digital image on a computer screen.

Left: ArtEvent ET/Shutterstock; **Right:** © Robert Kneschke/Shutterstock

a. **Dental Implant Treatment Planning**
   1. Accurate three-dimensional imaging is indispensable for treatment planning the surgical placement of dental implants, as well as for complex surgeries involving the head and neck structures, such as orthognathic surgery and sinus surgery.
   2. Using conventional radiography for treatment planning implant cases is no longer considered an accepted practice and is highly risky considering all the complex anatomy in the head and neck region that potentially can be irreversibly damaged without a precise idea of where these anatomic structures are located. In contrast, CBCT imaging is now considered part of the routine pre-operative (pre-surgical) imaging scans required for all implant cases. As such, CBCT is now the standard of care when treatment planning implant cases.
   3. CBCTs offers the clinician a tremendous amount of valuable pre-operative treatment planning–related information that ensures a higher degree of safety and greater predictability of the final outcome. Some of these benefits are:
      a. Precise placement and orientation of implants within the bone.
      b. Correct selection of appropriately sized implant for optimal support, maximum integration, and enhanced mechanical stability within the bone.
      c. Prevention of injury to anatomical vital structures, such as nerves and maxillary sinus (when placing implants in the maxillary posterior region). For example, as seen on the CBCT's panoramic view in Figure 20-26, a gold-colored line traces the path of the inferior alveolar nerve which is one of the most critical anatomic structures that a clinician should be aware of prior to placing an implant in the mandibular posterior region. A conventional radiograph would not give such precise information regarding the position and location of the inferior alveolar nerve.

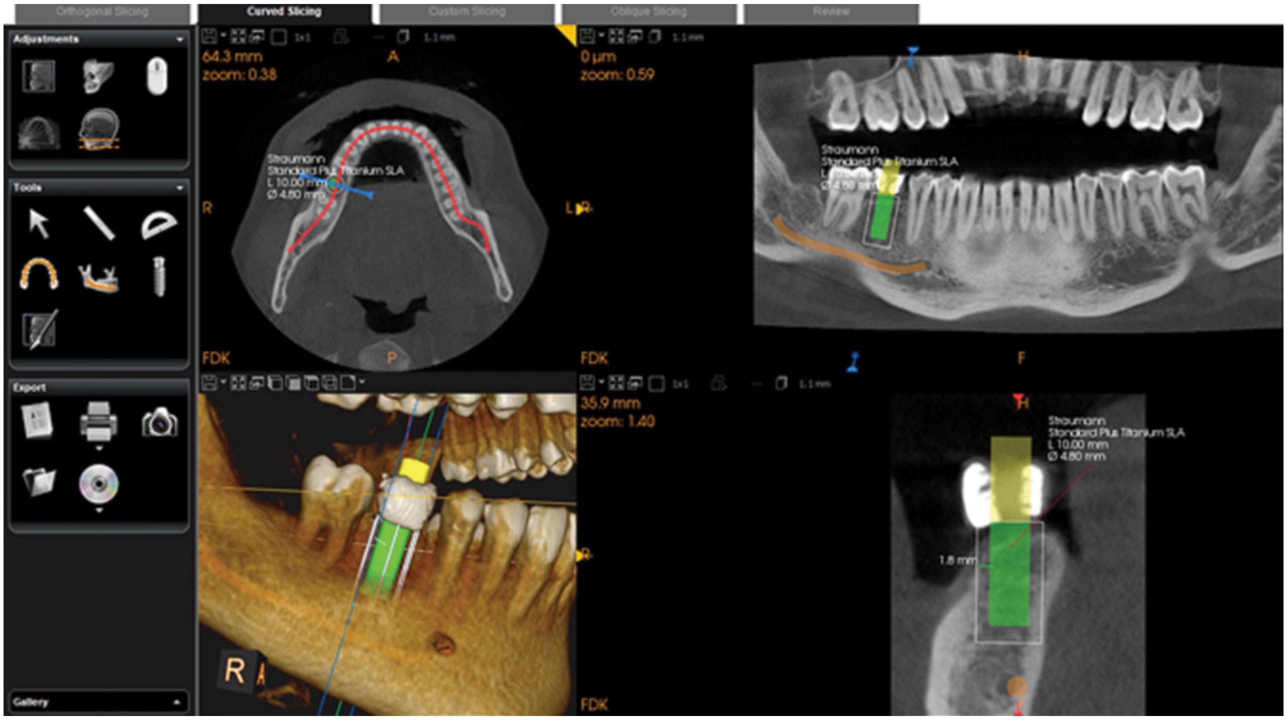

Figure 20-26 **CBCT Images.** Initial imaging assessment during the implant treatment planning stage (the pre-operative stage) is best achieved with CBCT imaging. Compared to conventional two-dimensional radiography, CBCT delivers much more superior images with enhanced visualization of the dentition, maxillofacial skeleton, and the anatomic vital structures, while, at the same time, gives the surgeon an opportunity to properly pre-operatively plan out the case before the day of surgery. Equally important is the fact that unlike two-dimensional radiography, a typical CBCT scan reconstructs a three-dimensional rendition of the patient's oral cavity so vital anatomic structures can be more precisely located. As seen in this figure, multiple images of the implant surgical site (in this case, the mandibular right posterior region) can be viewed: the upper left scan represents the axial view (superior-to-inferior view, or from top-to-bottom view); the upper right image represents the panoramic view; the lower left image represents the three-dimensional image view (this view can be rotated in any direction on the computer program); and the lower right image represents the sagittal view, which helps the clinician envision the precise location of the implant in a buccolingual dimension. Note the gold line on the panoramic, three-dimensional image and sagittal view that traces the precise path of the inferior alveolar nerve relative to the proposed implant site. Additionally, note that some CBCT programs give the clinician the capability to interactively simulate the position, location, and orientation of the future implant prior to the actual surgery. The implant simulation can be maneuvered and manipulated within the framework of the virtual three-dimensional imaging system.

Courtesy of Dr. Brandi Herron, Indianapolis, IN.

b. **Nondental Implant Treatment Planning (e.g., Flap for Access Surgeries)**
   1. Most dental clinicians rely on conventional intraoral radiographs, not CBCT scans, to detect and diagnose interproximal bone loss. This is because CBCT performance has not yet been confirmed as superior to that provided by conventional intraoral radiographs in the detection of interproximal bone loss.
   2. CBCT imaging does not allow for distinguishing different types of soft tissue, such as discriminating junctional epithelium from connective tissue.
   3. Also, unfortunately at this point, CBCT emits higher absorbed radiation doses to patients compared to conventional dental radiography.
   4. From the research available, there does not appear to be sufficient scientific evidence to justify the routine use of CBCT for the diagnosis and treatment planning for patients with periodontitis with infrabony/intrabony periodontal defects or furcation defects.[6]
   5. Recently, the American Academy of Periodontology convened an expert panel to assess the clinical value of using CBCT to diagnose, treatment plan, and manage periodontal disease. Although the

expert panel believed that the application of CBCT imaging could have potential value, the consensus was that there is currently limited evidence to support the routine use of CBCT in routine periodontal treatment planning. However, the panel suggested that CBCT imaging may be a useful adjunctive diagnostic tool to evaluate selected periodontal cases on a case-by-case basis.[7]

c. With its higher resolution and increased imaging accuracy, CBCT is an advanced point-of-care imaging modality. However, CBCT imaging should be used only when the potential benefits to the patient outweigh the risks. Dental health care professionals should consider CBCT imaging only when they expect the diagnostic information yielded will lead to better patient care, enhanced patient safety, and ultimately facilitate a more predictable, optimal treatment outcome. Further, it should be used selectively as an adjunct to, not in lieu of, conventional two-dimensional radiography.

Chapter Summary Statement

The use of dental radiographs to supplement information obtained from the clinical periodontal assessment is a critical step in patient care. Although there are limitations in the information obtained from conventional dental radiographs, these radiographic images are still an important diagnostic aid in the examination and diagnosis of patients with periodontitis. Radiographic images can be extremely useful tools in the detection of alveolar bone changes due to periodontitis such as crestal irregularities, triangulation, and interseptal bone loss, in addition to the detailed assessment of alveolar bone defects and furcation alveolar bone loss.

According to the current best available evidence, CBCT imaging plays a vital role in providing important information for treatment planning of implant or advanced oral reconstructive surgery. However, for the management and treatment planning of periodontitis cases, the routine use of CBCT may provide limited information that is not otherwise revealed by conventional radiographs.

Section 3

Focus on Patients

Clinical Patient Care

CASE 1

Mr. Jones is a new patient in your dental office. He brings with him some recent full-mouth radiographs that reveal no evidence of alveolar bone loss. While studying a copy of the patient's dental chart, you note that there is a diagnosis of Stage II, Grade A periodontitis. How might you explain the apparent discrepancy between the lack of radiographic evidence of bone loss and the diagnosis of periodontitis?

CASE 2

During a periodontal assessment for a new patient, you detect clinical attachment loss. When you suggest that the patient needs dental radiographs, the patient objects because she does not want to be exposed to "unnecessary x-rays." How might you address the patient's concerns?

CASE 3

While reviewing a new set of dental radiographs for a patient, you note numerous sites of obvious alveolar bone loss. How can you determine if the radiographs display horizontal or vertical bone loss?

Ethical Dilemma

Philomena C. is your first patient of the morning. She was born in Italy, and moved to the United States with her family when she was 10. She is a 60-year-old homemaker, who appears slightly overweight, and admits to high blood pressure and high cholesterol, both controlled by medication. She has been a patient in the practice of your dentist's sister, Dr. Lynne, who has an office across town, for the last 30 years. She recently decided to change dental practices as our office is near her residence.

Philomena has been faithful with her recall visits, and has followed all the treatments that Dr. Lynne and the various hygienists have suggested during her years as a patient in that practice, including routine radiographs.

You begin probing, and Philomena questions what you are doing. She states that "no one has ever done that to her teeth and gums," and quite frankly finds it very uncomfortable. Philomena says that she thought her oral health could be evaluated by the "full set of x-rays" that she received every few years.

Your clinical exam reveals that Philomena presents with generalized tooth mobility and early furcation involvement, especially on the maxillary molars. Her probe readings are generalized 4 to 6 mm in the posterior sextants. She assumed that her mouth was in good health, and is shocked to find out otherwise.

1. What ethical principles are in conflict in this dilemma?
2. What is the best way for you to handle this ethical dilemma?
3. What is the best way to address/discuss Philomena's treatment plan?

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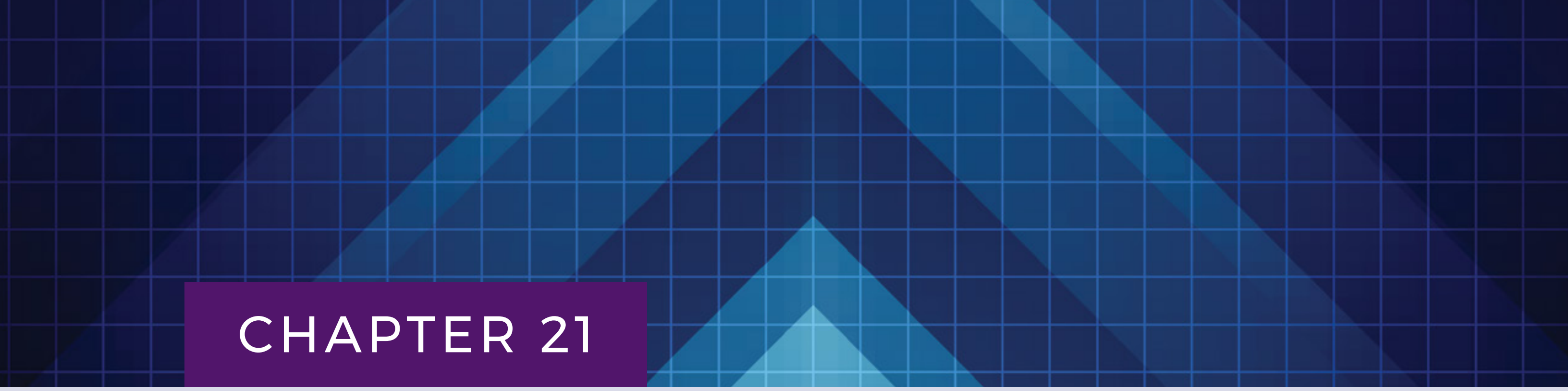

CHAPTER 21

Clinical Decision-Making for Periodontal Care

- Section 1 Guidelines for Arriving at a Periodontal Diagnosis
- Section 2 Guidelines for Periodontal Treatment Sequencing
- Section 3 The Need for Ongoing Decision-Making
- Section 4 Focus on Patients
  - Clinical Patient Care

CLINICAL APPLICATION

Members of the dental team are equipped with a broad array of skills that can be called upon when caring for patients with periodontal disease. All members of the dental team should be comfortable in providing many of the periodontal therapies when indicated. However, it is vital for the clinician to be capable of making sound decisions related to patient care to be an effective member of the health care team. This chapter outlines guidelines for decision-making by dental health care providers, including those related to arriving at a periodontal diagnosis, sequencing periodontal treatment, and obtaining consent for treatment and the ongoing need for decision-making.

LEARNING OBJECTIVES

- List the three fundamental diagnostic questions used when assigning a periodontal diagnosis.
- List the two fundamental diagnostic questions used when assigning a peri-implant diagnosis.
- Explain how to arrive at appropriate answers to each of the fundamental diagnostic questions.
- Explain the difference between the *signs* of a disease versus the *symptoms* of a disease.
- List several overt and hidden signs of periodontal inflammation.
- Define *silent disease*.
- Describe what is meant by the term *clinical attachment loss*.
- Describe the elements of a well-written diagnosis for periodontitis.
- List the phases of treatment.
- Explain why a patient's diagnosis and treatment plan may require modifications at a later point in time.

KEY TERMS

Clinical decision-making
Signs of periodontal disease
Symptoms of periodontal disease
Silent disease
Overt signs
Hidden signs
Natural level of the gingival attachment
Clinical attachment loss (CAL)
Master treatment plan
Assessment and preliminary therapy phase
Nonsurgical periodontal therapy phase
Surgical therapy phase
Restorative therapy phase
Periodontal maintenance phase

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Section 1

Guidelines for Arriving at a Periodontal Diagnosis

Clinical decision-making is the process whereby all members of the dental team use the information gathered during the comprehensive periodontal assessment to arrive at an appropriate diagnosis and identify treatment strategies that meet the individual needs of the patient.

The first step to periodontal treatment plannning is assigning a correct periodontal diagnosis (or correct diagnoses, if the patient has more than one known periodontal condition). Determination of a periodontal diagnosis can be simplified by asking and answering three fundamental clinical questions in a systematic manner (**Figure 21-1**). These three fundamental questions can be used to guide the dental team through the diagnostic process. Many decisions, including assigning a periodontal diagnosis and planning nonsurgical/surgical therapy, revolve around the answers to these fundamental questions.

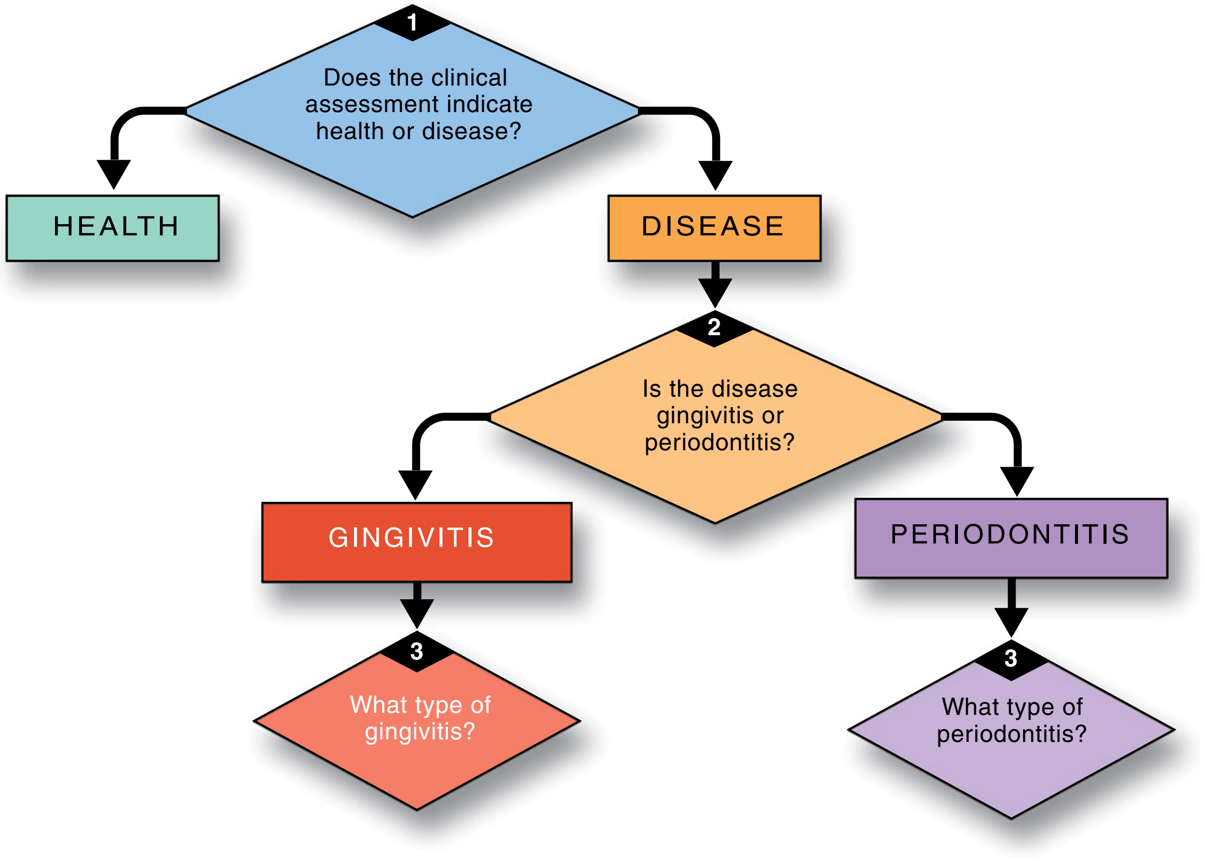
1
Does the clinical assessment indicate health or disease?
HEALTH
DISEASE
2
Is the disease gingivitis or periodontitis?
GINGIVITIS
PERIODONTITIS
3
What type of gingivitis?
3
What type of periodontitis?

Figure 21-1 Decision Tree for Determining a Periodontal Diagnosis. A decision tree illustrating the three fundamental questions for determining an initial periodontal diagnosis.

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1. **Answering Fundamental Diagnostic Questions When Assigning a Periodontal Diagnosis**
    a. **First Fundamental Diagnostic Question**: **"Does the clinical assessment indicate periodontal health or inflammatory disease?"**
        1. The answer to this question should be based on the clinical signs of inflammation that are noted and recorded during the clinical assessment. By the very nature of the question, it is not usually difficult for members of the dental team to answer.
            a. **Signs of Periodontal Disease.** Members of the dental team should be able to identify the *signs* of a disease and the *symptoms* of a disease. **Signs of periodontal disease** are the features of a disease that can be observed or are measurable by the dental clinician.

1. Signs of periodontal disease include gingival erythema (redness), gingival edema (swelling), bleeding on gentle probing, loss of attachment, tooth mobility, or loss of alveolar bone support.
2. Although some of these signs are easily detectable by the clinician (i.e., gingival erythema or gingival redness); other signs require careful assessment to identify (i.e., loss of attachment, furcation involvement).

b. **Symptoms of Periodontal Disease.** **Symptoms of periodontal disease** are features of a disease that are noticeable only to the patient.

1. For example, symptoms of Stage IV periodontitis might include loose teeth, difficulty in mastication (chewing), profuse gingival bleeding, or a bad taste in the mouth.
2. Note that in many patients the early symptoms of periodontitis are so subtle that they go undetected or unnoticed by the patient until the disease has progressed to such an advanced state that it causes serious complications that affect the survivability of the tooth. As such, some clinicians refer to periodontitis as a **silent disease** because of the lack of, or "silence," of early symptoms that are not obvious to the patient until the disease has progressed to cause considerable damage to the periodontium and leads to tooth loss.
3. Calling periodontitis a silent disease underscores the frequent observation that periodontitis can exist in patients who are totally unaware of its presence until the symptoms become more overt.

c. It is critical for members of the dental team to recognize that signs of inflammation in the periodontium include both **overt signs** of inflammation (i.e., those signs that are readily visible) and **hidden signs** of inflammation (i.e., those signs that are not readily visible) (**Table 21-1**).

Table 21-1 Signs of Inflammation in the Periodontium

Overt (Readily Visible) Signs	Hidden Signs
Color changes in the gingiva Contour changes in the gingiva Changes in consistency in the gingiva	Bone loss (*for periodontitis only*) Purulence (exudate) Bleeding on probing

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1. Examples of overt signs of inflammation are changes in the color, contour, and consistency of the gingival tissue.
2. Examples of hidden signs of inflammation are alveolar bone loss, bleeding on probing, and sometimes purulence or exudate.

2. Health as an answer to the first diagnostic question.
   a. If the clinical periodontal assessment reveals an absence of overt or hidden signs of inflammation in the periodontium, then the answer to the first diagnostic question is periodontal health (i.e., a periodontium free of inflammation).
   b. This means that inflammatory disease is not present and, though other problems in the periodontium may well be present (gingival recession caused by non-periodontitis reasons), the patient certainly does not have either gingivitis or periodontitis.
3. Inflammatory disease as an answer to the first diagnostic question.
   a. If the clinical periodontal assessment reveals the presence of either overt or hidden signs of inflammation in the periodontium, then the answer to the first diagnostic question is, of course, inflammatory disease.
   b. This means that some type of inflammatory disease is present (i.e., some type of gingivitis or periodontitis) and that further diagnostic decisions related to this inflammatory disease will need to be made by the team.

4. Additional diagnostic measures.
    a. Note that even in the absence of any inflammatory disease, some patients will require further examination to aid in the diagnostic decision-making process.
    b. For example, a patient with no periodontal inflammation but with severe gingival recession accompanied by cervical abrasion of the teeth may need to be evaluated for possible history of traumatic tooth brushing.

b. **Second Fundamental Diagnostic Question: "If the clinical assessment indicates inflammatory disease, is the disease gingivitis or is it periodontitis?"**
    1. **Using Attachment Loss to Answer the Second Fundamental Diagnostic Question**
        a. The answer to this second fundamental question is based on the clinical evidence of attachment loss as determined from the findings recorded during the clinical assessment.
            1. The **natural level of the gingival attachment** to the tooth is slightly coronal to (or in a sense above) the level of the cementoenamel junction (CEJ).
            2. **Clinical attachment loss (CAL)**, or attachment loss, refers to migration of the junctional epithelium to a position apical to (or in a sense below) the level of the CEJ.
        b. **Gingivitis.** If the clinical assessment reveals no attachment loss in the presence of inflammation, then the answer to the second diagnostic question is gingivitis. The reader may refer to Chapter 5 to review the various categories and subcategories of gingivitis.
        c. **Periodontitis.** If the clinical assessment reveals attachment loss in the presence of inflammation, then the answer to the second diagnostic question is periodontitis. The reader may refer to Chapter 6 to review the staging and grading criteria for periodontitis.
    2. It is important for the dental team to use dental radiographs as well as the clinical findings during the clinical assessment process.
        a. In most patients with periodontitis, alveolar bone loss will be evident on the radiographs.
        b. However, even before there is radiographic evidence of alveolar bone loss, clinical attachment loss should be apparent and detectable by an alert clinician.
        c. The members of the dental team must make every effort to detect periodontitis before there is obvious radiographic evidence of alveolar bone loss.

c. **Third Fundamental Diagnostic Question**: **"If the patient has gingivitis, what type of gingivitis?" or "If the patient has periodontitis, what type of periodontitis?"**
    1. The classification of various types of gingival diseases/conditions and the various types of periodontal diseases are covered in previous chapters of this textbook (Chapters 3 to 9).
    2. The dentist will use these disease classifications to assign a specific periodontal diagnosis based on the clinical features outlined in those chapters.

d. **Classification of Peri-Implant Diseases and Conditions**
    1. One of the key features to the 2017 classification system is the introduction of the categories of peri-implant health, peri-implant mucositis, and peri-implantitis.
    2. Determination of a proper peri-implant classification can be simplified by asking and answering two fundamental clinical questions in a systematic manner (**Figure 21-2**).

2. **Recognizing the Need for Flexibility When Assigning a Periodontal/Peri-Implant Diagnosis**
    a. **Flexibility.** The members of the dental team must be aware of the need for some flexibility when assigning a periodontal diagnosis and peri-implant diagnosis (when applicable) because a patient may have more than one periodontal condition or peri-implant condition, if applicable.
        1. Flexibility refers to a realization that the important fundamental decision as to the presence of either gingivitis or periodontitis may not describe the overall periodontal condition of the patient.
        2. Other periodontal conditions may indeed be present, and this finding must be noted when documenting a periodontal diagnosis in addition to any fundamental decision about the presence of gingivitis or periodontitis.
    b. **Other Findings.** Examples of some of these other periodontal conditions might be recession of the gingival margin, occlusal trauma, or aberrant frenum position.

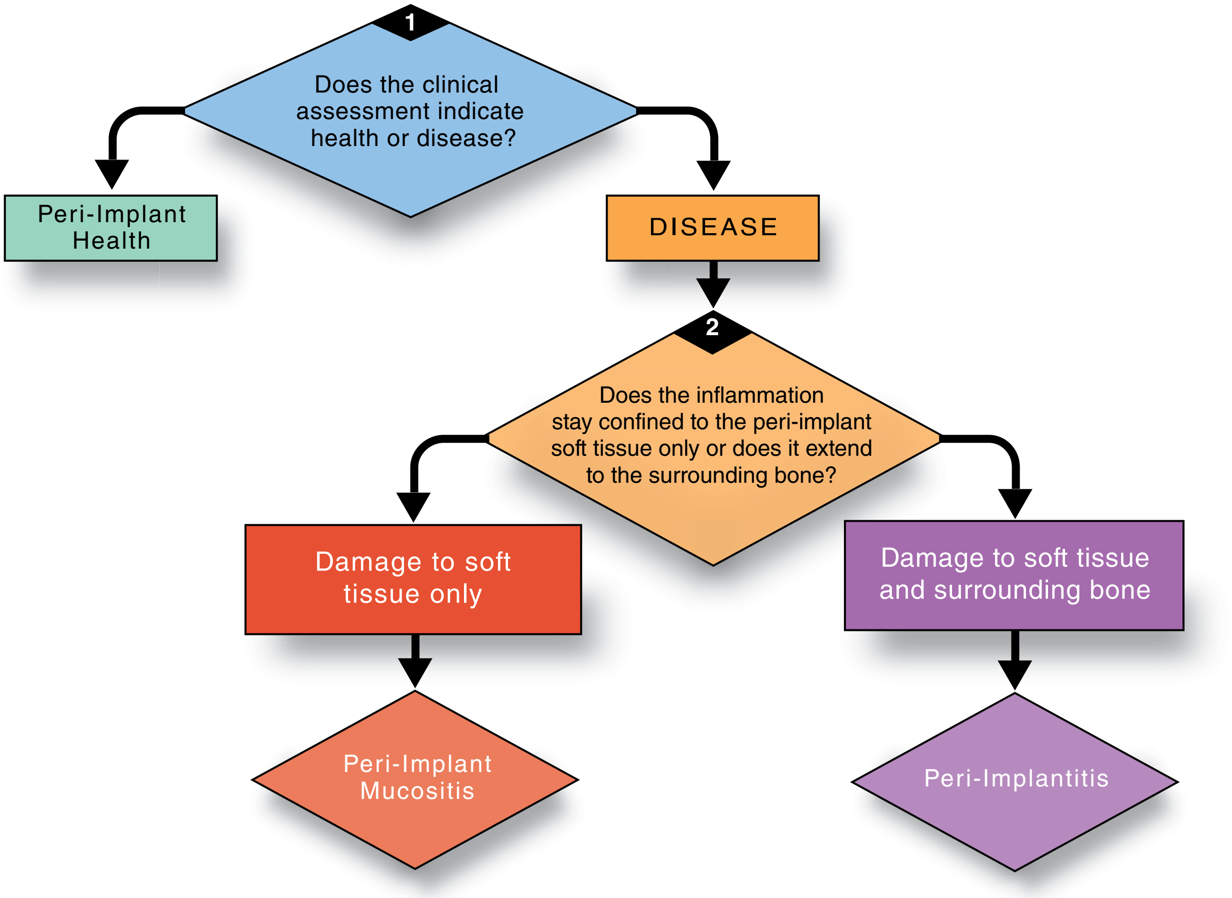
1
Does the clinical assessment indicate health or disease?
Peri-Implant Health
DISEASE
2
Does the inflammation stay confined to the peri-implant soft tissue only or does it extend to the surrounding bone?
Damage to soft tissue only
Damage to soft tissue and surrounding bone
Peri-Implant Mucositis
Peri-Implantitis

Figure 21-2 Decision Tree for Determining a Peri-Implant Diagnosis. A decision tree illustrating fundamental questions for determining an initial peri-implant diagnosis.

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3. **Thorough Documentation of the Periodontal/Peri-Implant Diagnosis.** Documenting the periodontal diagnosis is a critical skill for the dental team, and adhering to a standard format for such documentation is helpful. The same applies for the peri-implant diagnosis if the patient has a dental implant(s). The following are some guidelines for documenting periodontal diagnoses and, when applicable, peri-implant diagnoses.
   a. **Diagnostic Term.** As part of the diagnosis, the clinician should include the correct diagnostic term as outlined in the classification scheme, such as gingivitis or periodontitis or, when applicable, peri-implant health, peri-implant mucositis, or peri-implantitis (as discussed in Chapters 3–9).
   b. **Disease Stage.** When assigning a periodontitis diagnosis, the clinician should classify the disease using a multidimensional staging system. Staging of periodontitis defines the severity (according to the level of interdental clinical attachment loss, radiographic bone loss, and tooth loss), complexity of management, and extent and distribution of the disease. When assigning a peri-implant diagnosis, the clinician does not assign an implant stage or an implant grade. Rather the clinician, solely determines if the implant diagnosis is peri-implant health, peri-implant mucositis, or peri-implantitis.
      1. **Table 21-2** shows how the terms Stage I (initial periodontitis), Stage II (moderate periodontitis), Stage III (severe periodontitis with potential for tooth loss), and Stage IV (advanced periodontitis with extensive tooth loss and potential for loss of dentition) should be used as a part of the documentation of a periodontitis diagnosis.

Table 21-2 Use of Modifiers in Documenting Disease Stage, Grade, and Extent

	Descriptive Modifier	Definition
Disease stage	Stage I Stage II Stage III Stage IV	Initial periodontitis Moderate periodontitis Severe periodontitis with potential for tooth loss Advanced periodontitis with extensive tooth loss and potential for loss of dentition
Disease grade	Grade A Grade B Grade C	Slow rate of anticipated disease progression Moderate rate of anticipated disease progression Rapid rate of anticipated disease progression
Disease extent	Localized Generalized Molar/incisor pattern	30% or less of the teeth in the mouth are involved More than 30% of the teeth in the mouth are involved Only molars and incisors exhibit periodontal breakdown

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2. Note that the term *clinical attachment loss* (CAL) is used to underscore that these measurements are for sites that have lost attachment due to disease, and will not coincide exactly with precise histologic measurements. As explained in Chapter 19, the clinician should not confuse this concept with *clinical attachment level*.

c. **Disease Grading.** In addition to using the multidimensional staging system, the clinician should classify the disease using a multidimensional grading system. Grading should be used as an indicator of the anticipated rate of periodontitis progression.
   1. Table 21-2 shows how the terms *Grade A* (slow rate of periodontitis progression), *Grade B* (moderate rate of periodontitis progression), and *Grade C* (rapid rate of periodontitis progression) should be used as part of documentation of a periodontitis diagnosis.
   2. The primary criteria for grading periodontitis are either direct or indirect evidence of progression. Whenever available, direct evidence based on longitudinal data should be used (i.e., comparing radiographic bone loss or CAL that has occurred at two different time periods). When direct evidence is not available, the clinician relies on an indirect estimation by calculating bone loss as a function of age at the most affected tooth or case presentation (radiographic bone loss [RBL] expressed as percentage of root length divided by the age of the subject, RBL/age).
   3. Note that both staging and grading only apply to the classification of periodontitis and should not be used to classify other periodontal diseases/conditions or peri-implant diseases/conditions detailed in the new classification system.

d. **Disease Extent.** When assigning a stage to the periodontitis, a descriptive modifier such as *localized pattern*, *generalized pattern*, or *molar/incisor pattern* of the disease should be added to describe the number and the distribution of teeth affected with detectable periodontal breakdown.
   1. Table 21-2 also shows how the terms *localized*, *generalized*, and *molar/incisor pattern* should be used as part of documentation of the extent of periodontitis.
   2. Examples of appropriate periodontal diagnoses might be generalized Stage I, Grade A periodontitis or localized Stage IV, Grade C periodontitis. **Table 21-3** provides some examples of well-written periodontal diagnoses.

Table 21-3 Examples of Well-Written Periodontal Diagnoses

Extent	Stage	Grade	Name of Disease
Localized	Stage I	Grade A	Periodontitis
Localized	Stage III	Grade C	Periodontitis
Generalized	Stage II	Grade B	Periodontitis
Generalized	Stage IV	Grade A	Periodontitis
Molar/incisor pattern	Stage I	Grade C	Periodontitis

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4. **Using the American Dental Association (ADA) Case Type System for Periodontal Patients**
    a. **Case Types**
        1. Although the case type system is somewhat limited in value, it may be useful to assign a periodontal case type to periodontal patients when reporting to insurance carriers or to third-party payers.
        2. Assigning a periodontal case type is included in the initial decision-making process in most dental offices and is included here as additional information related to assigning a periodontal diagnosis.
            a. Case Type I: Patients with gingivitis only.
            b. Case Type II: Patients with slight (mild) periodontitis.
            c. Case Type III: Patients with moderate periodontitis.
            d. Case Type IV: Patients with severe periodontitis.
    b. **Limited Value of the Case Type System.** The value of the case type system is very limited because the case type alone does not specify the precise periodontal disease classification.
        1. The case type system fails to specify the precise disease extent. For example, a Case Type III patient could either be a patient who has localized periodontitis, localized to one sextant of the mouth, or a patient with generalized periodontitis throughout the entire mouth.
        2. Furthermore, a patient with Case Type III does not provide any information regarding the staging of the condition. In other words, the case type system does not define any specific factors that may influence the complexity of controlling current disease, such as a deep vertical defect.
        3. The case type system does not give the clinician much flexibility in assigning periodontal diagnosis. For instance, a Case Type III implies that the patient has moderate periodontitis but may not reflect other types of periodontal conditions affecting the periodontium.
        4. It is important for all members of the dental team to use the formal periodontal diagnosis (e.g., generalized Stage I, Grade C periodontitis) when describing the periodontal status of a patient and to use the case type only as a supplemental description, if called for.

Section 2

Guidelines for Periodontal Treatment Sequencing

1. **The Periodontal Master Treatment Plan.** The **master treatment plan** is a sequential outline of the steps to be carried out by the dentist, the dental hygienist, and the patient to eliminate disease and restore a healthy periodontal environment. It can be thought of as a blueprint of all treatment strategies recommended to attain and maintain the long-term periodontal health of the patient and should involve all members of the health care team and the patient.
    a. The master treatment plan can be used to coordinate and to sequence all treatment and educational steps employed.
    b. Although some of the treatment included in the master treatment plan may not involve the dental hygienist directly, it is important that the hygienist understand how all phases of treatment contribute to the goal of restoring a healthy mouth.
2. **Understanding the Phases of Periodontal Treatment.** The master treatment plan can be sequenced into phases. An overview of the sequence of phases of treatment is presented in **Figure 21-3** as well as in the discussion below. Refer to **Table 21-4** for examples of components of each of the phases in the management of periodontal patients. Note that individual treatment phases may overlap one another, especially in the management of patients who need both restorative and periodontal therapy.
    a. **Assessment and Preliminary Therapy Phase**
        1. The **assessment and preliminary therapy phase** includes: (1) assessment of collected data and (2) urgent care for any acute conditions, such as emergency dental care. Details of the clinical periodontal assessment are discussed in Chapter 19, and acute periodontal emergencies are discussed in Chapter 9.
        2. This phase of care also has been referred to as emergency therapy by some authors.

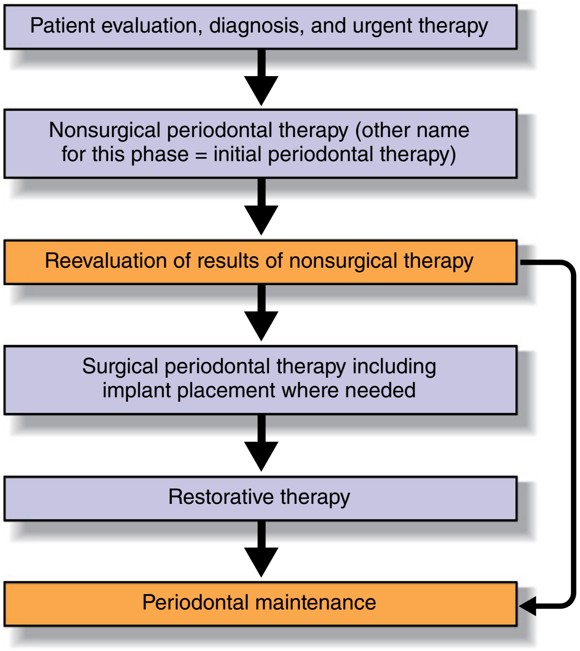
Patient evaluation, diagnosis, and urgent therapy
Nonsurgical periodontal therapy (other name for this phase = initial periodontal therapy)
Reevaluation of results of nonsurgical therapy
Surgical periodontal therapy including implant placement where needed
Restorative therapy
Periodontal maintenance

Figure 21-3 **Sequencing of Treatment Phases.** This flowchart provides an overview of the sequence of the normal phases of treatment for a patient. After periodontal instrumentation has been completed, reevaluation of results is typically done 4 to 6 weeks after initial therapy. The reevaluation allows the clinician to revisit the periodontium and reassess its response to initial therapy. If periodontitis has been successfully arrested, then a stable state has been achieved, and the patient can be enrolled in a periodontal maintenance program. If, however, periodontitis remains active in spite of initial therapy, then the patient should be referred to a specialist to initiate surgical periodontal therapy. Note that for healthy patients or gingivitis patients with a reduced periodontium, the reevaluation is done at each recall visit.

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Table 21-4 **Examples of Measures and Procedures Performed at Each Phase of Therapy**

Phase	Measures and Procedures
Assessment phase and preliminary therapy	Health history Comprehensive oral examination Assessment data collection Radiographs as indicated Diagnosis of oral conditions Treatment of urgent conditions Planning of nonsurgical therapy Referral for care of medical conditions Extraction of hopeless teeth
Nonsurgical periodontal therapy	Self-care education Nutritional counseling Nicotine cessation counseling Periodontal instrumentation Antimicrobial therapy Correction of local risk factors Fluoride therapy Caries control and temporary restorations Occlusal therapy Minor orthodontic treatment Reevaluation of Phase I therapy

Phase	Measures and Procedures
Surgical therapy	Periodontal surgery Endodontic surgery Dental implant placement
Restorative therapy	Dental restorations, fixed and removable prostheses Reevaluation of overall response to treatment
Periodontal maintenance	Ongoing care at specified intervals

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- b. **Nonsurgical Periodontal Therapy Phase**
  1. The **nonsurgical periodontal therapy phase** of treatment includes all the nonsurgical measures used to control gingivitis and periodontitis.
     - a. This phase includes intensive nonsurgical periodontal instrumentation and comprehensive patient educational measures.
     - b. This phase can also include steps to minimize the impact of local contributing factors.
  2. The nonsurgical periodontal therapy phase has also been called initial periodontal therapy, Phase I therapy, bacterial control, and anti-infective therapy.
- c. **Surgical Therapy Phase**
  1. The **surgical therapy phase** of treatment includes any necessary periodontal surgery to control or eliminate periodontal disease that cannot be managed by nonsurgical therapy alone. The surgical phase also includes the placement of dental implants to replace missing teeth and reestablish form and function of the oral dentition.
  2. The surgical therapy phase of care has also been called Phase II therapy. Periodontal surgical procedures are discussed in Chapter 30.
  3. If the periodontal health can be reestablished solely by nonsurgical periodontal therapy, then this phase of treatment is not indicated for the patient.
- d. **Restorative Therapy Phase**
  1. The **restorative therapy phase** of treatment may include placement of dental restorations and replacement of missing teeth by fixed or removable prostheses.
  2. The restorative therapy phase of care has also been called Phase III therapy.
- e. **Periodontal Maintenance Phase**
  1. The **periodontal maintenance phase** of treatment includes all measures used by the dental team and the patient to keep periodontitis from recurring once the inflammatory disease is brought under control.
  2. The objective of the periodontal maintenance phase is to maintain the teeth functioning throughout the life of the patient and may be needed for the rest of the patient's life.
  3. The periodontal maintenance phase of care has also been called Phase IV therapy. Periodontal maintenance is discussed in detail in Chapter 31.

Section 3

The Need for Ongoing Decision-Making

Because most patients are monitored for many years or even decades, clinical decision-making is an ongoing, evolving process. The aim of periodontal therapy is to preserve the periodontium, optimize oral health, and contribute to the general well-being of the individual.

In addition, the periodontium is composed of dynamic and continuously changing tissues (even in the absence of disease) that are subject to physiologic tissue remodeling or pathologic periodontal destruction throughout the lifetime of the tooth. Therefore, an individual's plan for periodontal care frequently requires adjustment over time because of these changes.

The dental team must be aware that a perfectly sensible periodontal diagnosis and plan for therapy at one point in time may require modification at a later date, and this fact must be continuously communicated to the patient at each visit.

Chapter Summary Statement

This chapter outlines guidelines that can be helpful to the members of the dental team during the management of patients with periodontal diseases. These guidelines include those related to formulating a periodontal diagnosis, sequencing periodontal treatment, and staying flexible to the changing needs of the individual.

Section 4

Focus on Patients

Clinical Patient Care

CASE 1

Periodontal assessment of a new patient reveals generalized bleeding on probing but very little gingival erythema (redness) and very little gingival edema (swelling). How would you answer the first fundamental diagnostic question for this patient?

CASE 2

Periodontal assessment of a new patient reveals definite signs of inflammation in the periodontium. Explain how to answer the second diagnostic question for this patient.

CASE 3

Periodontal assessment of a new patient reveals localized signs of gingival inflammation but no attachment loss. The findings also include a site of gingival recession and toothbrush abrasion on the facial surface of a canine tooth. At this site of recession, there is no sign of inflammation of the gingiva. How should this site of gingival recession due to traumatic brushing affect the basic diagnostic questions?

CASE 4

Following thorough clinical assessment of the periodontal condition of a patient, you are convinced that the patient has periodontitis. Explain how you would determine the severity of the periodontitis.

Suggested Readings

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CHAPTER 22

Shared Decision-Making for Periodontal Care

- Section 1 Shared Decision-Making
- Section 2 A Model for Shared Decision-Making in Periodontal Care
  - The SHARE Approach: Essential Steps of Shared Decision-Making
- Section 3 Decision Aids for Periodontal Patients
- Section 4 Guidelines Related to Consent for Periodontal Treatment
- Section 5 Focus on Patients
  - Ethical Scenarios

CLINICAL APPLICATION

Periodontal treatment is always based upon the best available scientific evidence. Oftentimes, however, there will be more than a single course of treatment that could benefit a patient. To be an active participant in the clinical decision-making process, a patient needs information about his or her oral condition and all the possible treatment options. Dental health care providers must be able to work collaboratively and communicate effectively with the patient. Well-informed patients tend to have more realistic expectations of periodontal treatment outcomes and their role in maintaining periodontal health. This chapter discusses shared decision-making, decision aids, and informed consent.

LEARNING OBJECTIVES

- Define *shared decision-making*.
- Explain key steps for engaging in shared decision-making.
- Describe how patient decision aids facilitate shared decision-making.
- Describe the importance of informed consent to successful periodontal care.
- List guidelines for obtaining informed consent.
- Describe two formats for documenting informed consent.

KEY TERMS

Shared decision-making
Evidence-based health care
Systematic review
Cochrane Database of Systemic Reviews
SHARE Approach
Patient decision aids
Informed consent
Reasonable patient standard
Informed refusal

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Section 1

Shared Decision-Making

In the past, medical and dental health care providers took responsibility not only for being well informed about the risks and benefits of all treatment options, but also for judging their value in the best interests of their patients. More recently, a shared decision-making approach has been advocated in which the patient is recognized as a mutual expert for judging the value of treatments. Shared decision-making—as opposed to clinicians making decisions on behalf of patients—is gaining increasing importance in health care policy.[1–5] Shared decision-making has been called the "crux of patient-centered care" and is identified as a key part of change for improved quality in health care.[6]

1. **What is shared decision-making? Shared decision-making** is a collaborative process that recognizes a patient's right to make decisions about his or her care once fully informed of the treatment options. Periodontal treatment is always based upon the best available scientific evidence, but many times there will be more than a single course of treatment that could benefit an individual patient. Shared decision-making is a process in which clinicians and patients work together to formulate decisions that are best for the patient. The final decision rests on evidence-based information about periodontal care options, the clinician's knowledge and experience, and the patient's informed preferences. As depicted in **Figure 22-1**, shared decision-making involves agreement between the clinician and patient on the patient's care plan.

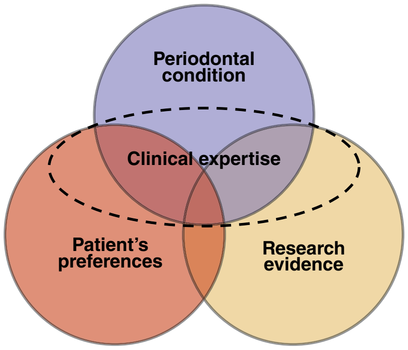

Figure 22-1 Shared Decision-Making Model for Evidence-Based Decisions. In shared decision-making, the patient's knowledge and preferences are considered, alongside the clinician's expertise, and the decisions they reach—in agreement with each other—are founded on valid evidence on effective treatment options.

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   a. **When is shared decision-making appropriate?** Shared decision-making is appropriate for any dental health decision where there is more than one reasonable option.
   b. **What is evidence-based information?**
      1. **Evidence-based health care** is a systematic approach to clinical problem solving that allows the integration of the best available research evidence with clinical expertise and patient values.[7]
      2. A **systematic review** summarizes the results of available carefully designed health care studies (controlled trials) and provides a high level of evidence on the effectiveness of health care interventions.
      3. *Cochrane Database of Systemic Reviews*
         a. Many dental clinicians do not have the expertise needed to conduct their own systematic reviews. Fortunately, there is a trustworthy resource—the *Cochrane Database of Systemic Reviews*—for busy practitioners who want to integrate high-quality science into patient care.
         b. Cochrane (previously known as the Cochrane Collaboration) is an independent, nonprofit, nongovernmental organization consisting of a group of volunteers from more than 130 countries. It is the worldwide leader in collecting high-quality, independent systematic reviews of scientific research that facilitates the choices that health professionals, patients, policy makers, and others face in health care interventions according to the principles of evidence-based care.

2. **Characteristics of Shared Decision-Making.** A key characteristic of shared decision-making on the dental health care provider's part is the conscientious offering of care options for the patient's consideration.[8] In addition to the patient, shared decision-making may involve a team of health professionals, involve significant others (partners, family members, guardians, caregivers), and differ across cultural, social, and age groups. As illustrated in **Figure 22-2**, a dental health care provider should be able to inform patients and engage them in shared decision-making by being able to do the following[9,10]:

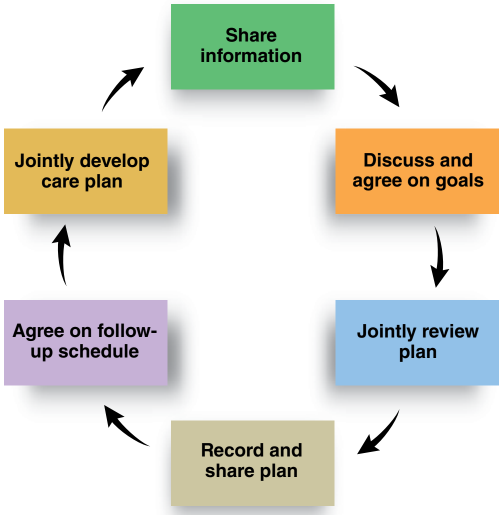
Share information
Discuss and agree on goals
Jointly review plan
Record and share plan
Agree on follow-up schedule
Jointly develop care plan

Figure 22-2 Shared Care Planning. A depiction of the steps in shared care planning.

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   a. Develop a partnership with a patient.
   b. Establish the patient's preferences for his or her role in decision-making (degree of involvement of self and others, such as family members).
   c. Elicit and respond to the patient's ideas, concerns, and expectations of periodontal therapy.
   d. Identify choices and evaluate the research evidence in relation to the individual patient.
   e. Help the patient reflect on and assess the impact of alternative decisions with regard to his or her values.
   f. Develop a periodontal care plan in partnership with the patient.
   g. Document the agreed-upon periodontal care plan, and complete arrangements for follow-up.
3. **A Mutual Decision: Why Patients' Preferences Matter**
   a. **An Ethical Imperative**
      1. The most important reason for practicing shared decision-making is that it is the right thing to do. Conveying unbiased communication and easy-to-understand information on options, uncertainties, and risks and benefits is an ethical necessity. Failure to do so should be taken as evidence of poor-quality care.
      2. No treatment is 100% reliable, and there are very few clinical situations where there is just one course of action that should be followed in all cases.
         a. In many situations, there is no single "right" care decision because choices about periodontal care come with pros and cons. There usually is a choice between undergoing a procedure or not, or—more commonly—a choice between different options.
         b. In circumstances where there are several options leading to different outcomes, the "right" decision depends on a patient's own unique set of needs, expectations, and outcome goals.[11] The decision might be whether to:
            1. Undergo a diagnostic procedure such as a full series of radiographs.
            2. Undergo a periodontal surgical procedure.
            3. Increase frequency of recall/maintenance appointments.
            4. Take a medication.
            5. Undertake a lifestyle change such as quitting smoking.

b. **Two Sources of Expertise.** At the heart of shared decision-making is the recognition that clinicians and patients bring different but equally important experiences, attitudes, perceptions, and expectations to the decision-making process (**Table 22-1**). Shared decision-making is appropriate where there is more than one reasonable option.
   1. The clinician is an expert in dental care, such as knowledge of the diagnosis, likely course of the disease, treatment options, and the range of possible outcomes.
   2. The patient is an expert on his or her own life, circumstances, and what matters most to him or her. The patient knows about the impact of the condition on his or her daily life, personal attitude toward risk, values, and preferences. For example, referring to Table 22-1, the dental health care provider may suggest counseling for smoking cessation. The patient's perspective might be as follows:

Table 22-1 **Sharing Expertise**

Clinician's Expertise	Patient's Expertise
Diagnosis of condition	Experience of condition
Disease etiology	Circumstances (finances, family values)
Prognosis	Attitude to risk
Treatment options	Values
Outcome probabilities	Preferences

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      a. Patient's experience of condition: aware of coughing in morning, but not concerned about the cough.
      b. Patient's social circumstances: immediate family members all smoke.
      c. Patient's attitude to risk: feels that smoking does not present a health risk because her grandfather smoked heavily and lived to be 90 years of age.
      d. Patient's values: enjoys smoking, especially in social situations and believes smoking helps to control her weight.
      e. Patient's preferences: is not interested in quitting.

c. **Improved Treatment Outcomes**
   1. Research shows that patients frequently do not understand recommended treatment options or risks and benefits. A Cochrane review reported consistent evidence that as patients become better informed, they make better informed decisions and feel more confident about their choices.[12]
   2. However, evidence suggests that clinicians do not fully grasp the treatment outcomes that patients prefer (**Figure 22-3**). Several studies document substantial gaps between the outcomes patients prefer and the outcomes clinicians perceive patients prefer.[13–16]

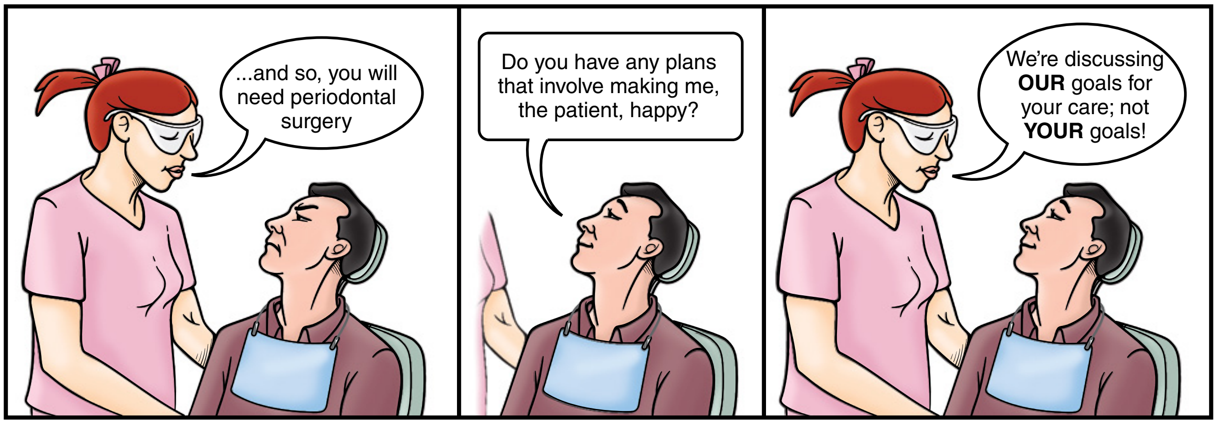
...and so, you will need periodontal surgery
Do you have any plans that involve making me, the patient, happy?
We're discussing **OUR** goals for your care; not **YOUR** goals!

Figure 22-3 **Treatment Outcomes.** Evidence suggests that the outcomes that clinicians prefer may differ from those of their patients.

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3. **Figure 22-4** depicts the problems created by lack of information by patients and misconceptions of clinicians that result in poor decision quality. Shared decision-making helps the patient understand what the provider is trying to do (the goals of treatment).

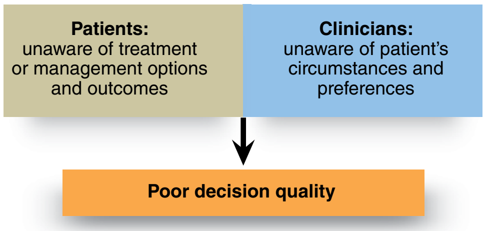
Patients: unaware of treatment or management options and outcomes
Clinicians: unaware of patient's circumstances and preferences
Poor decision quality

Figure 22-4 The Clinical Decision Problem. Less than ideal care results when patients do not understand the proposed treatment and clinicians are unaware of the patient's circumstances and preferences.

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4. When patients participate in decision-making and understand what they need to do, they are more likely to follow through. Shared decisions can result in a course of action for which the patient understands the need to take responsibility (e.g., meticulous daily self-care for biofilm control). In this case, patients are more likely to make behavioral changes if they have made the decision for themselves.
5. Shared decision-making facilitates a trusting relationship between the clinician and patient and increases patient and provider satisfaction.

Section 2

A Model for Shared Decision-Making in Periodontal Care

The concept of engaging patients in decision-making to improve the quality of health care is firmly grounded in the 2001 Institute of Medicine (now known as the National Academy of Medicine) report: *Crossing the Quality Chasm*.[17] This report defines patient-centered care as "care that is respectful of and responsive to individual patient preferences, needs, and values" and that ensures "patient values guide all clinical decisions."

Several studies suggest that many health care professionals believe patients are not interested in taking an active role in care planning.[18–21] Evidence suggests, however, that many patients would prefer to be given more information and would like to be involved in decision-making about their health care.[19,20]

The SHARE Approach: Essential Steps of Shared Decision-Making

The U.S. Department of Health and Human Services, Agency for Healthcare Research and Quality (AHRQ) developed a model to support the training of health care professionals on how to engage patients in their health care decision-making. AHRQ's **SHARE Approach** is a five-step process—Seek, Help, Assess, Reach, Evaluate—for shared decision-making that includes exploring the benefits and risks of each care/treatment option through meaningful discussion about what matters most to the patient.[22] The mnemonic SHARE is a learning device to assist in remembering the five steps (**Figure 22-5**).

The five steps are intended to serve as prompts to help health care providers ensure that they are engaging patients in a meaningful discussion of treatment options. The dialogue is an individualized process with each individual patient and some steps may overlap with one another. The SHARE Approach usually begins with inviting the patient to play an active role in the decision-making process.

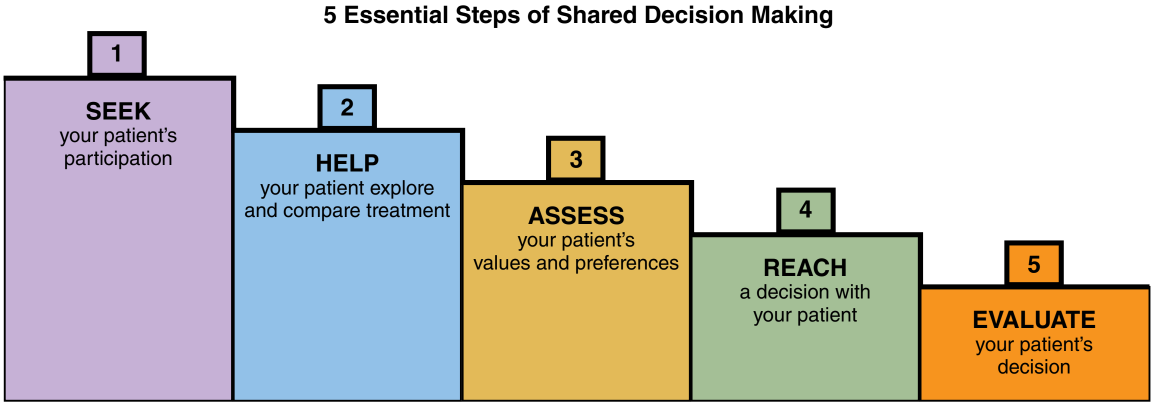
5 Essential Steps of Shared Decision Making
1
SEEK
your patient's participation
2
HELP
your patient explore and compare treatment
3
ASSESS
your patient's values and preferences
4
REACH
a decision with your patient
5
EVALUATE
your patient's decision

Figure 22-5 **The SHARE Approach.** The five essential steps of the SHARE decision-making model developed by the U.S. Department of Health and Human Services, Agency for Healthcare Research and Quality.

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Step 1: Seek Your Patient's Participation

Many patients are not aware that they can participate in health care decisions or that there may be different care options. Some patients may not want—or be ready—to participate in care decisions.[20,21] Clinicians should respect a patient's preference not to participate in the decision-making process or to delegate the decision to a family member or caregiver.

- Summarize the patient's periodontal condition. Describe the problem clearly.
- Ask the patient to participate. Help the patient understand that he or she is being invited to ask questions and discuss options. Inviting patients to participate lets them know that they have options and that their goals and concerns are a key part of the decision-making process.
- If appropriate, include family members and/or caregivers in discussions. Ask if the patient would like to have family members and/or caregivers participate in the discussion.
- Remind the patient that his or her participation is important. For example, say, "What is your next question?" or "I would like your input." **Box 22-1** provides several examples of conversation starters to facilitate patient participation.

Box 22-1 Conversation Starters for Step 1

"Now that you understand the problem with the tissues that support your teeth, it is time for us to think about what to do next."

"I want to go over all the options so we can decide together what is best for you."

"There is good information about how these treatment outcomes differ that I'd like to discuss with you before we decide on an approach that is best for you."

If the patient wants the clinician to make the decision for them, the following might be useful to try: "I am happy to share my views and help you make a good decision. Before I do, would you like more details about your options?"

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Step 2: Help Your Patient Explore and Compare Treatment Options

Letting the patient know that there are several options for home care or treatment is helpful in building a trusting patient–clinician relationship. Clinicians should guide patients through the benefits and risks of each option. **Box 22-2** provides several examples of conversation starters to facilitate patient participation.

- Describe each option in plain language. Avoid using technical or dental jargon (e.g., say, "both sides" instead of "bilateral").
- Clearly communicate the benefits and risks of all options.
- Offer evidence-based decision aid tools whenever possible. (Decision aids are discussed in Section 3 of this chapter.)
- After presenting information about options, make sure that the patient understands the information. Ask the patient to summarize the options in his or her own words.

Box 22-2 Conversation Starters for Step 2

"Let me list the options for removing plaque biofilm from between your teeth before we get into more detail about them."
"Here are some choices we can consider. Treatments have different consequences, some will matter more to you than they do to other people."
"These options will have different implications for the health status of your mouth. Let me tell you what the research says about the benefits and risks of these two treatment options."
"The options that I just described are not always effective for everyone."
"We've talked about your treatment options. To make sure I've explained things well, would you tell me how they're different?"

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Step 3: Assess Your Patient's Values and Preferences

Several studies document substantial gaps between the outcomes patients prefer and the outcomes health care providers think patients prefer.[21,23] Additionally, several researchers have shown that treatment decisions change after patients become well informed. An international Cochrane Review reports consistent evidence that as patients become better informed, they make different decisions and feel more confident in their decisions.[2,24,25] **Box 22-3** provides several examples of conversation starters to facilitate patient participation.

- Assist patients in evaluating options based on their goals and concerns. To understand patients' preferences, ask them what is important to them and what they are concerned about.
- Encourage the patient to talk about what matters most to him or her. For example, how important is it to the patient to relieve the symptoms he or she is experiencing? The patient might state that recovery time, cost, or having a certain level of functioning is the most important.
- Listen to the patient. Encourage the patient to continue talking. For example, "Go on," or "Tell me more about that."
- Show empathy and interest in the effect that a problem is having on the patient's life. For example, say, "That sounds really upsetting."
- Agree on what is important to your patient.

Box 22-3 Conversation Starters for Step 3

"As you think about your options, what matters most to you?"
"Is there anything that may get in the way of doing this?"
"Tell me more about your fear that you will lose your teeth no matter what you do."
"It sounds like you are upset that your previous dental hygienist did not tell you about the loss of bone support around your molar teeth."

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Step 4: Reach a Decision with Your Patient

Deciding may take time, especially if the treatment is irreversible, such as periodontal surgery. **Box 22-4** provides several examples of conversation starters to facilitate patient participation.

- Help the patient move to a decision. Ask if he or she is ready to decide or if he or she has additional questions.
- Confirm the decision with the patient.
- Assist patients to follow through on the decision. Lay out the next steps and the timing of these actions for patients, check for understanding, and discuss any possible challenges with carrying out the decision.
- Assist patients in managing barriers to implementing their decisions.

Box 22-4 Conversation Starters for Step 4

"Are you ready to decide?" "Do you have more questions?" "Are there more things that we should discuss to help you make your decision?"
"Now that we've discussed your treatment options, which treatment do you think is right for you?"

(continues)

Box 22-4 **Conversation Starters for Step 4** *(continued)*

"So, we have talked about two options for removing the plaque biofilm and calculus: a series of four shorter appointments treating one-fourth of your mouth at a time or a series of two longer appointments treating one-half of your mouth at a time. Would one of these options work better for you in taking time away from your office?"

"Now that I've shown you two options for removing plaque biofilm from between your teeth—an interdental brush or a water flossing device—which one do you think will work best for you at home?"

"We discussed two options—surgery or frequent professional care with the hygienist. There is good evidence about how these treatment options differ that I'd like to discuss with you before we decide on an option that is best for you."

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Step 5: Evaluate Your Patient's Decision

Once a decision has been made, it is important to follow-up with the patient on how he or she is doing. Because periodontal disease is a chronic condition, treatment decisions should be revisited at frequent intervals. **Box 22-5** provides several examples of conversation starters to facilitate patient participation.

- Make plans to review the decision in the future. Remind the patient that decisions may be revised and revisited in the future, and some may be changed if they are not working well. For example, a patient may decide against having periodontal surgery—at first—but may reconsider that decision at the next maintenance appointment.
- Monitor the extent to which the treatment decision is implemented. For example, the patient may decide to use an interdental brush for biofilm control, however, discover that he finds the brush too time-consuming to use.
- Revisit the decision with your patient to determine if other decisions need to be made.

Box 22-5 **Conversation Starters for Step 5**

"Can we talk at your next appointment in 3 months to see how you are doing?"
"Let's review how things are going for you in 3 months."
"If you feel things are not improving, please call immediately so we can plan a different approach."
"If you find that you do not like using this interdental brush, we have other options."

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Section 3

Decision Aids for Periodontal Patients

Often there will be more than a single option for periodontal treatment that could benefit an individual patient. Patients need clear, comprehensive information about periodontal conditions and treatment or maintenance options to play an effective role in the decision-making process. Clear and consistent research shows that most patients want to be informed and involved in decisions about their care.[26]

Patient decision aids are the best known and proven tools to support shared decision-making.[12] **Patient decision aids** are information resources such as written materials, videos, or web-based tools that are designed to facilitate shared decision-making and patient participation in health care decisions. Patient decision aids provide an evidence-based guide for patients: they present the decision to be taken, the options available, and the associated outcomes (including benefits, harms, and uncertainties).[27] Findings show that when patients use decision aids they (1) expand their knowledge of options, (2) feel more informed and clearer about what matters most to them, (3) have more accurate expectations of possible benefits and harms, and (4) participate more in decision-making.[12] Patient decision aids are different from more traditional patient information materials because they do not tell the patient what to do. Instead, they set out the facts and help the patient carefully consider all the options. **Box 22-6** provides an example of a conversation starter for introducing a patient decision aid. **Box 22-7** summarizes the information that patient decision aids usually contain. **Figures 22-6 and 22-7** provide two examples of patient decision aids.

Box 22-6 **Conversation Starters When Introducing a Decision Aid**

"This decision aid is designed to help you understand your options in more detail."

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Box 22-7 Typical Information for Patient Decision Aids

- A description of the condition and symptoms (e.g., gingivitis)
- The likely prognosis (disease progression) with and without treatment
- The treatment and self-management options and outcome probabilities
- What's known from the evidence and not known (uncertainties)
- The most frequent complications of the treatment options

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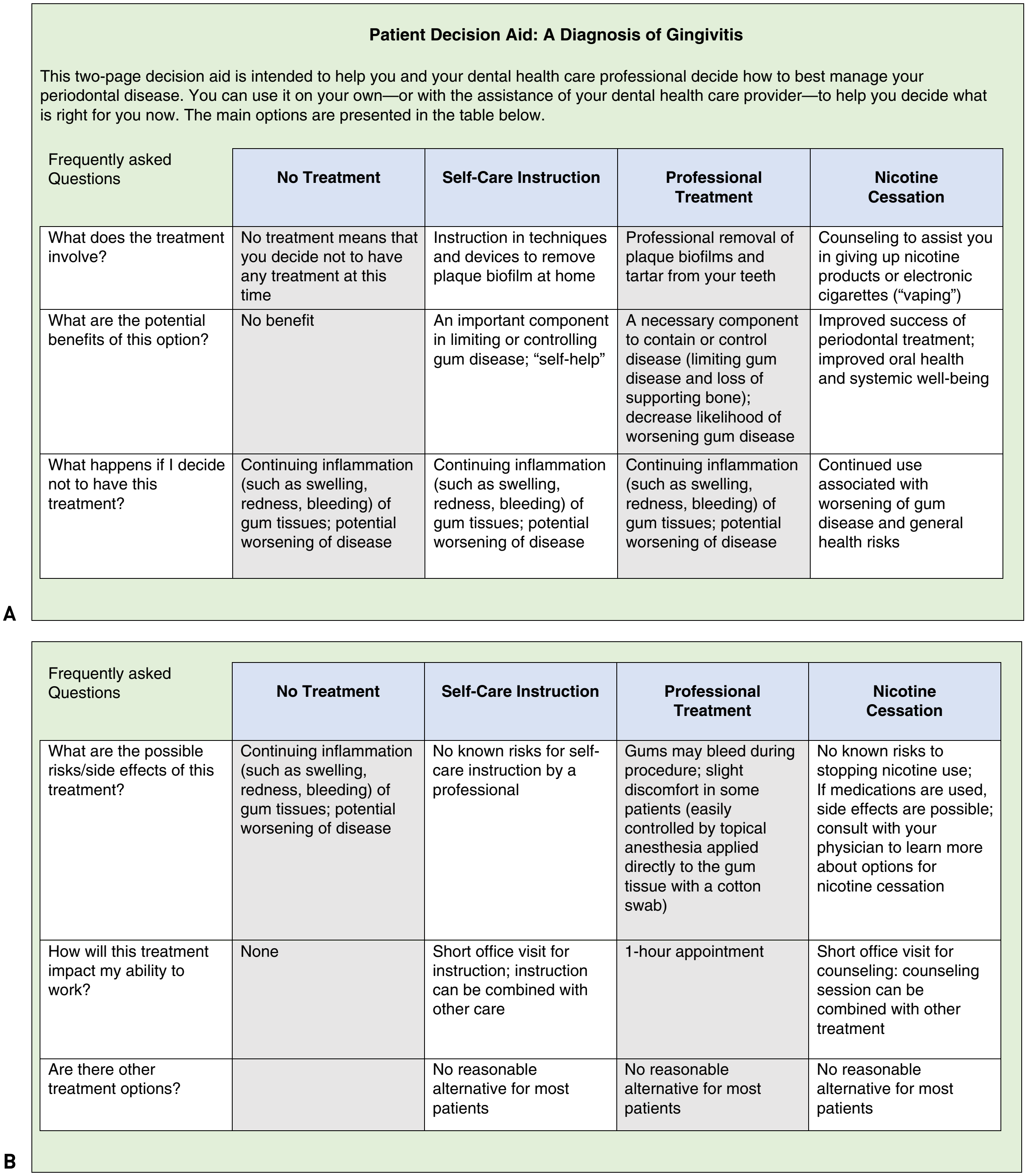

Patient Decision Aid: A Diagnosis of Gingivitis

This two-page decision aid is intended to help you and your dental health care professional decide how to best manage your periodontal disease. You can use it on your own—or with the assistance of your dental health care provider—to help you decide what is right for you now. The main options are presented in the table below.

Frequently asked Questions	No Treatment	Self-Care Instruction	Professional Treatment	Nicotine Cessation
What does the treatment involve?	No treatment means that you decide not to have any treatment at this time	Instruction in techniques and devices to remove plaque biofilm at home	Professional removal of plaque biofilms and tartar from your teeth	Counseling to assist you in giving up nicotine products or electronic cigarettes ("vaping")
What are the potential benefits of this option?	No benefit	An important component in limiting or controlling gum disease; "self-help"	A necessary component to contain or control disease (limiting gum disease and loss of supporting bone); decrease likelihood of worsening gum disease	Improved success of periodontal treatment; improved oral health and systemic well-being
What happens if I decide not to have this treatment?	Continuing inflammation (such as swelling, redness, bleeding) of gum tissues; potential worsening of disease	Continuing inflammation (such as swelling, redness, bleeding) of gum tissues; potential worsening of disease	Continuing inflammation (such as swelling, redness, bleeding) of gum tissues; potential worsening of disease	Continued use associated with worsening of gum disease and general health risks

A

Frequently asked Questions	No Treatment	Self-Care Instruction	Professional Treatment	Nicotine Cessation
What are the possible risks/side effects of this treatment?	Continuing inflammation (such as swelling, redness, bleeding) of gum tissues; potential worsening of disease	No known risks for self-care instruction by a professional	Gums may bleed during procedure; slight discomfort in some patients (easily controlled by topical anesthesia applied directly to the gum tissue with a cotton swab)	No known risks to stopping nicotine use; If medications are used, side effects are possible; consult with your physician to learn more about options for nicotine cessation
How will this treatment impact my ability to work?	None	Short office visit for instruction; instruction can be combined with other care	1-hour appointment	Short office visit for counseling: counseling session can be combined with other treatment
Are there other treatment options?		No reasonable alternative for most patients	No reasonable alternative for most patients	No reasonable alternative for most patients

B

Figure 22-6 Patient Decision Aid for Diagnosis of Gingivitis. A. Page one of a sample patient decision aid for a diagnosis of gingivitis. **B.** Page two of a sample patient decision aid for a diagnosis of gingivitis.

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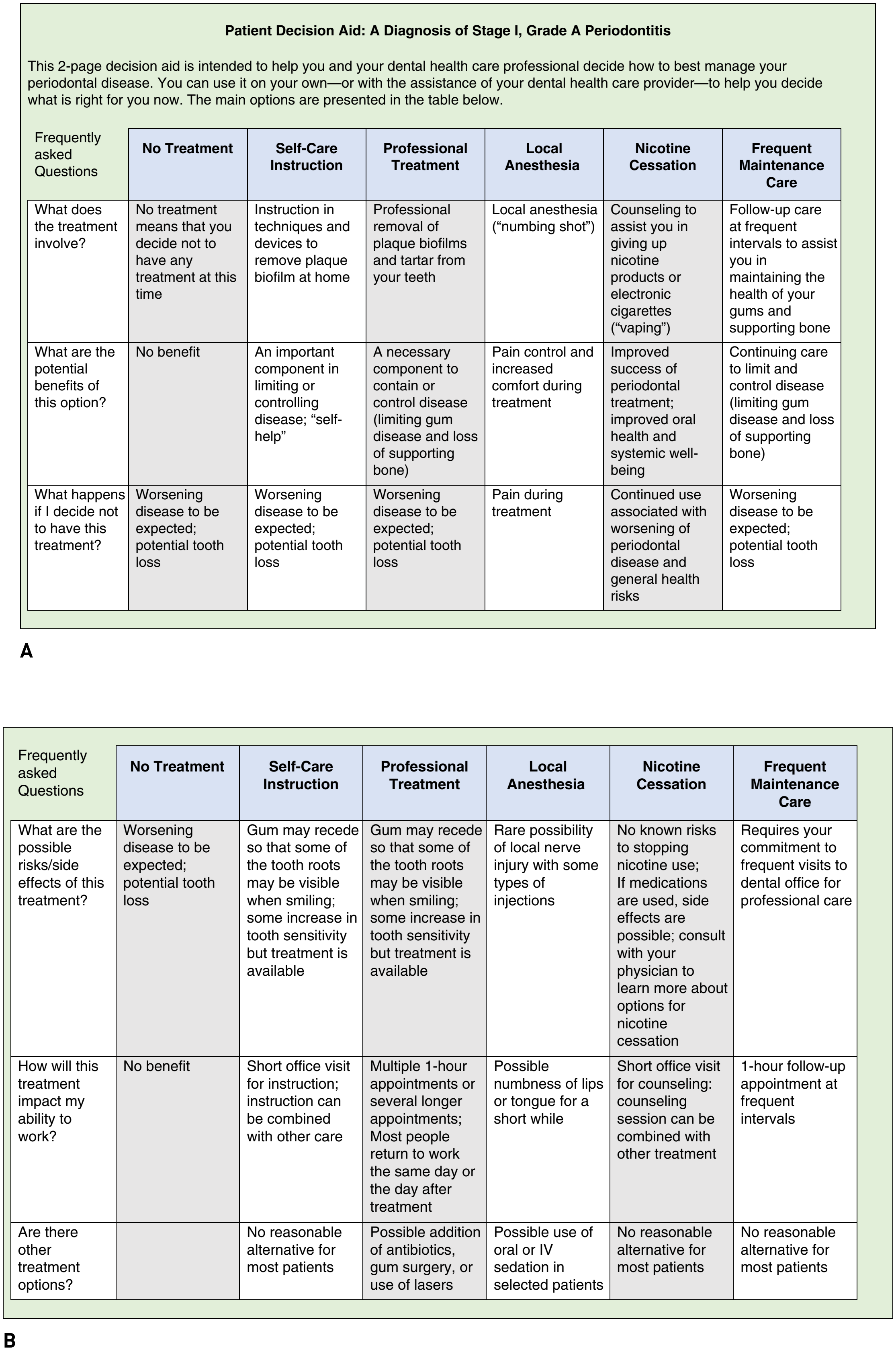

Patient Decision Aid: A Diagnosis of Stage I, Grade A Periodontitis

This 2-page decision aid is intended to help you and your dental health care professional decide how to best manage your periodontal disease. You can use it on your own—or with the assistance of your dental health care provider—to help you decide what is right for you now. The main options are presented in the table below.

Frequently asked Questions	No Treatment	Self-Care Instruction	Professional Treatment	Local Anesthesia	Nicotine Cessation	Frequent Maintenance Care
What does the treatment involve?	No treatment means that you decide not to have any treatment at this time	Instruction in techniques and devices to remove plaque biofilm at home	Professional removal of plaque biofilms and tartar from your teeth	Local anesthesia ("numbing shot")	Counseling to assist you in giving up nicotine products or electronic cigarettes ("vaping")	Follow-up care at frequent intervals to assist you in maintaining the health of your gums and supporting bone
What are the potential benefits of this option?	No benefit	An important component in limiting or controlling disease; "self-help"	A necessary component to contain or control disease (limiting gum disease and loss of supporting bone)	Pain control and increased comfort during treatment	Improved success of periodontal treatment; improved oral health and systemic well-being	Continuing care to limit and control disease (limiting gum disease and loss of supporting bone)
What happens if I decide not to have this treatment?	Worsening disease to be expected; potential tooth loss	Worsening disease to be expected; potential tooth loss	Worsening disease to be expected; potential tooth loss	Pain during treatment	Continued use associated with worsening of periodontal disease and general health risks	Worsening disease to be expected; potential tooth loss

A

Frequently asked Questions	No Treatment	Self-Care Instruction	Professional Treatment	Local Anesthesia	Nicotine Cessation	Frequent Maintenance Care
What are the possible risks/side effects of this treatment?	Worsening disease to be expected; potential tooth loss	Gum may recede so that some of the tooth roots may be visible when smiling; some increase in tooth sensitivity but treatment is available	Gum may recede so that some of the tooth roots may be visible when smiling; some increase in tooth sensitivity but treatment is available	Rare possibility of local nerve injury with some types of injections	No known risks to stopping nicotine use; If medications are used, side effects are possible; consult with your physician to learn more about options for nicotine cessation	Requires your commitment to frequent visits to dental office for professional care
How will this treatment impact my ability to work?	No benefit	Short office visit for instruction; instruction can be combined with other care	Multiple 1-hour appointments or several longer appointments; Most people return to work the same day or the day after treatment	Possible numbness of lips or tongue for a short while	Short office visit for counseling: counseling session can be combined with other treatment	1-hour follow-up appointment at frequent intervals
Are there other treatment options?		No reasonable alternative for most patients	Possible addition of antibiotics, gum surgery, or use of lasers	Possible use of oral or IV sedation in selected patients	No reasonable alternative for most patients	No reasonable alternative for most patients

B

Figure 22-7 Patient Decision Aid for Diagnosis of Stage I, Grade A Periodontitis. A. Page one of a sample patient decision aid for diagnosis of Stage I, Grade A periodontitis. **B.** Page two of a sample patient decision aid for diagnosis of Stage I, Grade A periodontitis.

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Section 4

Guidelines Related to Consent for Periodontal Treatment

According to the American Dental Hygienists' Association (ADHA) Code of Ethics, a patient has a right to informed consent prior to treatment, and they have the right to full disclosure of all relevant information so that they can make informed choices about their care.[1] This makes the patient a critical and willing participant in any plan involving periodontal therapy. As such, communication between the patient and dental practitioner is vital as the treatment plan develops.

Studies demonstrate that patients who believe that they have been well informed regarding their condition and who have had their questions answered by members of the dental team are more compliant with treatment recommendations, have more trust in their health care providers, and are more satisfied with their care. These factors lead to better treatment outcomes and reduced malpractice risk.

1. **Informed Consent for Periodontal Treatment.** **Informed consent** is a patient's voluntary agreement to proposed treatment. A patient can only give informed consent to treatment after achieving an understanding of the relevant facts, benefits, and risks involved.[2]
    a. **What Constitutes Informed Consent?**
        1. Informed consent is not the same thing as a consent form that a patient is asked to sign. Rather, informed consent is the process of communication between a patient and a health care provider. It is a process that allows the patient to make a knowledgeable decision about his or her own dental care.
        2. An individual's consent is informed only if the recommended treatment, alternate treatment options, and the benefits and risks of treatment have been thoroughly described to the person in language understood by the patient.[3]
        3. Informed consent must be voluntary, and this informed consent originates from (1) a person's legal right to direct what happens to his or her body and (2) the ethical duty of the dental health care provider to involve the individual in his or her own dental care.
2. **The Reasonable Patient and Informed Consent**
    a. **Legal Claims.** Legal claims against dental health care providers usually consist of three parts: the clinician did not perform the treatment as he or she should have done; the clinician did not inform the patient properly about the risks involved with treatment; and the clinician did not keep complete and accurate records.[28]
        1. When speaking about risks, judges tend to look at what a "reasonable patient" would want to know.[28]
        2. The **reasonable patient standard** says a health care provider must disclose all information that a rational patient would want before making a choice to pursue or reject a treatment or procedure.
        3. **Box 22-8** provides an example of the information a reasonable patient would need regarding local anesthesia prior to periodontal surgery.

Box 22-8 What Would a Reasonable Patient Need to Know?

Hypothetical Scenario: A clinician plans periodontal treatment in the mandible. What information should he provide to the patient about the risks of local anesthesia?

- **What are the risks?** There is a possibility of injuring a nerve in the jaw. Nerve injury is not a life-threatening risk, but it can be annoying. The risk of injuring the lingual nerve while giving local anesthesia is very low. A recent study found that injuring the lingual nerve while giving local anesthesia occurred in 1 out of 2,667 cases.[29]
- **Are there alternative options?** The anesthesia could be given around the teeth or gum (directly into the periodontal ligament or gingiva). The risk of nerve damage is much lower and the quality of anesthesia will be good. However, this technique does not provide profound local anesthesia and may be of shorter duration compared to a conventional mandibular block. Furthermore, this technique may require multiple injections in several papillae to adequately anesthetize the periodontal tissues in the mandible.
- **What if no anesthesia is used?** Periodontal treatment may be painful without anesthesia. Without periodontal treatment, the likely outcome is unfavorable and tooth loss is probable.

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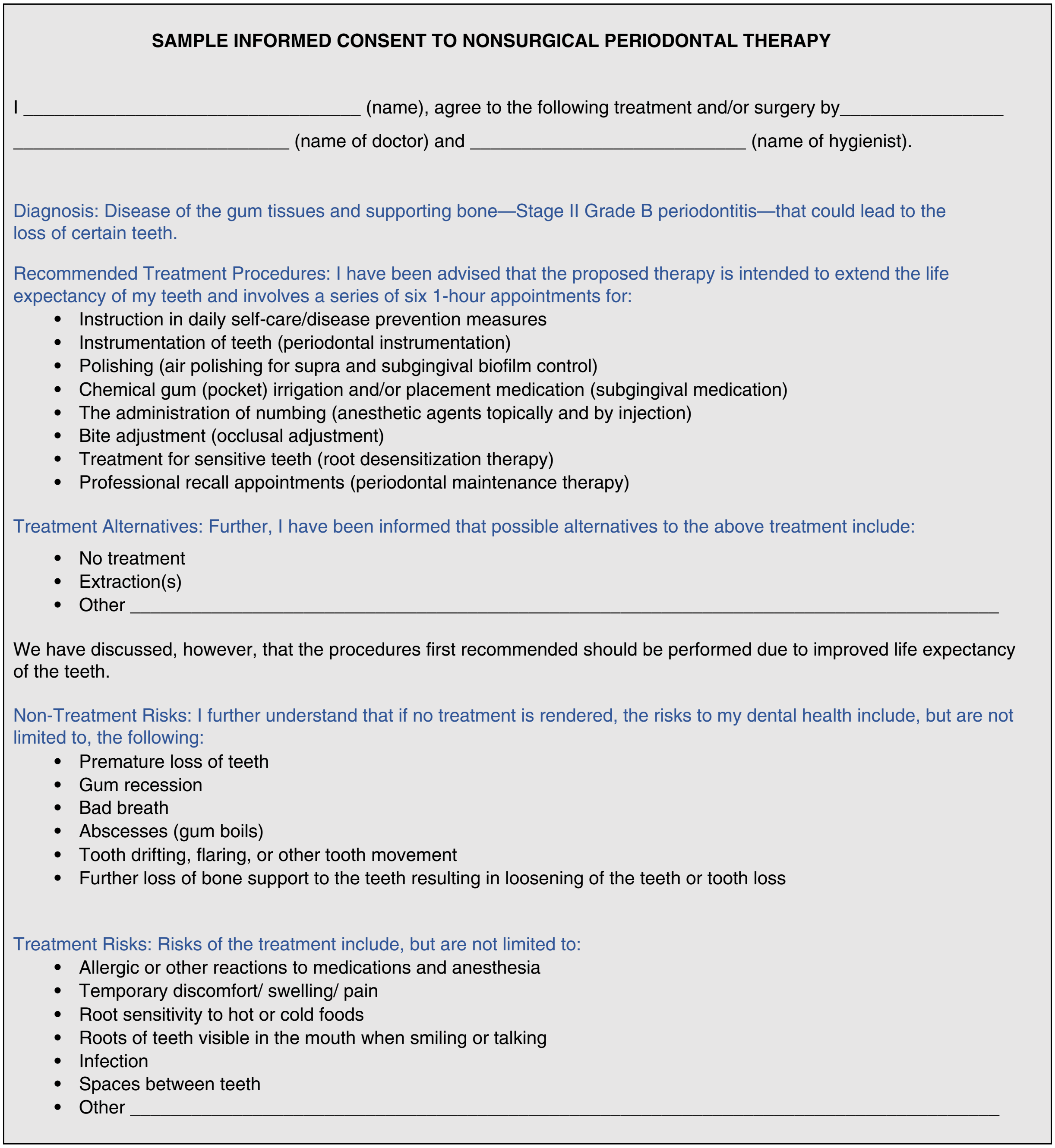
   b. **Who Is Responsible for Informing the Patient?** The standard is that the health care provider who performs the treatment is ultimately responsible to informing the patient and obtaining consent.
   c. **What Are the Goals of Informed Consent?** The most important goal of informed consent is to provide an individual an opportunity to be an informed participant in health care decisions, and it is generally accepted that a thorough informed consent includes a discussion of the following elements:
      1. The diagnosis and an explanation of the periodontal condition that warrants the proposed treatment.
      2. An explanation of the purpose of the proposed periodontal treatment.
      3. A description of the proposed treatment and the individual patient's role and responsibilities during and after periodontal treatment.
      4. A discussion of the known risks and benefits of the proposed periodontal treatment.
      5. An assessment of the likelihood that the proposed treatment will accomplish the desired objectives.
         a. Note that when discussing treatment outcomes it is important not to appear to guarantee treatment outcomes to the patient.
         b. Dental health care providers should remember that individual patients may respond differently to similar treatments.
      6. A presentation of alternative treatment options, if any, and the known risks and benefits of these options.
      7. The risks and benefits of not receiving the proposed periodontal treatment.
      8. A discussion of the prognosis (or outcomes expected) if no treatment is provided.
      9. A discussion of the actual costs associated with the proposed treatment.
      10. Reinforcement of the individual's right to refuse consent to the proposed treatment.
         a. Keep in mind that some patients feel powerless when interacting with health care providers.
         b. To encourage the patient's voluntary consent, the dental health care provider should make it clear to the patient that she or he is participating in a decision, not merely signing a consent form.

3. **Informed Refusal for Periodontal Treatment.** **Informed refusal** is an individual's right to refuse all or a portion of the proposed treatment. Informed refusal requires that recommended treatment, alternate treatment options, and the likely consequences of declining treatment have been explained in language understood by the patient. A patient always has a legal right to refuse proposed periodontal care.
4. **Obtaining Informed Consent from Patients.** The doctrine of informed consent reminds dental health care providers to respect patients by fully and accurately providing information relevant to their health care decisions. The following are some guidelines to consider when obtaining informed consent from a patient.
   a. **Use Understandable Language.** Information should be provided in language that is easily understood by the patient.
      1. Use simple, straightforward sentences.
      2. Use commonly recognizable terms.
      3. Avoid the use of professional jargon or technical terms and explain any terms that may not be readily understood.
      4. Use a translator if the patient does not speak English or speaks English with little understanding.
   b. **Provide Opportunities for Patient Questions.** An opportunity should be provided for the patient to ask questions. Foster an open exchange of information and encourage the patient to ask questions. Using open-ended and nondirective questions such as those below can simplify this process.
      1. "What more would you like to know?"
      2. "What are your concerns?"
      3. "What is your next question?"
   c. **Assess Patient Understanding.** An assessment should be made of the patient's understanding of the information provided.
      1. A simple strategy to assess understanding is to let the patient know that it is ok to ask questions: "Many people have difficulty understanding the information that I give them or have questions that they need answered. So, please let's discuss anything you do not understand."

    2. Another strategy is to make a comment such as, "Most of my patients want to explain my treatment suggestions to another family member. What additional information can I give you to help you explain this treatment to your spouse?"

5. **Legal Responsibility Related to the Consent Process**
    a. **The Dental Practitioner's Legal Responsibility Related to Consent**
        1. The dental hygienist has a legal responsibility for the services that he or she provides. Failure to obtain consent from the patient for services can have serious legal consequences.
        2. A patient could claim battery (i.e., unconsented touching) for dental services provided without the patient's consent.
        3. Patients also can claim negligence (i.e., lack of reasonable and prudent care resulting in harm) for failure to provide sufficient information for the patient to make an informed decision. This type of negligence also is called malpractice.
    b. **Basic Legal Requirement to Demonstrate Informed Consent.** Basic requirements for demonstrating that informed consent has been obtained are listed below. If any element were missing, it would indicate that informed consent is not complete.
        1. Patient's periodontal diagnosis presented in language that is easily understood by the patient.
        2. Discussion of proposed periodontal treatment and benefits presented in language that is easily understood by the patient.
        3. Discussion of the risks and likelihood of success of the proposed periodontal therapy.
        4. Discussion of alternative treatments.
        5. Documentation that the patient was encouraged to ask questions and that answers were provided in language that is easily understood by the patient.
        6. Patient's signature on the consent form.
    c. **Legal Requirements Necessary for a Patient to Give Informed Consent.** Certain legal requirements must be met for a patient to give informed consent. These legal requirements are listed below.
        1. The patient is fully informed (as discussed in point B above).
        2. The patient is of legal age. Legal age is determined by state law—not federal law—and so may vary from state to state. Dental health care providers should know the legal age for the state in which they practice.
        3. The patient is mentally competent (understands information presented and can decide about the proposed treatment).
        4. The patient can give voluntary consent (without coercion from care providers, family members, or others).
6. **Format for the Consent Process.** The format for the consent process may be either verbal or written. Some states have statutes or regulations requiring dental professionals to secure written informed consent from patients.[1,30] Dental team members should ensure that they are familiar with and in compliance with the informed consent laws in their states.
    a. **Written Consent.** Most dental health care providers prefer that the patient sign and date a written consent form for documentation of the consent process. **Figure 22-8** shows an example of a written informed consent/informed refusal form.
        1. In addition, the written consent document should be signed and dated by the dentist and a witness (generally, another staff member).
        2. Once signed, a written consent document becomes part of the individual's permanent dental record.
    b. **Verbal Consent.** If a written consent document is not used, the patient's verbal consent should be documented in the patient chart. An example of documentation of verbal consent would be a written entry in the patient's chart that says, "Discussed the diagnosis; purpose, description, benefits, and risks of the proposed treatment; alternative treatment options; the prognosis of no treatment; and costs. The patient asked questions and demonstrates that he understands all information presented during the discussion. Informed consent was obtained for the attached treatment plan."



SAMPLE INFORMED CONSENT TO NONSURGICAL PERIODONTAL THERAPY

I ________________________________ (name), agree to the following treatment and/or surgery by________________

__________________________ (name of doctor) and __________________________ (name of hygienist).

Diagnosis: Disease of the gum tissues and supporting bone—Stage II Grade B periodontitis—that could lead to the loss of certain teeth.

Recommended Treatment Procedures: I have been advised that the proposed therapy is intended to extend the life expectancy of my teeth and involves a series of six 1-hour appointments for:

- Instruction in daily self-care/disease prevention measures
- Instrumentation of teeth (periodontal instrumentation)
- Polishing (air polishing for supra and subgingival biofilm control)
- Chemical gum (pocket) irrigation and/or placement medication (subgingival medication)
- The administration of numbing (anesthetic agents topically and by injection)
- Bite adjustment (occlusal adjustment)
- Treatment for sensitive teeth (root desensitization therapy)
- Professional recall appointments (periodontal maintenance therapy)

Treatment Alternatives: Further, I have been informed that possible alternatives to the above treatment include:

- No treatment
- Extraction(s)
- Other ____

We have discussed, however, that the procedures first recommended should be performed due to improved life expectancy of the teeth.

Non-Treatment Risks: I further understand that if no treatment is rendered, the risks to my dental health include, but are not limited to, the following:

- Premature loss of teeth
- Gum recession
- Bad breath
- Abscesses (gum boils)
- Tooth drifting, flaring, or other tooth movement
- Further loss of bone support to the teeth resulting in loosening of the teeth or tooth loss

Treatment Risks: Risks of the treatment include, but are not limited to:

- Allergic or other reactions to medications and anesthesia
- Temporary discomfort/ swelling/ pain
- Root sensitivity to hot or cold foods
- Roots of teeth visible in the mouth when smiling or talking
- Infection
- Spaces between teeth
- Other ____

A

Figure 22-8 Sample Consent for Treatment Form. A. Page one of a sample consent for nonsurgical periodontal treatment.

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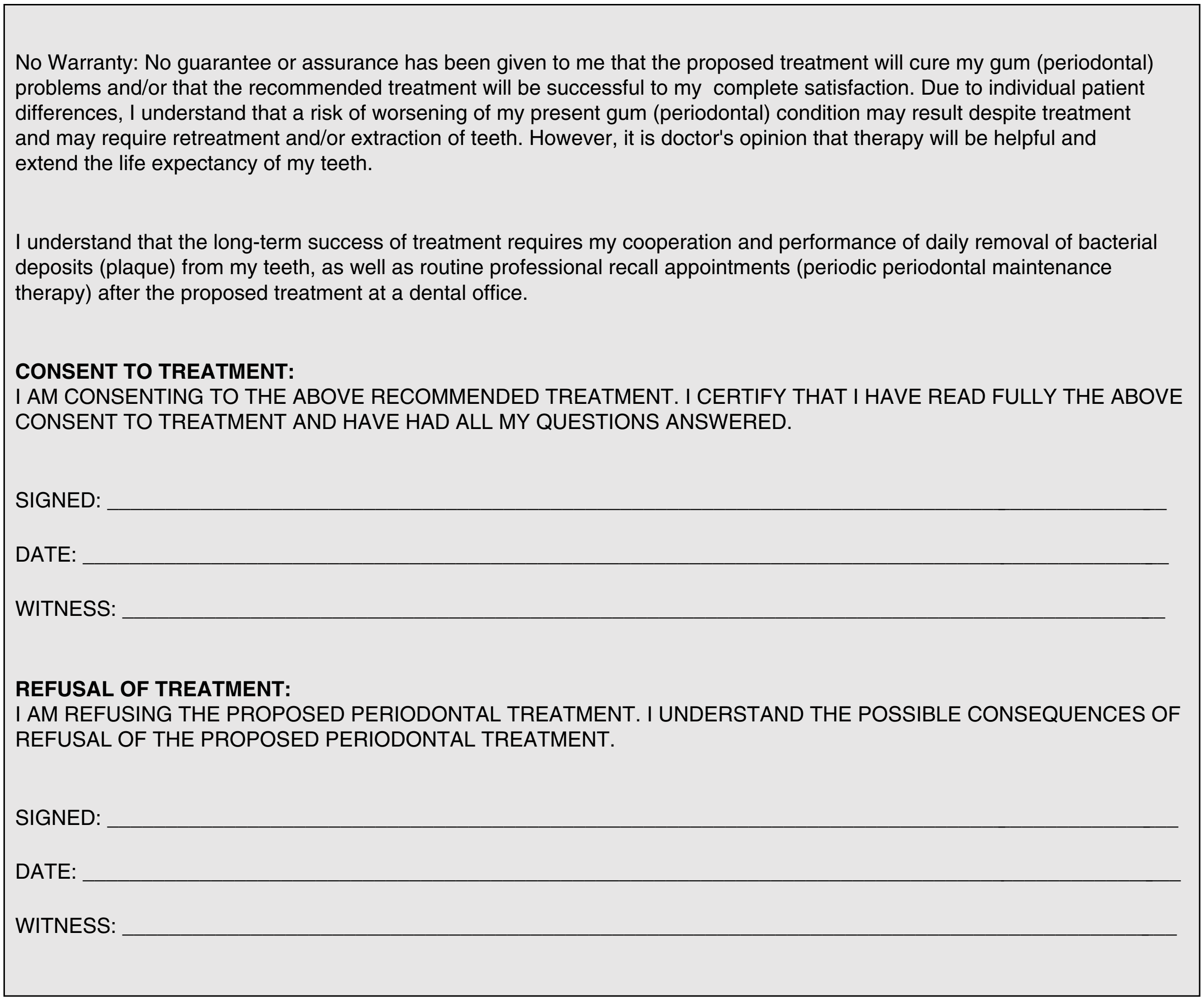

No Warranty: No guarantee or assurance has been given to me that the proposed treatment will cure my gum (periodontal) problems and/or that the recommended treatment will be successful to my complete satisfaction. Due to individual patient differences, I understand that a risk of worsening of my present gum (periodontal) condition may result despite treatment and may require retreatment and/or extraction of teeth. However, it is doctor's opinion that therapy will be helpful and extend the life expectancy of my teeth.

I understand that the long-term success of treatment requires my cooperation and performance of daily removal of bacterial deposits (plaque) from my teeth, as well as routine professional recall appointments (periodic periodontal maintenance therapy) after the proposed treatment at a dental office.

CONSENT TO TREATMENT:
I AM CONSENTING TO THE ABOVE RECOMMENDED TREATMENT. I CERTIFY THAT I HAVE READ FULLY THE ABOVE CONSENT TO TREATMENT AND HAVE HAD ALL MY QUESTIONS ANSWERED.

SIGNED: ______________________________

DATE: ______________________________

WITNESS: ______________________________

REFUSAL OF TREATMENT:
I AM REFUSING THE PROPOSED PERIODONTAL TREATMENT. I UNDERSTAND THE POSSIBLE CONSEQUENCES OF REFUSAL OF THE PROPOSED PERIODONTAL TREATMENT.

SIGNED: ______________________________

DATE: ______________________________

WITNESS: ______________________________

B

Figure 22-8 **B.** Page two of a sample consent for nonsurgical periodontal treatment. (*Continued*)

Chapter Summary Statement

Shared decision-making is a communication approach that seeks to balance clinicians' expertise with patients' attitudes, expectations, and preferences. This process involves providing the patient with evidence-based information about options, outcomes, and uncertainties. When patients are given decision aids to help them make choices, they are more knowledgeable and satisfied with their periodontal care options. Through this interactive process, the clinician and patient reach a mutual decision about the subsequent periodontal care. A patient always has a legal right to refuse proposed periodontal care. Dental health care providers have a legal responsibility for the services that they provide. Failure to obtain informed consent from a patient can have serious legal ramifications.

Section 5

Focus on Patients

Ethical Scenario 1

Mrs. Emmerich lives in a rural area and drives 2 hours for dental care. Several years ago (Dental Office A), she was diagnosed with generalized periodontitis. Her general dentist recommended that she see a periodontist. Mrs. Emmerich—who did not want to make the long drive for a consultation with a periodontist—told her general dentist that she would just continue to keep her regular 6-month appointments with the hygienist. Neither the general dentist nor the hygienist pursued the discussion any further with Mrs. Emmerich.

Several years later, Mrs. Emmerich moved to the city to be nearer her adult daughter. She is a new patient in your general dentistry practice (Dental Office B), and a comprehensive periodontal examination reveals attachment loss and mobility of the molar teeth. You carefully explain the findings to Mrs. Emmerich using clear, easily understood words. Once Mrs. Emmerich fully understands the periodontal diagnosis, you begin the process of shared decision-making with her.

Mrs. Emmerich expresses her regret to you that no one at the previous dental office explained her periodontal condition or her treatment options. She believes that if she had understood the diagnosis that she would have sought a consultation with a periodontist. She feels that her condition is much worse now than it might have been.

1. How do the approaches to care planning in the two dental offices differ?
2. How do you think that shared decision-making at the time of the initial diagnosis (Dental Office A) might have affected the patient's choices?
3. How do you think that shared decision-making at the time of the initial diagnosis (Dental Office A) might have affected the long-term prognosis of Mrs. Emmerich's dentition?
4. Why do you think Mrs. Emmerich was willing to accept a referral to a periodontist (from Dental Office B)? Was it just that she now lives in a city?

Ethical Scenario 2

Mrs. Nguyen is the first patient of the afternoon in your periodontal office. She is new to the practice. She is 50 years old and has only lived in the United States for the last 3 years. Her English skills are minimal, but she works as a medical researcher, so you assume that she understands medical terminology.

Mrs. Nguyen has Stage II, Grade B periodontitis with generalized horizontal bone loss on her posterior teeth. You determine that periodontal instrumentation using local anesthesia is indicated. The next phase of treatment will be based on the findings at the time of the reevaluation and will be determined by Dr. Evans, one of the periodontists in the office.

You review the proposed periodontal treatment plan with her. She asks no questions and signs on the signature line at the bottom of the consent for treatment form. You tell her that you will begin the recommended treatment at today's appointment. You prepare the anesthesia syringe and as you approach Mrs. Nguyen she becomes very agitated, screaming and covering her mouth with her hands. She refuses to allow any further treatment today. She gets up abruptly and leaves the office.

1. What could you have done differently to avoid the above situation?
2. What ethical principles are in conflict in this dilemma?
3. Now that Mrs. Nguyen's experience has been a negative one for her, what is the best way to handle this ethical dilemma?
4. What changes might you make when explaining the proposed treatment to future patients?

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CHAPTER 23

Encouraging Patient Behavior Change with Motivational Interviewing

- Section 1 Introduction to Human Behavior Change
- Section 2 Components of Motivational Interviewing
- Section 3 Implications for Dental Hygiene Practice
  - Motivational Interviewing: Example 1
  - Motivational Interviewing: Example 2
  - Motivational Interviewing: Example 3
- Section 4 Focus on Patients
  - Clinical Patient Care
  - Ethical Dilemma

CLINICAL APPLICATION

The SHARE Approach is a five-step collaborative process for shared decision-making. One of the core components of this approach is the recognition that individuals can advance their own health by making changes to their behaviors. Eliciting such changes to an individual's health behavior(s), however, can be challenging for both the patient and the health care provider. Nevertheless, a patient-centered interventional strategy known as motivational interviewing (MI) can facilitate behavior change. Rather than confronting and compelling a patient to make the necessary changes, MI centers on a more patient-friendly, collaborative, conversational approach between the patient and provider that encourages the patient to self-explore their initial reluctance or ambivalence to change, which, in turn, often sparks his or her motivation and commitment to change. By doing so, the patient—not the health care provider—takes the lead in committing himself or herself to taking the necessary steps to make the positive change(s). As such, MI empowers the individual patient—rather than the health care provider—to take on greater ownership to promoting and preserving his or her own health through essential changes in behavior. The aim of this chapter is to review the essential role that MI plays in encouraging patients to express their own barriers to change and to explore and resolve their own ambivalence and reluctance to behavior change(s).

LEARNING OBJECTIVES

- Recognize the role of ambivalence in patient behavior change and explain the goal of motivational interviewing with respect to ambivalence.
- Describe the primary difference between how hygienists often approach patient education and the motivational interviewing approach.
- Identify the four key elements of motivational interviewing.
- Give examples of specific motivational interviewing methods and how they are used to enhance patient motivation for change.

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KEY TERMS

Motivational interviewing (MI)
Patient-centered
Guiding style
Ambivalence
Partnership
Acceptance
Compassion
Evocation
OARS
Open-ended questions
Reflective listening
Affirm
Summaries
Elicit–provide–elicit
Engaging
Focusing
Evoking
Planning
Change talk
Sustain talk
Developing discrepancy

Section 1

Introduction to Human Behavior Change

1. Improved oral health often requires behavior change. The SHARE Approach, which was presented in Chapter 22, is a five-step collaborative process that involves the joint efforts of the health care provider and the patient to formulating a health care decision(s) that is best for the patient. Utilizing the SHARE Approach can facilitate better interaction between the patient and the health care provider, increased patient satisfaction, improved patient-centered care delivery, and enhanced patient compliance to treatment recommendations. One of the key components of the SHARE Approach is recognizing that the long-term success of health promotion hinges on patient self-awareness and patient motivation to shift away from negative behaviors (like sporadic self-care or smoking) and toward positive behaviors that facilitates good oral health.
    a. Behavior change is important for prevention of periodontal diseases and maintenance of the periodontium.
        1. Periodontitis is a largely preventable oral disease but prevention often requires the patient to be actively involved in making and maintaining changes in his or her oral self-care and lifestyle habits.
        2. Oral health care providers can recommend to their patients the importance of maintaining certain behaviors that reduce the risk of disease onset or arrest the progression of the disease. Some of the essential recommendations an oral health care practitioner can make to a patient is the daily use of a soft bristled toothbrushes and interdental cleaning aids.
    b. Helping patients change behavior can be very challenging.
        1. Oral health care providers often encounter periodontal patients who do not practice effective oral self-care measures nor adhere to recommendations for professional periodontal maintenance. To motivate patients, oral health care providers can play a leading role in encouraging all patients to make positive changes to their lifestyle habits. If patients are continuously encouraged to stick with these changes for the long term, then these changes will become part of their daily lives and lead to better health.
        2. Unfortunately, education and professional advice are usually not very effective in getting patients to change their behavior. Patients often do not follow through and may even argue with their oral health care provider about why they are unable to make the change. Patients can become resistant, and health care providers can become frustrated and even convinced that talking about behavior change is pointless (**Figure 23-1**).
    c. There are many reasons why patients do not change their behavior.
        1. Although it is tempting to blame the patient for being non-compliant, a great deal of research has shown there are many understandable reasons why patients struggle with health behavior change.
        2. Patient motivation is affected by such things as past life experiences (e.g., attitudes, beliefs, and habits developed over time), their current circumstances (e.g., lack of time, lack of money, health literacy, and knowledge), and their confidence and skills to make the necessary behavior change.[1,2] These can be very big barriers and probably explain why even some oral health care providers do not lead perfectly healthy lives!

Figure 23-1 The Self-Care Struggle. Patients often struggle with professional recommendations for self-care, such as flossing, leading to frustration or even reluctance to adhering to oral self-care routines.

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3. The communication style used by the oral health care provider can either reinforce or curtail patient motivation to change.[3,4] Motivation is important because change in behavior usually takes energy and time. Patients are asked to make behavior change an important priority even though they may not have decided whether it is an important priority.

d. Education and advice can reduce patient motivation.
   1. Health care providers typically try to persuade their patients to change by offering facts about oral diseases—such as dental caries and periodontal disease—and giving advice on what the patient needs to do differently. This "directive" style of communication in which the provider acts as the "expert" and the patient is the "recipient" often reduces motivation or makes the patient resistant or ambivalent to change.
   2. When patients are not ready to change or struggle with change, being directed to change can often make them feel embarrassed, guilty, or even ashamed. Patients often become defensive, explaining why they cannot or have not been doing the right thing (**Figure 23-2**). They may ignore the advice given or delay returning for treatment.[5–8]

Figure 23-2 Defensive Patient Response. Health care education given to a patient who does not have a good insight into his or her oral condition or may be reluctant or even ambivalent to initiate/continue treatment may react to oral hygiene instructions with a sense of defensiveness.

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    3. Studies have shown that approximately 23–60% of health information provided by clinicians is forgotten within an hour! Also, when asked to recall what advice was given and what next steps were agreed to during an encounter, patients and providers do not agree.[9,10] Therefore, it is not surprising that 50% of health recommendations provided by clinicians are not followed.[10]
    4. To better help patients with behavior change, an alternative to the usual expert-oriented, directive, educational approach is needed. Motivational interviewing is an alternative method that is effective at instilling behavior change.

2. **Motivational interviewing (MI)** is a patient-centered approach that aims to influence change by guiding the patient to make lifestyle changes rather than directing. MI offers a way of working with patients on behavior change that differs from directive education or advice. Rather than assuming the reason patients do not change their behavior is because they need information and advice, MI focuses on exploring what the patient thinks about changing his or her behavior.
    a. MI is a way of building motivation for change.
        1. MI guides and counsels patients to instill positive behavioral change. The goal is to collaborate with patients in strengthening their own (internal) self-motivation and commitment to change rather than being directed by another individual.[6]
    b. MI is "patient-centered."
        1. Being **patient-centered** means the clinician works to understand the way the patient sees the situation. For example, the clinician may want a patient not to smoke to prevent periodontitis. Although the patient might want to have healthier gingiva, he or she may enjoy smoking too much to quit. It is important for the clinician to understand, acknowledge, and be accepting of the fact that the patient finds smoking enjoyable.
        2. Although the approach is patient-centered, this does not mean that the clinician should forego encouraging behavior change. In MI, the clinician *does* influence and guide the patient toward healthy behavior change, but not in a directive and paternalistic way.
        3. The alternative to being directive is to use a guiding style.[11] In the **guiding style**, a clinician does not tell the patient what to do, but rather asks the patient what his or her thoughts are about trying to floss every day and how this behavior change would impact the patient's life.
    c. Patients are ambivalent about change.
        1. One of the assumptions of MI is that it is normal to have mixed feelings or **ambivalence** about *any* behavior change process. For example, how do you feel about recommended health behaviors like eating at least five servings of fruit and vegetables every day, or exercising for 40 minutes 5 to 7 days a week? Almost everyone has experience with struggling to engage in some behavior they know they ought to be doing. When you think about your own behavior, it is usually quite easy to see the reasons why you want to engage in the healthy behavior and reasons why you do not or cannot.
        2. MI also assumes that, when possible, patients *want* to be healthy. Even though patients may emphasize why they are not able or motivated to make a change, they almost always prefer to be healthy.
    d. MI focuses on internal motivation.
        1. In MI, the clinician encourages patients to talk about their reasons for change because the patient's own reasons for making a change are considered much more likely to lead to behavior change than reasons a clinician might provide.
        2. One way to think about this is that patients are more likely to act if they are internally rather than externally motivated. Specific strategies are used to increase internal motivation.

3. MI is effective and useful. MI is being used more and more in health care because of its effectiveness and usefulness.
    a. MI is clinically proven.
        1. You may be thinking that MI sounds quite complicated and wondering if trying to understand it is worth it. One reason you may want to try applying MI in your practice is that research shows it is effective.
        2. Studies that combine the results of multiple studies find MI to be effective in changing many different behaviors, including those that improve health.[12–16] Studies with periodontal patients have also had generally good results.[17–24]

    3. Patient outcomes also seem to improve in relation to the amount of time or number of sessions of MI that are provided.[13] This suggests that hygienists could maximize their impact on periodontal patients if they applied MI repeatedly at every recall visit.
  b. Dental hygiene students are learning and using MI.
    1. MI training is increasingly becoming part of the dental hygiene curriculum in the United States and abroad.
    2. A recent study found that both dental hygiene faculty and students liked having MI as part of the curriculum. Students who received MI training also performed better on a standardized patient assessment of their ability to motivate patients.[25]
    3. Although clinicians are often worried about the time it may take to do MI, as little as 15 minutes has been shown to be effective in the majority of studies.[12,26]

Section 2

Components of Motivational Interviewing

1. **The "Spirit" of Motivational Interviewing**
  a. **Key Elements.** MI is more than just a set of techniques or strategies. MI requires clinicians to adopt a particular philosophy or "spirit" of working with their patients. The philosophy or "spirit" of MI is captured in four key elements (**Figure 23-3**).

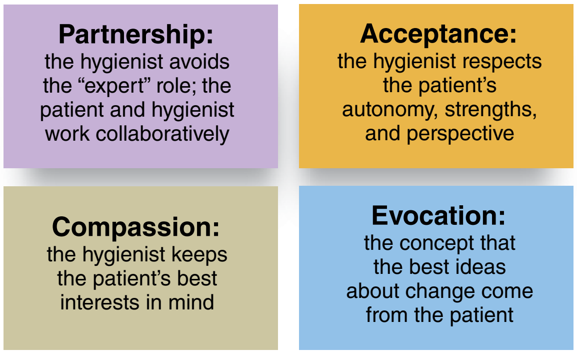
Partnership:
the hygienist avoids the "expert" role; the patient and hygienist work collaboratively
Acceptance:
the hygienist respects the patient's autonomy, strengths, and perspective
Compassion:
the hygienist keeps the patient's best interests in mind
Evocation:
the concept that the best ideas about change come from the patient

Figure 23-3 Four Key Elements of Motivational Interviewing. The MI strategy integrates four key elements: partnership, acceptance, compassion, and evocation.

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    1. The first element is **partnership** between the patient and clinician. In the MI approach, the clinician tries to avoid being "the expert." The patient, not the clinician, is viewed as the expert on his or her life and the challenges of behavior change.
    2. The second element is complete **acceptance** of the patient and his or her choices. Acceptance includes avoiding judgment, supporting the patient's freedom to make their own choices, understanding the patient's perspective, and affirming their strengths and efforts.
    3. The third element is **compassion**, which refers to the importance of using MI only for the welfare of the patient.
    4. The fourth element is **evocation**, in which the clinician encourages the patient to do the talking. This is how the clinician learns about the way the patient views the situation and finds out about any internal motivation the patient may have. The clinician does not attempt to persuade the patient.
  b. **Responsibility for Change.** Although it is sometimes easy for a health care provider to feel frustrated with the behavior of a patient, in MI, the clinician recognizes that he or she not responsible for the patient's decisions. It is ultimately not the clinician's choice or life.
    1. Patients are far more likely to choose and succeed with behavior change when they have voiced their own reasons for change and made their own decision to commit.

2. The final decision for change rests with the patient. The clinician may communicate respect for the patient's freedom to choose directly such as "So how would you like to proceed?" or indirectly such as at the beginning of the visit by asking permission to talk about oral self-care (**Figure 23-4**). Paradoxically, patients who feel their provider is giving them the freedom to choose are more likely to follow the guidance of their provider.

Figure 23-4 **Asking Permission.** Starting with asking the patient's permission can help foster a collaborative partnership.

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2. **MI Core Skills and Strategies.** MI requires skillful use of four core communication skills. **OARS** is a brief way to remember these four skills: Open-ended questions, Affirmations, Reflections, and Summaries. In health care settings, it is also important to know how to give advice in a patient-centered style (see E below). We have added this as another core skill of MI.
   a. **Open-Ended Questions**
      1. **Open-ended questions** are inquiries that are framed to avoid a simple yes/no response. For example, "How do you feel about using dental floss?" or "How does brushing and flossing fit into your routine?" rather than "Do you use dental floss?" or "Do you brush twice a day?"
      2. The open-ended style of questioning encourages patients to elaborate and provides much more information to the clinician regarding the patient's perspective (**Figure 23-5**).

Figure 23-5 **Use of Open-Ended Questions.** Open-ended questions—questions that cannot be answered with a simple "yes" or "no" response—encourage the patient to provide additional information.

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b. Reflective Listening

1. **Reflective listening** (also referred to as active listening) is a communication process in which the health care provider listens to the patient's remarks, then paraphrases what the clinician heard the patient say. This approach allows the clinician to check if he or she is correctly understanding the meaning of what the patient is saying (**Figure 23-6**).

Figure 23-6 **Reflective Listening.** By paraphrasing the patient's remarks, the clinician reassures the patient that he or she is listening and understands what the patient is saying.

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2. Reflective listening helps patients feel understood and accepted, even if their behaviors are not healthy. Expressing understanding (or empathy) also typically encourages the patient to elaborate so that the clinician can learn more about how the patient sees the situation.
3. Skilled reflective listening incorporates body language and tone of voice and may also involve inferring what is implied by the patient's remarks. For example, the patient's tone of voice may suggest emotions (such as worry or fear) that are not directly expressed in words (e.g., "Talking about quitting smoking is difficult because it makes you think about serious health effects that scare you.").
4. Through the process of open-ended questioning and reflective listening, the clinician shifts from being a directive expert to applying a patient-centered guiding style. Unfortunately, research shows that the average health care provider interrupts patient disclosures after 18 seconds.[10] Interrupting the patient sends a clear message that the patient's input is not respected or seen as relevant.
5. Inviting patients to explain their view of the situation through open questions and reflective listening increases patients' willingness to honestly express themselves about their ambivalence or mixed feelings about change (**Figure 23-7**).

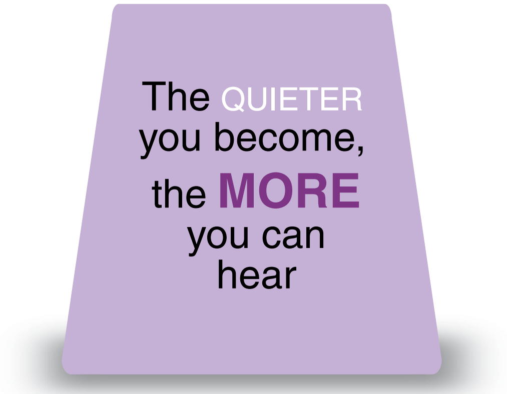

Figure 23-7 **Listening.** As clinicians, we all need to be reminded of how important it is to sometimes just sit back and listen to what our patients have to say.

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c. **Affirm**
   1. In MI, the clinician should **affirm** (acknowledge, support) the patient's willingness to discuss a difficult topic (i.e., a change about which they may feel embarrassment, guilt, or ambivalence).
   2. The clinician may express appreciation for the patient being willing to discuss their smoking or taking some small steps toward change. Affirming can strengthen the bond between the clinician and the patient and encourage the patient to engage further in the behavior change discussion (e.g., "Thank you for taking the time to help me understand how busy your days are now that you are caring for your elderly mother in your home.").

d. **Summarize**
   1. Brief **summaries** are used in MI to link and reinforce what has been discussed and provide an opportunity for the clinician to demonstrate empathy; for example, "So I think I now have a fairly good picture of how you view your smoking habit …".
   2. Summaries can be used to move the conversation in a new direction; for example, the summary can lead to a statement like "So given what we've talked about so far, what are your thoughts about the next step …".
   3. Summaries also are used to tie different elements of the conversation together. For example, after summarizing the patient's perspective on the difficulties of brushing regularly, the clinician might link to an earlier part of the conversation on the patient's dislike of the pain that he is experiencing: "So it sounds like it's a real challenge to get a good brushing routine but at the same time you are really unhappy with the pain you are experiencing."

e. **Providing Information and Advice in MI**
   1. As discussed, it is very common for clinicians to take on the role of expert and to give their patients advice. Yet there are times when it would be helpful to provide information to a patient (e.g., if the patient asks for information, or if new information might help in overcoming obstacles to change). MI offers strategies for giving information that help avoid "taking on the expert role." One strategy is to first ask for permission: "Would you be interested in hearing some more information about the benefits of quitting smoking for your oral health?" (**Figure 23-8**). When providing information, the clinician can further avoid coming across as the expert by referring to other sources for the information, such as "Many patients tell me …" or "Research seems to indicate …" rather than saying "I have found …" or "I would recommend …".

Figure 23-8 Providing Information or Advice. In this example, the clinician supports the patient's autonomy by asking permission before providing information.

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   2. Another strategy is to use a three-step process known as **elicit-provide-elicit**. The first step (elicit) involves determining what the patient already knows. For example, "What have you heard about the benefits of flossing on a daily basis?" The second step (provide) involves providing relevant information and advice: "Yes, you are right, those are some good reasons to floss daily. There are also some

other reasons dental hygienists encourage flossing for cases like yours, such as …". The final step (elicit) involves checking back with the patient to see what he or she has taken from the information or advice provided. For example, "So I'm wondering what you think about that research and how it applies to you?"

3. **The Four Processes of Motivational Interviewing.** The four processes of MI are engaging, focusing, evoking, and planning. These four processes may not all occur in one appointment or in sequence. Rather, the four processes are employed, as needed, throughout the conversation.
    a. **Engaging: The "Connection" Process.** The first process upon meeting a patient is to engage the patient in wanting to work together. **Engaging** establishes the connection or rapport that is the foundation for everything that follows.
        1. Core skills, or OARS, are used in all four stages of MI. Rather than beginning the interaction with a series of closed-ended questions to gather patient information, in MI the clinician immediately encourages the patient to talk using the OARS skills:
            a. An open-ended question can be used to initiate the discussion (e.g., "What kinds of problems have you been having with your teeth lately?").
            b. Reflective listening can then be used to encourage the patient to elaborate while at the same time communicating empathy and acceptance (e.g., "You've noticed your gums are bleeding a lot.").
            c. During the listening process, the clinician pays attention to any opportunities to affirm the patient for any strengths or efforts (e.g., "You've really been making an effort to try to brush more regularly.").
            d. Summaries can be used to tie the conversation together highlighting the most relevant parts of the patient's story.
        2. During this engaging process, the clinician is focused on developing an understanding of the situation as the patient sees it. There is no attempt to provide education or advice.
        3. The clinician should be mindful of the following during engaging:
            a. Am I using open-ended questions to encourage the patient to talk openly?
            b. Am I using reflective listening once the patient starts answering my questions?
            c. Can I summarize the patient's view of the problem?
    b. **Focusing: The "What" Process.** The focusing process is about collaborating with the patient to find a clear direction or goal. At this stage, the OARS skills are used to develop an understanding of patient priorities; as well as, to collaborate with the patient to determine focus for behavior change.
        1. **Focusing** is the process of clarifying, in collaboration with the patient, what direction the conversation about behavior change should take.
        2. A guiding style can be used to explore the patient's preferences, concerns, and priorities while considering the clinician's concerns and priorities. For example, the clinician may offer a range of alternative options for discussion (e.g., "We should decide what is most important to focus on today. We could talk about some of the challenges you're having with your brushing and flossing routine, we could discuss how to go about changing some of your eating habits to protect your teeth and gums, or we could talk about your smoking and how I could perhaps be of some help.").
        3. During the focusing stage, it may be necessary to provide information to the patient. The clinician can use the elicit–provide–elicit method to accomplish this style consistent with MI.
        4. The clinician should be mindful of the following during engagement:
            a. What goals for change does this patient have?
            b. Do I have different goals for change for this patient?
            c. Are we working together with a common purpose? Or moving in different directions?
    c. **Evoking: The "Why."** With the goal in mind, the process of **evoking** is used to encourage the patient to express his or her motivation and confidence to change. The OARS skills are used to encourage patients to talk about and elaborate on their reasons for change and, as necessary, to acknowledge reasons he or she does not want to change.
        1. As discussed previously, most patients have mixed feelings about change. In this process, the role of the clinician is to encourage the patient to talk about ambivalent feelings and elicit reasons that he or she has for wanting to change. The task of the clinician is to evoke, facilitate, and strengthen the patient's own desire for change.

  a. Statements made by the patient "in the direction" of change are referred to as **change talk** (e.g., "I know smoking is bad for me; I would like to do a better job with taking care of my teeth."). Research indicates that the more patients express change talk, the more likely they change their behavior.[6,27]
  b. Change talk can be encouraged with a simple open-ended question such as "What advantages, if any, do you see of cutting back on sugary foods?"
  c. Another approach is to use the "motivation" or "importance ruler" in which the clinician asks, "On a scale of 0 to 10 with 10 being most important, how important is to you to improve your oral self-care habits?" (**Figure 23-9**). Because patients are generally ambivalent they will rarely answer this question with a "1." This implies that they place at least some importance on making a change.

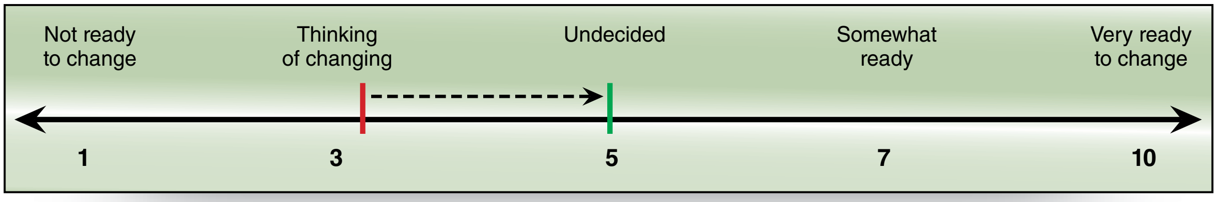

Figure 23-9 **A Motivation Ruler.** A motivation ruler gauges the patient's interest to making changes to his or her health and lifestyle. A patient who rates his or her motivation to change as a "1" is unmotivated and unwilling to commit to making the necessary behavior changes. In contrast, a patient who rates his or her motivation as a "10" is very interested in making changes to his or her health and lifestyle and is prepared to make the necessary behavior changes. The motivation ruler is an effective and simple measurement tool to use in MI as it is designed to facilitate and enhance change and commitment.

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    1. Once the patient identifies the self-rated importance of change (red bar), the clinician encourages change talk by asking, "What made you pick [number] rather than zero?" Asking this open-ended question encourages the patient to express any importance that he or she *does* place on improving oral self-care behaviors even if the number is low. This is change talk.
    2. After asking about and reflecting change talk, barriers to change can also be explored by asking, "What would it take for you to increase the importance from a [number] to a [higher number]?" (green bar). This approach can also be used to encourage talk about the patient's confidence (or self-efficacy) to make a change. Having confidence to make a change is a key contributor to an individual's motivation and success in making behavior change.

2. In general, once the clinician has encouraged the initial expression of change talk, the goal is to encourage even more change talk.
  a. Reflective listening is very effective means of encouraging elaboration of a topic.
  b. For example, a reflection might sound like: "You want to cut down on sugary foods partly because you want to set a good example to your children." This encourages elaboration by the patient of the desire to set a good example.
3. Because some patients are ambivalent, they are also likely to talk about the reasons they do not want to change or why they find it difficult to change. Statements that are against change (or in favor of continuing with their current behavior) are referred to as **sustain talk** (e.g., "I don't have enough time to floss every day.").
  a. When the patient expresses reasons not to change, this is an ideal opportunity to listen well and acknowledge the patient's perspective rather than attempting to compel or pressure the patient to make the recommended changes by providing counterarguments.
  b. Acknowledging the patient's mixed feelings can most simply be done with reflective listening (**Figure 23-10**). Patients whose concerns or hesitations about change have been validated through reflective listening are usually more prepared to divulge the reasons why they might want to change.

Figure 23-10 Reflecting Sustain Talk. When a patient expresses hesitation about change, the clinician should listen well and explore the patient's point of view rather than attempting to compel or pressure the patient that change is needed.

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4. As mentioned previously, a key component to encouraging behavior change is having the confidence to overcome obstacles to making the change.[2] It is common for patients to bring up obstacles to change after they have acknowledged the need to change or the desire to change. To help encourage patients to overcome obstacles, the clinician can evoke confidence in various ways, including brainstorming possible solutions, providing information and advice, or reviewing past successes.
5. Evoking may also involve **developing discrepancy** in which inconsistencies between a patient's values and goals and current behavior are explored to enhance motivation for change. For example, most patients want to be healthy even though they may miss appointments for periodontal maintenance.
   a. The discrepancy between what would be ideal (e.g., be healthy or have no pain) and the status quo (poor self-care that results in periodontitis) can be elicited through open-ended questions and reflective listening (**Figure 23-11**).

Figure 23-11 Discovering Discrepancy. Open-ended questions and reflective listening can expose discrepancies between what would be ideal (e.g., improved health) and the status quo (continuing to smoke).

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    b. For example, the hygienist might highlight the discrepancy with this reflection: "It sounds like you find it hard to stick with the thorough brushing and flossing each day, but you would really like to have healthy gums." This type of reflective statement highlights the discrepancy between the patient's behavior and what he or she says that he or she values (healthy gums).
  6. The clinician should be mindful of the following during evoking:
    a. What are the patient's reasons for wanting to change?
    b. Is the patient's reluctance due to a lack of confidence in his or her ability to change or is it because the patient feels that the change is not important?
    c. What "change talk" am I hearing?

d. **Planning: The "How" Process.** When patients become sufficiently interested in making change, the process of **planning** can begin. The goal is to encourage commitment to change and to develop a specific plan of action for change. The OARS skills are used in this process to ensure the plan for change is developed collaboratively rather than falling into the trap of telling the patient what to do.
  1. The planning process can begin when the patient is ready to discuss changing. Signs of readiness include the patient expressing more change talk and less sustain talk, asking questions about change, or taking some small steps toward change. At this stage of the process, the hygienist should remain alert to any renewed patient ambivalence that might call for a return to one of the earlier three processes.
  2. The clinician guides the patient to consider whether he or she wants to make a change and how that change will occur.
    a. Effective planning involves discussing with the patient the precise "how?" and "when?" of making the change. For example, rather than simply agreeing with the patient that he or she will "try to do a better job of brushing" the clinician might help the patient to work out a specific plan that will increase the likelihood of brushing (e.g., to brush right after eating dinner rather waiting until just before going to bed when the patient feels too tired to do even one more thing).
    b. Once the plan is developed the clinician will attempt to strengthen commitment to the plan by evoking intention and commitment from the patient (e.g., "What do you think of that strategy?" or "Does it sound like something you want to try?").
  3. The clinician should be mindful of the following during planning:
    a. Does the patient seem on board with making a plan?
    b. Is the patient coming up with the main ideas for the plan?
    c. Once the plan is summarized, is the patient willing to commit to the plan?

Section 3

Implications for Dental Hygiene Practice

Moving from the clinician-centered perspective of oral health education to the patient-centered view of behavior change can enhance the patient's motivation for a healthy behavior change. Using motivational interviewing, the hygienist can engage in a more productive interaction in which he or she no longer feels responsible (and frustrated!) about the patient's decisions. Instead, the hygienist focuses on encouraging the patient to examine his or her attitudes about periodontal disease, healthy oral health behaviors, or the periodontal maintenance schedule.

Motivational Interviewing: Example 1

Mrs. J is a 50-year-old attorney who has been referred to your periodontal dental office for evaluation and treatment. Mrs. J has type 2 diabetes, a glycosylated hemoglobin value (HbA1c) of 8, and moderately severe chronic periodontitis. Mrs. J has been sporadic in visiting her regular dentist, and the letter from the referring dentist states that after a 1-year absence Mrs. J presented recently with increased bleeding on probing and evidence of increasing attachment loss. The general dentist is concerned that without periodontal therapy Mrs. J. will likely lose several teeth.

Hygienist: I see your general dentist has referred you here for periodontal treatment. Can you tell me a little about your past dental care and why you are here today?

Mrs. J: Yes, Dr. Smith thinks if I don't get specialty treatment I'm going to lose some of my back teeth. I take care of my teeth and I think he's over reacting to my gums bleeding a bit more than usual. My gums have always bled.

Hygienist: I am very happy you followed up with Dr. Smith and came in to see us. [*Affirmation*] So if it's okay with you, I'd like to spend a little time talking about the health of your gums. [*Getting permission*]

Mrs. J: That's fine with me.

Hygienist: Good. So, if you would, can you tell me what is a typical day is like for you and how taking care of your teeth fits into your normal day? [*Open-ended question*]

Mrs. J: Sure. Most days I need to be at my office by 8:23, so I generally get up around 7, shower, have some coffee and toast, check emails, brush my teeth and drive my 20-minute commute to the office. Once I'm at my office, my schedule is really full. I'm currently working on three really difficult cases that take up most of my time. I'm pretty stressed out! I don't eat a regular lunch but will usually snack on a granola bar to hold me over to dinner. I'm usually home around 6:23, have dinner, and then catch up on reading documents for the next day. Sometimes I brush my teeth before bed, but mostly I'm just too exhausted to care at night. I do try, but don't always succeed. The dental hygienist at Dr. Smith's is always on my case about not brushing enough, not flossing enough, and not coming in every 3 months.

Hygienist: It sounds like you have a really busy schedule that makes it difficult for you to keep up with recommendations given by Dr. Smith's hygienist. If I understand you, though, you do make a concerted effort to brush at least every morning. [*Reflective listening*]

Mrs. J: Yes, I am very good about brushing in the morning.

Hygienist: That's great. It's important to have a routine that works well for you. [*Affirmation*] You mentioned that the hygienist at Dr. Smith's office is concerned that you were not flossing enough or coming in every 3 months. Tell me about that. [*Open-ended questioning*]

Mrs. J: Shelley, that's her name, says that if I don't floss every day and come in to have my teeth scraped every 3 months that I'll end up losing my teeth. She lectures me every time I go in, and I really dread going to see her. I don't think she understands how busy I am. The only reason I agreed to come here was that Dr. Smith thought you might be able to do something to get rid of my pockets.

Hygienist: So, it sounds like you are interested in improving your gum health but don't like being lectured when you go to the dentist. [*Reflective listening*]

Mrs. J: That's right. I also read in a magazine that diabetes might make my gum disease worse, so I figured it's worth finding out. Is that true? Can my diabetes make my gums worse? Can you get rid of my pockets so this gum disease will stop?

Hygienist: There is quite a bit of new information about diabetes and periodontal disease, and there are some things that we can do at our office to reduce pockets. Research has shown that controlling gum disease, especially in diabetics, requires professional care but also requires patients to be actively involved in helping control the disease on a day-to-day basis. We have had many patients who have had very good success using this approach. Would you like to know more about it? [*Providing information that was requested/Supporting autonomy*]

Mrs. J: I would like to know more, but I'm skeptical that it's going to be more of the same that I hear from Shelley—"brush—floss—come to see me—brush—floss—come to see me."

Hygienist: Right … you want help but worry that it will still involve more effort on your part than you really have time for. [*Reflect*]

Mrs. J: Exactly!

Hygienist: Well, I want to emphasize that how we move forward is up to you. The time and effort you put in brushing and flossing is something you will need to determine based on what's most important to you. [*Support autonomy*] If it's ok with you [*Support autonomy/Ask permission*], I'd like to try to go slowly, try to work with your busy schedule, and help you keep your teeth. After I conduct my exam, I'd also like to give you some information on diabetes and gum disease that you can read before your next visit and then we can talk more about how that might be contributing to your gum disease. Are you interested in that? [*Support autonomy/Ask permission*]

Mrs. J: That sounds good—the magazine article didn't give much detail, but it did say that diabetes can make gum disease worse. I'll make it a point of adding it to my evening reading tonight.

Hygienist: Excellent! I think you might find the information helpful as we work toward getting your gum disease under control.

Motivational Interviewing: Example 2

Mr. A is a 58-year-old banker who has come to the dental office wanting an implant for a first premolar that was lost due to periodontal disease last year. At the time, he was not interested in discussing a replacement but has made an appointment today, because he wants to get an implant for replacement. Although he brushes and flosses regularly, his oral self-care has only been fair for many years, he has a 23-year tobacco habit, and he is currently smoking about a pack and a half of cigarettes a day.

Hygienist: Good morning Mr. A. What brings you in today? [*Open ended questioning*]

Mr. A: Well, you know, I have this missing tooth here and when I chew it really bothers me. You know, food gets stuck up in there and on top of that it just doesn't look good when I smile. A buddy at work told me about implants and I'm seriously interested in getting one.

Hygienist: So, where your tooth is missing is causing you some problems and you aren't happy with the appearance of that gap. [*Reflective listening*]

Mr. A: Yeah, every time I eat I get food caught. It doesn't hurt but it's gotten really irritating. I know we talked about it when you took the tooth out, but I don't want a partial. I really want the implant.

Hygienist: Your friend told you about dental implants and you think it's the right choice for replacing that tooth. [*Reflective listening*]

Mr. A: That's right. I know when I was here before you said that my smoking might be a problem with getting an implant.

Hygienist: Yes, smoking is a major barrier for using implants because it makes it much harder for healing to occur and then maintain the health of tissues around the implant. But it sounds like you are really interested in getting an implant. [*Providing information in response to an implied question/Reflective listening*]

Mr. A: I am!

Hygienist: Well, can we spend a few minutes talking about your smoking, and what your thoughts and feelings about smoking are? [*Asking permission*]

Mr. A: Sure, I'm a smoker and not ashamed of it.

Hygienist: Okay, let me put it another way. On the one hand you want an implant but on the other hand you really like smoking? [*Developing discrepancy*]

Mr. A: Well I've tried to quit before and it wasn't any fun. I'm not sure I could quit if I wanted to.

Hygienist: Okay. You have tried to stop before without any luck so you have the sense that even if you wanted to try to quit again, you don't think you could. It's something that is very hard for you. [*Reflective listening*]

Mr. A: I went through that agony before and it didn't work. I don't want to put myself through that again.

Hygienist: Trying to quit would be too painful and it's not important enough to you to stop right now. [*Rolling with resistance*]

Mr. A: Yes, I enjoy it and haven't had any ill effects from smoking. I'm pretty happy to continue as I am.

Hygienist: Well, other than the possible disadvantage that smoking has to getting this implant, are there any other disadvantages that you see to smoking? [*Evoking change talk*]

Mr. A: Taxes keep going up, and it's an expensive habit, but I'm willing to pay the price. It's one of my luxuries in life.

Hygienist: Anything else? [*Evoking change talk*]

Mr. A: Only one thing—my kids really want me to quit. They are afraid I'm going to end up with something bad, but I haven't had any ill effects from smoking.

Hygienist: So, it sounds like the cost of smoking bothers you, but not too much, but you really haven't had any health problems. Your family worries though. [*Reflective listening to evoke more change talk*]

Mr. A: Yes, that's right.

Hygienist: I have the sense that you're not ready to quit right now, but I'd like to learn more regarding your thoughts about smoking if that's ok with you. Where would you put yourself on a scale of 0 to 10 where 0 is "no motivation at all to quit" and 10 is "very motivated"? I know you aren't a 10, but where would you be. [*Evoking change talk using the motivation ruler*]

Mr. A: Hmmmm. I'd probably put myself at a 2 or 3, somewhere in there.

Hygienist: So, you have some small amount of motivation to quit—what gives you that level of motivation? [*Evoking change talk using the motivation ruler*]

Mr. A: Well I know that smoking isn't good for me, I'm not dumb. And like I said before, my family really wants me to quit. It would be a nice gesture to do that for my family. They really do have my best interests in mind.

Hygienist: You mentioned you know smoking isn't good for you and your family worries about your health. Can you tell me what ill health effects worry you? [*Evoking change talk*]

Mr. A: Sure—cancer and lung disease.

Hygienist: So, there are some disadvantages besides the implant that you've thought about but you still aren't sufficiently motivated to quit. Earlier, though, you mentioned that you weren't sure that you would be able to quit even if you wanted to. [*Reflective listening to explore influence of confidence on motivation*] How motivated would you be if you were more confident that you could quit?

Mr. A: I guess if I knew I could succeed with quitting I'd be more motivated.

Hygienist: If we could help you with improving your confidence in quitting, would you find that helpful? [*Evoking change talk/Assess interest before providing information or advice to support autonomy*]

Mr. A: Yes, it might.

Hygienist: There are several options that we could work on to improve your confidence to quit smoking. We could focus on doing that while also discussing the feasibility of the implant. Do you think you might be interested in pursuing this? [*Evoke commitment/change talk while supporting autonomy*]

Mr. A: Sure. I guess I don't have anything to lose.

Motivational Interviewing: Example 3

Ms. S is a 35-year-old administrative assistant at the local community college. She has come to the dental office as a new patient and desires tooth whitening for her "yellowed teeth." During the routine dental evaluation, no caries are found; however, there is generalized moderate gingival inflammation with slight bone loss in the interproximal posterior regions. The patient reports that she had a partial "deep cleaning" 2 years ago at her previous dentist but had really sensitive teeth and didn't return for completing treatment.

Hygienist: Hello Ms. S. What brings you to our office? [*Open-ended questioning*]

Ms. S: My friend recently came to see you to have his teeth whitened and they look terrific. I haven't been happy with the color of my teeth for a long time, so I thought you could make my smile whiter, too.

Hygienist: Okay. Can we first spend a few minutes talking about your mouth and oral health? [*Getting permission*]

Ms. S: Sure, that's fine.

Hygienist: Good. Can you tell me a little about problems you have had and how taking care of your teeth fits into your regular day's events? [*Open-ended questioning*]

Ms. S: I have really healthy teeth and have never had any cavities. I'm really not happy with the yellow color of my teeth and want to do something about that.

Hygienist: Anything else?

Ms. S: Well, the last dentist I went to said I had some gum disease. My gums have always bled when I brush, but they made a big deal out of it. I had to see the hygienist for a deep cleaning, but my teeth got so sensitive afterward, I didn't go back.

Hygienist: Tell me a little more about your gum treatment. [*Open-ended questioning*]

Ms. S: The dentist and hygienist told me I had to have that deep cleaning or I could lose my teeth. Everyone in my family has gum disease and they still have most of their teeth. I must have inherited it. I'm really only interested in tooth whitening. I don't want to have any more deep cleanings.

Hygienist: It sounds like the deep cleaning you had was unpleasant and your gum disease is not very important to you. [*Reflective listening*]

Ms. S: I didn't say it wasn't important—I just want my teeth to look better. My teeth were so sensitive after having half of my mouth deep cleaned, I could barely drink anything with ice in it. If that is what it takes to have healthy gums, I'm happy just as I am.

Hygienist: The effects of the deep cleaning treatment were so bad that you don't want to go through it again even if it means not having healthy gums. [*Responding to sustain talk with reflective listening*]

Ms. S: It's not that it isn't important. I don't want to lose my teeth, but I also don't want all of that sensitivity. I was thinking that getting my teeth whitened will make me look better and wouldn't hurt as much.

Hygienist: Avoiding pain is important to you. [*Responding to sustain talk with reflective listening*]

Ms. S: Yeah, I guess I sound pretty wimpy. My teeth are important but if I have to be in pain for a long time afterward it's not worth it to me.

Hygienist: You want healthy teeth and if you could get your teeth whitened and gum disease treated without a lot of pain afterward, it might be worth doing. [*Reflective listening*]

Ms. S: If I knew that I could get my gums healthy and not have to go through what I did before, I might be willing to discuss gum treatments. Is that possible?

Hygienist: There are several things that have worked for others to reduce the temperature sensitivity after deep cleaning. Are you interested in learning a little bit more? [*Ask permission before providing information*]

Ms. S: I might be.

Hygienist: All right, let me give you some information and we can talk about strategies for controlling the sensitivity after treatment. We can also talk about a plan for whitening your teeth once we get the gum disease under control. How would that meet your need for appearance and health? [*Support autonomy*]

Chapter Summary Statement

A major challenge for all oral health care practitioners is working with patients to foster positive change to behavior. Motivational interviewing (MI) provides an empirically supported collaborative approach to the challenges of counseling a patient to health behavior change, rather than compelling or pressuring the patient to make changes. It rests on a foundation of partnership, acceptance, and compassion and uses specific methods such as open-ended questions and reflective listening to evoke patients' own reasons for change. Use of this patient-centered approach to advance changes in behavior can enhance the quality of encounters between patients and health care providers by fostering greater patient motivation for change and strengthening the patient–provider relationship. In the end, the MI approach—as part of the shared decision-making process—can be the foundation to building upon a trusting and lasting relationship between the patient and the health care provider.

Section 4

Focus on Patients

Clinical Patient Care

CASE 1

Refer to the case of Mrs. J, the 50-year-old attorney, to answer the following questions:

1. Why does the hygienist begin with the question "Can you tell me a little about ... why you are here today?"
2. Describe Mrs. J's ambivalence regarding behavior change (i.e., what cons *and* pros for change does *she* see?).
3. What examples of "change talk" can you identify in the dialogue?

CASE 2

Refer to the case of Mr. A, the 58-year-old banker, to answer the following questions:

1. When the conversation first turns to smoking, the hygienist asks Mr. A, "Are you sufficiently motivated about the implant to consider stopping smoking?" This is labeled as an example of developing discrepancy. What discrepancy does this refer to, and how is this meant to foster motivation for change?
2. What is Mr. A's ambivalence regarding behavior change (i.e., what cons *and* pros for change does *he* see?)?
3. Mr. A provided lots of change talk in the dialogue. What is the key barrier for change that, if the hygienist can help address it, will significantly increase his motivation?

CASE 3

Using the information in the case of Ms. S, the 35-year-old administrative assistant, try the following:

1. With a classmate, take turns assuming the role of Ms. S and the hygienist. Practice asking an open-ended question to begin the visit followed by a reflection of Ms. S's response.
2. Ms. S could benefit from information on how to get her gums healthy while reducing sensitivity. With a classmate, practice using the three steps—elicit–provide–elicit—to give her some relevant information.

Ethical Dilemma

You have been working as a hygienist in the same periodontal practice for the last 10 years and are quite happy there. There are four dentists in the group, so you have a very full schedule, which allows you to work four 10-hour days. The practice is very flexible and generous with their resources and benefits. They also pay for continuing education (CE) courses for all staff members. Last year, you had the opportunity to participate in numerous informative CE courses on the topics of nonsurgical therapy, periodontal diagnosis, and implant maintenance. However, with respect to implant surgery, you are still not comfortable with this topic due to the lack of surgical experience.

Your next patient, Richie, is a 63-year-old real estate agent, who presents today for his periodontal maintenance recall visit. In reviewing his dental history, Richie states that "I had my upper right front tooth pulled last month by an oral surgeon> "(patient points to the missing maxillary right central incisor area). Since the extraction, Richie states: "I've done my homework by using the internet to look up implants, and now I have questions that I'd like you to help me with."

While you want to be a part of the treatment decision making process, you feel very uncomfortable discussing this topic because you do not know much about surgical implantology.

1. What ethical principles are in conflict in this dilemma?
2. What is the best way for you to handle this ethical dilemma?

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CHAPTER 24

Best Practices for Periodontal Care

- Section 1 What Is Best Practice?
- Section 2 Role of Evidence-Based Decision-Making in Best Practice
- Section 3 Finding Clinically Relevant Information
  - Clinical Case Example
- Section 4 Lifelong Learning Skills for Best Practice
- Section 5 Focus on Patients
  - Clinical Patient Care
  - Evidence in Action
  - Ethical Dilemma

CLINICAL APPLICATION

All clinicians face the daunting task of ensuring that they are providing the best possible care for their patients. Dental hygienists must commit to staying updated in their knowledge about periodontal diseases and the care patients with these diseases require. Faced with the fact that there is continuous publication of scientific information about these topics, sifting through information to find the most appropriate and applicable is both time-consuming and challenging. This chapter outlines strategies that any dental hygienist (or any member of the dental team) can employ to ensure that he or she is indeed able to provide patient care that is based upon the best scientific evidence available.

LEARNING OBJECTIVES

- Summarize how the explosion of knowledge is impacting practitioners and patients.
- Identify the three components of evidence-based decision-making.
- Discuss the benefits and limitations of experience.
- Describe the role of the patient in the evidence-based model.
- List locations for accessing systematic reviews.
- Explain the difference between a peer-reviewed journal and trade magazine.
- State three desired outcomes from attending continuing education courses.
- Formulate a question using the PICO process.

KEY TERMS

Best practice
Association
Causal factor
Evidence-based practice
Confirmation bias
Best evidence
Levels of evidence
Systematic review
PICO process
Databases
MEDLINE
PubMed
Cochrane Oral Health database
Peer-reviewed journals

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Section 1

What Is Best Practice?

Providing the best possible care to patients is the foremost goal of all dental health care providers. Yet, it is generally acknowledged that periodontal care may vary from office to office and even from region to region. As new procedures and techniques become available, fully committed hygienists must regularly update and adapt their strategies to providing patient care. The approach, known as "best practice," is an important tool in helping hygienists provide high-quality care to their patients.

1. **Overview of the Concept of Best Practice**
   a. **Definition. Best practice** refers to treatment that is accepted by experts as proper treatment for a certain type of disease and is widely used by health care providers. It may also be called the standard of care or standard therapy.[1]
   b. **Goals and Considerations**
      1. The goal of best practice is the use of concepts, interventions, and treatments that are known to promote a higher quality of care.
         a. The outcome should be measurable, such as a reduction in probing depths.
         b. The outcome should be reproducible. For example, if a technique produces a certain result on one patient, it is reasonable to expect a similar outcome when the technique is used with other patients.
      2. Best practice is derived from evidence-based care. It is the process of using the best available evidence in the delivery of the standard of care.[1]
2. **Circumstances That Prompted the Best Practice Approach to Patient Care**
   a. **Direct Access to Rapidly Emerging Clinical Research Information**
      1. **Volume of Information**
         a. Studies on new techniques, tests, procedures, and products for periodontal care are emerging at an astonishing rate. Hundreds of articles are published in dental journals each year.
         b. In addition to the information in dental journals, relevant articles are published each year in medical and specialty journals. An example of articles in other disciplines that are relevant to periodontal health are those on the topic of the oral–systemic link; important research on this topic can be found in journals such the *New England Journal of Medicine* or *Diabetes Care*.
      2. **Direct Access to Information**
         a. In the past, dental health care providers relied on what they learned in school and the advice of recognized experts to determine how to provide care. Patients had little-to-no input into this process. Knowledge of new or cutting-edge research was limited to a few practitioners with access to an educational or health care database.
         b. With digital media, clinicians and patients have instant access to the results of federally funded clinical trials on treatment methods, equipment, and materials. PubMed, a gateway to more than 24 million research citations, can be accessed by anyone for free.
         c. There are now more than 10,000 open access scholarly journals. Open access means that the scientific studies and papers are open to any reader—health care provider or patient—without any financial, legal, or technical barriers.
         d. Practicing dental hygienists should strive to remain current with new techniques, devices, and materials and make judgments about whether something newer will result in improvements in periodontal care. Research has shown that there is an inverse relationship between the number of years in practice and the quality of care provided. This means the longer you are in practice, the more at-risk you are for providing a lower quality of care.[2]
   b. **Active Patient Role in Decision-Making**
      1. Before the widespread use of information technology, patients depended on the expertise of a health care provider for advice, and in most cases accepted that advice without question.
      2. Many of today's patients expect to be a partner in the decision-making process about their own periodontal care. Patients may arrive at the dental office with information downloaded from the internet.

Practitioners with good communication skills who engage patients in the decision-making process increase the likelihood that patients will adhere to recommended treatments or care.[3]

3. **Ability to Interpret the Literature**
    a. **Not All Studies Are Significant to Clinical Care.** Even though hundreds of studies are published yearly, not all are valid and even fewer are significant enough on their own to merit a change in clinical care.
        1. The merit or weight of study is influenced by its design. For example, a randomized clinical trial offers a higher level of evidence than a case series.
        2. No study is completely free of bias. Reputable journals require investigators to declare a conflict of interest and disclose corporate financial support for studies.
        3. Many studies either are not designed to provide an answer to the needs of the clinician or provide results that are too weak to merit implementation.
    b. **New Does Not Necessarily Mean Better**
        1. New treatments and products need to demonstrate consistent superiority to established methods.
        2. Some new products and therapies are also significantly costlier to implement, and these costs are ultimately passed down to patients.
    c. **Associations Are Not the Same as Cause and Effect**
        1. An **association** is a relationship between an exposure and a disease that implies the exposure might cause the disease.[4]
        2. A **causal factor** (causality) is an event or condition that leads to an occurrence of a disease.[4] An example of a causal factor is exposure to the bacterium called *Mycobacterium tuberculosis*. Exposure to this bacterium can cause an individual to develop the infectious disease tuberculosis (TB).
        3. Finding an association between an event and a disease does not make it causal.
            a. Over the past several years, many studies have looked at the relationship between periodontal disease and a host of systemic conditions.
            b. For instance, while many studies do show that periodontal disease is associated with cardiovascular disease, at the time of this writing, periodontal disease cannot be said to be a causal factor for cardiovascular disease.[5]

Section 2

Role of Evidence-Based Decision-Making in Best Practice

1. **Introduction to Evidence-Based Decision-Making.** Knowledge of the most recent and relevant evidence is the foundation for best practice. The American Dental Hygienists' Association (ADHA) advocates evidence-based, patient/client-centered dental hygiene practice.
    a. **Definition.** The ADHA defines **evidence-based practice** as the conscientious, explicit, and judicious use of current best evidence in making decisions about the care of individual patients. The practice of evidence-based dental hygiene requires the integration of individual clinical expertise and patient preferences with the best available external clinical evidence from systematic research.[6]
    b. **The Need for Evidence-Based Decision-Making**
        1. Using evidence-based care can help practitioners reduce knowledge gaps between what was "learned in school" and new or emerging treatments supported by best evidence.[7]
        2. The translation of research findings into sustainable improvements in treatment outcomes is challenging. The Agency for Healthcare Research and Quality (AHRQ) notes that it can take up to 20 years for original research to become part of routine clinical practice.[8]
        3. There are barriers to the implementation of evidence-based care. Staying current requires a time commitment. Having access to the best evidence may require a journal subscription or professional association membership to access peer-reviewed journals and/or clinical standards.[9]
        4. New evidence that conflicts with the practitioner's views, beliefs, attitudes, and experiences can also be a barrier to implementing evidence-based care.[7,9] Dental hygienists may encounter challenges with

this when trying to implement new evidence-based interventions or protocols that are not aligned with the dentist's or practice's philosophy.

c. **About Evidence-Based Decision-Making**
   1. Evidence-based decision-making emerged from the work of Dr. David Sackett and others at McMaster University in Ontario, Canada. Dr. Sackett is credited with crafting the term. Another driver of evidence-based decision-making is Archie Cochrane, a Scottish physician. Dr. Cochrane championed the use of randomized clinical trials to evaluate medical treatments. The Cochrane Collaboration is named after him.[10,11]
   2. Evidence-based decision-making includes three foundational elements: (1) incorporation of the best scientific evidence with (2) the health care provider's clinical expertise and judgment and (3) patient's preferences and values. **Figure 24-1** illustrates these three foundational elements of evidence-based health care. **Box 24-1** provides an example of evidence-based practice.

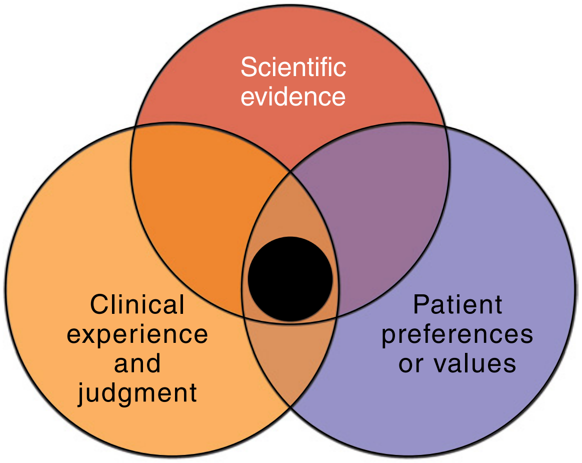
Scientific evidence
Clinical experience and judgment
Patient preferences or values

Figure 24-1 The Three Foundational Elements of Evidence-Based Health Care. Evidence-based care has three equal components: scientific evidence, clinical experience, and patient preferences or values.

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Box 24-1 Evidence-Based Practice: An Example

Evidence-based practice is built on information obtained from research. For example, perhaps a dental hygienist was taught in school that ultrasonic instrumentation should be used sparingly and only for the removal of large supragingival calculus deposits. In addition, the hygienist learned in school that hand instruments produce the best results for periodontal instrumentation.

After reading current research on ultrasonic instrumentation, the hygienist learns that modern slim-tipped ultrasonic instruments can be used subgingivally and that a combination of ultrasonic and hand instrumentation leads to excellent results.

This evidence motivates the dental hygienist to attend a continuing education course on ultrasonic instrumentation and to incorporate ultrasonic instrumentation in treating patients with periodontitis.

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   3. Evidence is not meant to replace experience or clinical skills. The addition of evidence to decision-making brings balance to the process. It helps close the gap between "what we do" and science.[7,9] It can enhance patient care and outcomes.[9]

d. **Foundational Elements of Evidence-Based Decision-Making**
   1. Evidence-based care recognizes that essential skills are needed to engage in evidence-based decision-making and obtain the best health outcomes.[10,11]
      a. Over time, health care providers gain clinical expertise by engaging in clinical experiences (i.e., treating patients and observing the results).

   b. Patients' preferences may be the result of many factors, including past dental experiences, current medical and dental status, perceived needs, health values, and economic considerations.
   c. The most challenging essential skill for a health care professional to develop is the ability to assess "evidence."
2. Clinical experience is both valuable and limiting. It signifies the ability of a clinician to grow in skill and knowledge through experience.
   a. Experience helps the practitioner make thoughtful clinical judgments about the applicability of research findings to specific patient situations. Yet, all patients are different; they may present with complicated or complex medical and dental histories.
   b. Experience is valuable when it is seen as an always evolving learning process. Ideally, clinicians learn from their clinical experiences to broaden their knowledge base so that better treatment options, supported by the most-up-to-date evidenced-based practice, can be offered to the patient.
   c. Experience can be limiting. The limitation is that not all individuals are able to learn and grow from experience. To acquire "practical wisdom," the clinician needs to learn how to be reflective and analyze his or her own performance.
      1. There is a human tendency to look for or interpret information that confirms our beliefs. This tendency is called **confirmation bias**.
      2. Confirmation bias can lead practitioners to misinterpret information based on beliefs, positive or negative, about a treatment or device.
3. Patient preference or values are important considerations in treatment selection. If due consideration is not given to the individual patient's preferences, values, and concerns, as well as their unique clinical circumstances, the likelihood of the patient fully accepting the clinician's recommendation diminishes.
   a. It is the dental hygienist's responsibility to understand the evidence and its implications for periodontal treatment and communicate it effectively to a patient. Ultimately, it will be the patient who chooses which therapy he or she prefers.
   b. While patients have choices, practitioners have the responsibility to recommend treatment that is safe, evidence-based, clinically sound, high quality, and equitable.[5] In helping a patient decide which periodontal treatment is right for him or her, several elements should be discussed, including:
      1. The evidence about a particular treatment option.
      2. The treatment of choice based on sound evidence.
      3. All possible evidence-based treatment alternatives.
      4. The risks of no treatment at all.
   c. In addition to the efficacy of a proposed treatment, a patient may place equal weight on other aspects of treatment, such as:
      1. **Cost.** Patients usually are concerned about what a treatment will cost. In addition, patients decide if the treatment has benefits that they perceive as being worth the cost.
      2. **Pain.** Assurances about pain control and management help lessen these concerns.
      3. **Time lost from work.** Different jobs and work environments have varying levels of flexibility in allowing employees time off for health-related matters.
      4. **Impact on family.** Caregivers of young children or elderly family members may feel that they do not have the time to devote to periodontal treatment. Individuals with chronic health problems may believe that periodontal care is no longer a priority.
      5. **Insurance benefits.** A practice reality is that patients will sometimes choose care based on what insurance will pay for versus the full treatment recommendation. However, it should be communicated to the patient that insurance coverage is not the same as the standard of care.

2. **Evaluation of Scientific Evidence.** All scientific evidence is not created equal.
   a. **Levels of Evidence. Best evidence** is the highest level of evidence available for a specific clinical question.
      1. **Levels of evidence** is a ranking system used in evidence-based care to describe the strength of the results measured in a clinical trial or research study. In simplistic terms, one way of looking at levels of evidence is as follows (the higher the level, the better the quality; the lower the level, the greater the bias). **Figure 24-2** illustrates the levels of evidence.

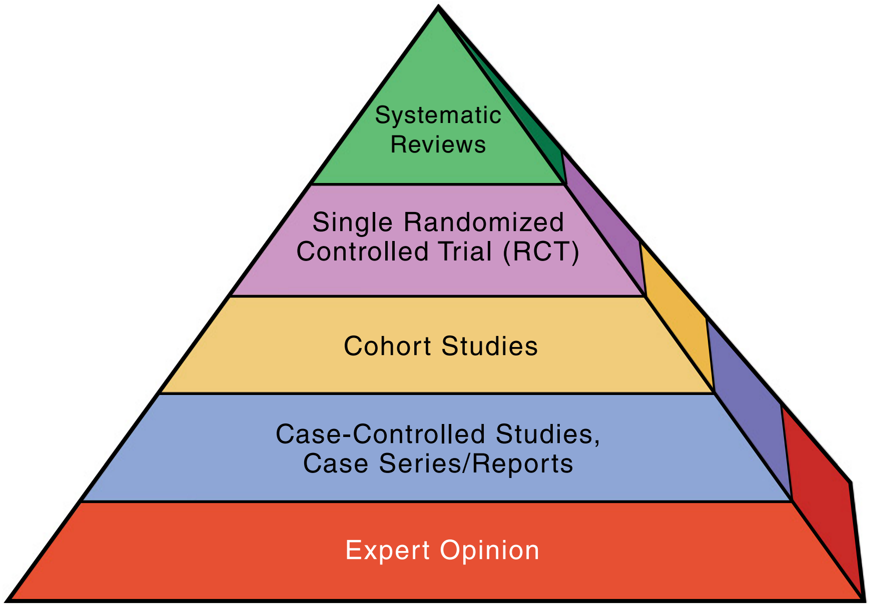
Systematic Reviews
Single Randomized Controlled Trial (RCT)
Cohort Studies
Case-Controlled Studies, Case Series/Reports
Expert Opinion

Figure 24-2 Levels of Evidence. The importance or merit of a research study usually is evaluated through its design. Systematic reviews and randomized controlled trials represent the best levels of evidence. Case reports and expert opinion are the lowest levels of evidence.

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2. Based on a hierarchy of levels of evidence, systematic reviews constitute the highest level of current best evidence; expert opinion is lower-level evidence.
3. The highest level of evidence available represents the current best evidence for a specific clinical question.

b. **Systematic Reviews: The Gold Standard in Evidence-Based Care**
   1. A **systematic review** is research methodology that systematically identifies all relevant research around a clinical question, critically appraises those studies, evaluates the findings, and establishes conclusions.[12]
      a. The systematic review process was designed to reduce bias and objectivity.[13]
      b. Strict protocols are used to ensure the inclusion of all clinically relevant studies.[13]
      c. When conducted well, a systematic review should provide the best possible estimate of any true effect.[13]
   2. Systematic reviews are, by their very nature, efficient. As an information management tool, they provide a way of coping with large volumes of data in a concise and manageable form.
      a. With more than two million articles published in medical and dental journals annually, it is impossible for one health care provider to read and utilize all the new information.
      b. Systematic reviews of randomized clinical trials represent one of the highest levels of evidence.[12,13]
      c. Systematic reviews also facilitate the development of clinical practice guidelines by bringing together all that is known about a given topic in a nonbiased manner. This provides useful mechanisms for bringing research to practice.[8]
   3. Because of the emphasis on evidence-based care, there are more systematic reviews conducted in dentistry than ever before.
   4. The systematic review makes incorporating evidence-based care easier. In the past, practitioners were encouraged to do their own searching for research. Because most busy practitioners do not have the time or expertise to do this, the systematic review fills this gap.

Section 3

Finding Clinically Relevant Information

Today, all that is required to have access to research information is a computer with Internet access. Determining which studies are of the highest evidence and the most clinically relevant for a particular need is much more challenging. The most comprehensive process for finding and critically evaluating clinical evidence involves a five-step process. This five-step process is summarized in **Box 24-2**. Steps 1 through 3 of this five-step process are discussed below.

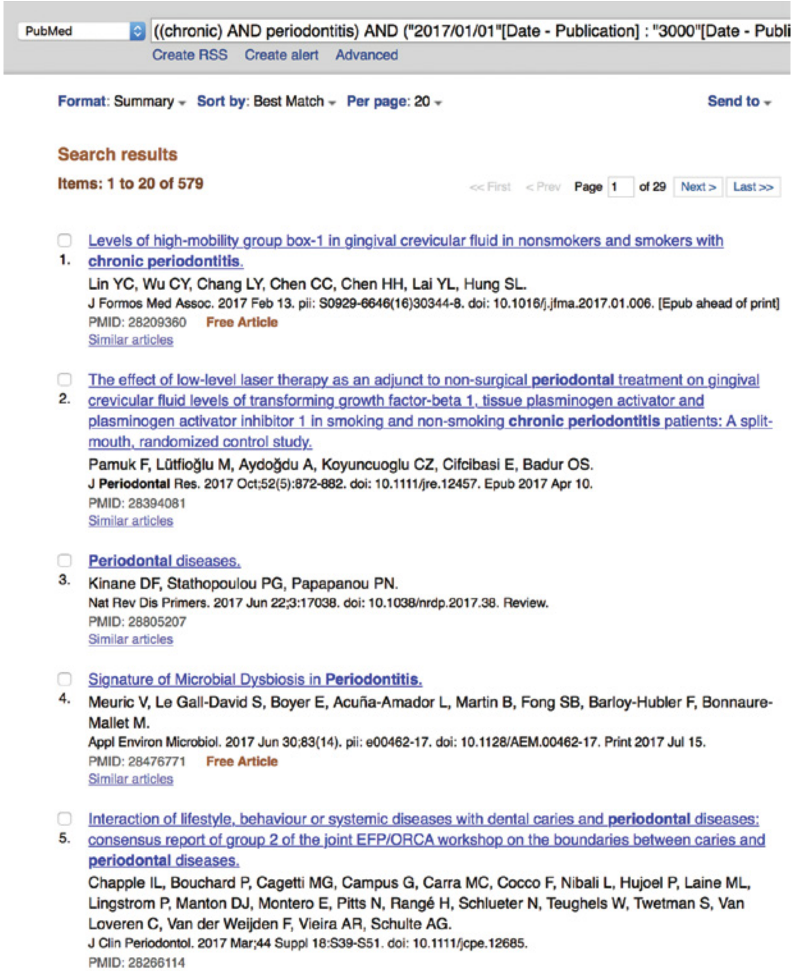
Box 24-2 Five-Step Process for Finding and Evaluating Clinical Evidence

1. Develop an answerable clinical question from a patient problem or need.
2. Conduct a computerized search to locate the best available evidence.
3. Critically appraise the evidence for clinical applicability.
4. Apply the results to your particular clinical situation.
5. Evaluate the process and the results.

Data from Forrest JL, Overman P. Keeping current: a commitment to patient care excellence through evidence-based practice. *J Dent Hyg*. 2013;87 Suppl 1:33–40.

1. **Developing an Answerable Question.** A clinical question may develop from questions that arise relative to patient care or from an area in which the hygienist wants updated knowledge. In order to find the best information to help patients, it is fundamental to learn how to ask the "right questions." This is more challenging than it seems. It involves converting problems into answerable questions.
    a. **Use Four Components to Structure the Question.** The structure for asking a clear and focused question entails four critical components, known as the **PICO process**.[10–12] The PICO process involves the combination of four separate components to form an answerable question: patient, intervention, comparison, and outcome.
        1. **P (Patient or Problem).** An example of the P component might be "A periodontal maintenance patient with bleeding and gingivitis."
        2. **I (Intervention)**
            a. An intervention is a specific diagnostic test, treatment, adjunctive therapy, medication, product, or clinical procedure.
            b. An example of an intervention being questioned is "brushing and daily home irrigation."
        3. **C (Comparison)**
            a. Identifies the specific alternative therapy or device that you wish to compare to the main intervention.
            b. An example of the "C" segment of the question is "compared to brushing and flossing."
        4. **O (Outcome)**
            a. Identifies the measurable outcome you plan to accomplish, improve, or influence.
            b. An example of the "O" segment of the question is "reduce gingivitis and bleeding within 4 weeks."
    b. **Formulate the Question.** Once each of the PICO components has been determined, the clinician combines them into an answerable question. Using the above examples, the question would read: "For a periodontal maintenance patient with bleeding and gingivitis, will brushing and daily home irrigation OR brushing and flossing provide a better reduction in bleeding and gingivitis within 4 weeks?"
2. **Conduct a Computerized Search to Find the Best Evidence**
    a. **Databases**
        1. The most efficient way to go about finding relevant research is to use an online index of published articles, such as PubMed, MEDLINE, or the Cumulative Index of Nursing and Allied Health Literature (CINAHL).
        2. These indexes—known as **databases**—list all articles published in each period of time by journals in a particular profession or group of professions.

3. **MEDLINE** is the U.S. National Library of Medicine's (NLM) premier database. It contains more than 33 million references to journal articles in the life sciences with a concentration on biomedicine. MEDLINE enables quick access to locate relevant clinical evidence in the published dental/periodontal literature.
    a. A distinctive feature of MEDLINE is that the records are indexed with NLM Medical Subject Headings (MeSH).
    b. Anyone can access MEDLINE for free using **PubMed**—a gateway hosted by the National Library of Medicine (**Figure 24-3**).



((chronic) AND periodontitis) AND ("2017/01/01"[Date - Publication] : "3000"[Date - Publi

Search results

Items: 1 to 20 of 579

☐ 1. Levels of high-mobility group box-1 in gingival crevicular fluid in nonsmokers and smokers with **chronic periodontitis**.
Lin YC, Wu CY, Chang LY, Chen CC, Chen HH, Lai YL, Hung SL.
J Formos Med Assoc. 2017 Feb 13. pii: S0929-6646(16)30344-8. doi: 10.1016/j.jfma.2017.01.006. [Epub ahead of print]
PMID: 28209360 Free Article
Similar articles

☐ 2. The effect of low-level laser therapy as an adjunct to non-surgical **periodontal** treatment on gingival crevicular fluid levels of transforming growth factor-beta 1, tissue plasminogen activator and plasminogen activator inhibitor 1 in smoking and non-smoking **chronic periodontitis** patients: A split-mouth, randomized control study.
Pamuk F, Lütfioğlu M, Aydoğdu A, Koyuncuoglu CZ, Cifcibasi E, Badur OS.
J **Periodontal** Res. 2017 Oct;52(5):872-882. doi: 10.1111/jre.12457. Epub 2017 Apr 10.
PMID: 28394081
Similar articles

☐ 3. **Periodontal** diseases.
Kinane DF, Stathopoulou PG, Papapanou PN.
Nat Rev Dis Primers. 2017 Jun 22;3:17038. doi: 10.1038/nrdp.2017.38. Review.
PMID: 28805207
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☐ 4. Signature of Microbial Dysbiosis in **Periodontitis**.
Meuric V, Le Gall-David S, Boyer E, Acuña-Amador L, Martin B, Fong SB, Barloy-Hubler F, Bonnaure-Mallet M.
Appl Environ Microbiol. 2017 Jun 30;83(14). pii: e00462-17. doi: 10.1128/AEM.00462-17. Print 2017 Jul 15.
PMID: 28476771 Free Article
Similar articles

☐ 5. Interaction of lifestyle, behaviour or systemic diseases with dental caries and **periodontal** diseases: consensus report of group 2 of the joint EFP/ORCA workshop on the boundaries between caries and **periodontal** diseases.
Chapple IL, Bouchard P, Cagetti MG, Campus G, Carra MC, Cocco F, Nibali L, Hujoel P, Laine ML, Lingstrom P, Manton DJ, Montero E, Pitts N, Rangé H, Schlueter N, Teughels W, Twetman S, Van Loveren C, Van der Weijden F, Vieira AR, Schulte AG.
J Clin Periodontol. 2017 Mar;44 Suppl 18:S39-S51. doi: 10.1111/jcpe.12685.
PMID: 28266114

Figure 24-3 PubMed Website. PubMed search results for the topic "chronic periodontitis."

Courtesy of the U.S. National Library of Medicine, Bethesda, MD.

    c. **Systematic Reviews.** Many health care providers do not have the time or expertise needed to do their own systematic reviews of a question. Fortunately, there are numerous trustworthy resources for busy practitioners who want to implement high-quality science into patient care.
4. **The Cochrane Oral Health Database of Systematic Reviews**
    a. The **Cochrane Oral Health database** was established in 1993 by a British epidemiologist who recognized that ready access to systematic reviews of available evidence would facilitate better-informed decisions by health care providers.
    b. The Cochrane Database of Systematic Reviews includes systematic reviews of health care interventions that are produced and disseminated by Cochrane Oral Health—a global nonprofit organization.
        1. The Cochrane Library is published online. Abstracts of reviews are free.
        2. Also, many health science libraries subscribe to the Cochrane databases so that faculty and students have online access.

   c. The Cochrane Review group relevant to periodontics is the Oral Health Review Group. An example of a systematic review conducted by the Oral Health Group is on the topic of psychological interventions to improve adherence to oral hygiene instructions in adults with periodontal diseases.
   d. A complete listing of topics and abstracts can be accessed at http://oralhealth.cochrane.org.
5. **The PubMed Clinical Query: The National Library of Medicine**
   a. One feature of the MEDLINE database is the PubMed Clinical Query, which provides specialized searches using an evidence-based filter.
   b. An online tutorial for the PubMed Clinical Query tool can be accessed online at http://www.nlm.nih.gov/bsd/disted/pubmedtutorial/020_570.html.
6. **Systematic Reviews by Professional Organizations.** Many professional organizations are developing systematic reviews. The American Dental Association (ADA) home for scientific findings and clinical practice guidelines is the ADA Science & Research Institute (https://www.ada.org/resources/research/science-and-research-institute).
7. **Systematic Reviews in Evidence-Based Journals**
   a. Evidence-based journals publish summaries of valid research studies to simplify the evidence-based process for dental health care providers.
   b. For example, *The Journal of Evidence-Based Dental Practice* scans the top dental journals and a panel reviews the selected articles for clinical relevance to practice.
   c. *Evidence-Based Dentistry* also reviews articles as well as published systematic reviews.
8. **Appraisal of the Evidence for Clinical Applicability.** In medicine, the use of point-of-care electronic databases and algorithms are emerging to help practitioners evaluate and implement evidence-based decision-making.[11]
   a. The use of electronic records is integral to this process because it allows individual patient characteristics to be automatically linked to the best evidence.[11]
   b. **Table 24-1** outlines preappraised evidence resources, including interactive drug databases, that are a key part of clinical care decision support systems.[11]

Table 24-1 Preappraised Evidence Resources

Clinical Decision Support Systems: Interactive Drug Databases	
Clinical Key	https://www.clinicalkey.com/#!/
Comprehensive Drug Database; Interactions	http://www.lexi.com
Integrative Medicine with Evidence-Based Grading System	http://www.naturalstandard.com
Up-to-Date	http://www.uptodate.com
Clinical Practice Guidelines	
American Academy of Pediatric Dentistry	https://www.aapd.org/research/oral-health-policies--recommendations/
ADA Science & Research Institute	https://www.ada.org/resources/research/science-and-research-institute/evidence-based-dental-research
American Heart Association	https://www.ahajournals.org/doi/10.1161/CIR.0000000000000969
Centers for Disease Control and Prevention/Infection Control for Healthcare workers	https://www.cdc.gov/oralhealth/infectioncontrol/statement-COVID.html
PubMed (article type limited to "Practice Guideline")	http://pubmed.gov

(*continues*)

Table 24-1 Preappraised Evidence Resources *(continued)*

Systemic Reviews	
ADA Science & Research Institute	https://www.aapd.org/research/oral-health-policies--recommendations/
Cochrane Oral Health Library	https://oralhealth.cochrane.org/
PubMed (article filter limit to "systemic review")	http://pubmed.gov
Evidence-Based Dentistry	http://www.nature.com/ebd/index.html
Journal of Evidence-Based Dental Practice	http://www.jebdp.com
Original Studies: Individual Research Studies (Original Studies, Not Preappraised)	
PubMed (limit to "RCT" or "clinical trial")	http://pubmed.gov
Journal publications (e.g., *Journal of Dental Hygiene*, *International Journal of Dental Hygiene*, dental specialty groups, etc.)	http://www.adha.org/publications/index.html https://jada.ada.org http://www.ifdh.org/publications.html

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3. **Using the PICO Process to Find Clinically Relevant Information.** Figure 24-4 shows how the PICO process can be incorporated in a three-step approach to finding clinically relevant information.

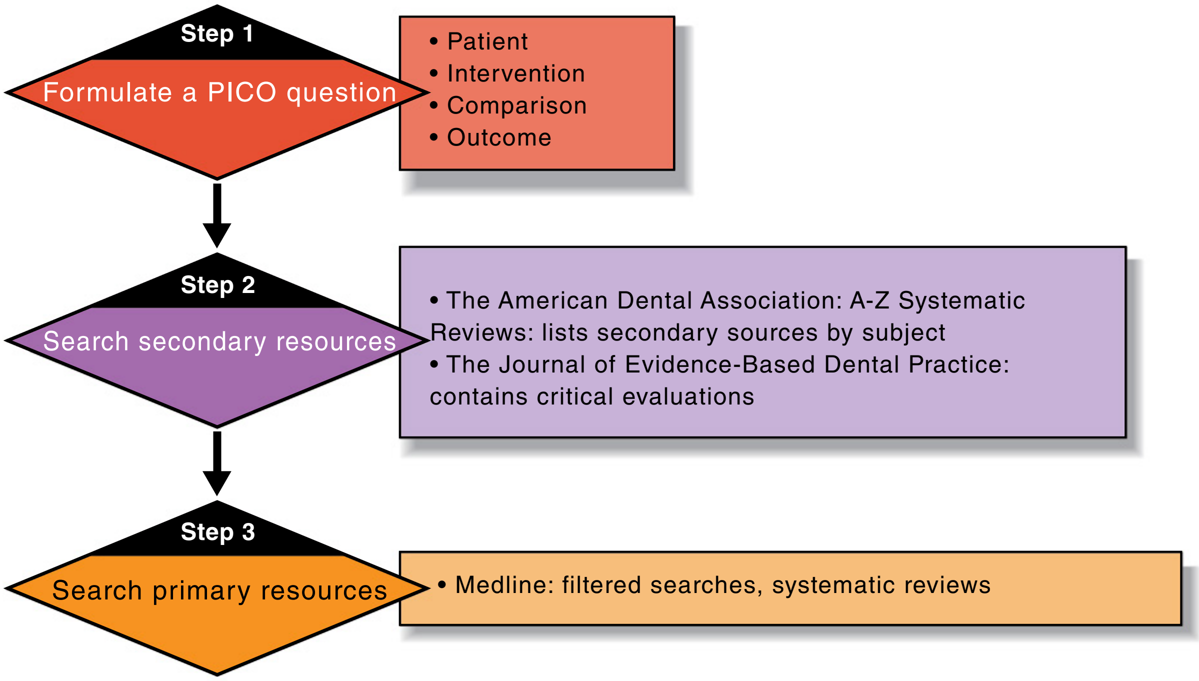
Step 1
Formulate a PICO question
• Patient
• Intervention
• Comparison
• Outcome
Step 2
Search secondary resources
• The American Dental Association: A-Z Systematic Reviews: lists secondary sources by subject
• The Journal of Evidence-Based Dental Practice: contains critical evaluations
Step 3
Search primary resources
• Medline: filtered searches, systematic reviews

Figure 24-4 Strategy for Finding Clinically Relevant Evidence. The Centre for Evidence-Based Medicine recommends a straightforward approach based on (1) formulating a PICO question, (2) searching evaluated (secondary) resources, and (3) then examining primary text documents.

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Clinical Case Example

A dental hygienist used the following steps to find evidence-based clinically relevant information.

1. **Step 1: Formulate a PICO Question**
   - **Patient:** adult female patient with generalized periodontitis; new patient in the dental office
   - **Intervention:** periodontal instrumentation

- **Comparison:** full-mouth disinfection (FMD) versus full-mouth scaling (FMS) versus quadrant instrumentation
- **Outcome:** resolution of inflammation
- **Question:** "For a patient with chronic periodontitis, will full-mouth disinfection (a nonsurgical periodontal protocol involving full-mouth subgingival instrumentation of all quadrants in a 24 hour period *with* adjunctive antiseptics) OR full-mouth scaling (a nonsurgical periodontal protocol involving full-mouth subgingival instrumentation within a 24 hour period *without* the use of antiseptics) provide better therapeutic results compared to conventional quadrant subgingival instrumentation (a nonsurgical periodontal protocol involving a series of multiple instrumentation visits at least one week apart)?"

2. **Step 2: Search Online Evidence-Based Sources**
   - Cochrane Database of Systematic Reviews at https://www.cochranelibrary.com/
   - PubMed at http://pubmed.gov
3. In this instance, the dental hygienist presented both options to the patient, explaining that both treatment options are equally effective.
4. The patient chose full-mouth debridement because it would be less disruptive for her to be away from work for 1 day rather than four shorter appointments over a period of several weeks.

Section 4

Lifelong Learning Skills for Best Practice

One of the most challenging aspects of best practice involves self-evaluation. Practitioners continually need to think about whether the care they are providing is still the best-level care. The dental hygienist should think about several questions on a regular basis to assess whether he or she is providing best-level care.

1. **How sure am I that what I do is right?**
   a. Do I know where to access systematic reviews? Do I keep up with journal reading?
      1. **Peer-Reviewed Journals. Peer-reviewed journals** (also called refereed journals) use a panel of experts to review research articles for study design, statistics, and conclusions.
         a. Peer-reviewed journals are good sources for randomized clinical trials and learning about new research findings. Sometimes they will publish systematic reviews.
         b. Subscriptions to peer-reviewed journals can be expensive. However, some highly ranked peer-reviewed journals from professional associations have begun allowing free access to some studies if they are groundbreaking and deemed highly clinically relevant. In addition, many reputable open access journals are available.
         c. Peer-reviewed journals are a good source of higher levels of evidence—systematic reviews and randomized clinical trials.
      2. **Practice or Trade Magazines**
         a. Can be commercial in nature.
         b. May or may not be peer-reviewed; generally provide more of the "expert" opinion.
         c. May or may not be supported with references.
         d. Vary widely in quality.
         e. Provide the lowest quality of evidence.
      3. **Textbooks**
         a. Provide a broad overview of a subject.
         b. May not provide specifics on the research.
         c. May be dated because of the amount of time involved in writing and publishing a textbook; always check the publication date.
   b. **Do I attend continuing education courses? How do I decide which courses to attend?**
      1. **Content:** Is the subject matter something you like or something that you need? It is important to take the time to evaluate learning/practice needs. Conferring with coworkers or your employer can facilitate more objective choices.

    2. **Speaker:** Is the individual an expert, a facilitator, or both? A well-rounded speaker will provide information on the latest research findings along with providing some practical advice based on experience.
    3. **Outcomes:** A well-rounded continuing education course will do three things[13]:
        a. **Reaffirm:** The course information provides support for your current ways of providing treatment.
        b. **Reenergize:** The course supports changes in areas that you have previously identified and provides the motivation and impetus to begin making those changes.
        c. **Reexamine:** The course addresses new research findings that merit further study and investigation as to the appropriateness of incorporation into practice.

c. **Am I active in my professional association?**
    1. Networking with colleagues exposes dental hygienists to other practicing professionals who can provide guidance and mentoring to younger members.
    2. Membership in a professional organization can provide free access to peer-reviewed journals.
    3. Active membership provides the opportunity to help shape evidence-based policies and guidelines for the organization.
    4. Provides immediate access to any clinical practice guidelines the association may develop.

d. **How well developed is my clinical judgment? Am I able to combine evidence and clinical experience to make a good decision?**

e. **Do I take into consideration what my patient wants?**
    1. Do I listen to my patients?
    2. Do I provide them with enough information and direction to make a good decision?
    3. Do I respect their autonomy and choices?

f. **Are there things that I should stop doing? Am I holding on to what I do because "that's what I learned in school" even though it was several years ago?**

g. **Are there things I need to change?**
    1. Are there better, more efficient, or more cost-effective tools available such as specific diagnostic tests, treatments, adjunctive therapies, medications, products, or procedures than what I am currently using?
    2. Do I have the appropriate amount of time scheduled or equipment provided for the highest level of patient care?

h. **Professional Responsibilities and Considerations**
    1. Per the ADHA Standards for Clinical Dental Hygiene Practice, dental hygienists are responsible and accountable for their dental hygiene practice, conduct, and decision-making.[5]
    2. Dental hygienists need to be able to access and utilize current, valid, reliable evidence in clinical decision-making through analyzing and interpreting the literature and other resources.[5]
    3. Dental hygienists need to maintain awareness of changing trends in dental hygiene, health, and society that impact dental hygiene care.[5]
    4. Dental hygienists need to participate in activities to enhance and maintain continuing competence and address professional issues as determined by appropriate self-assessment.[5]
    5. Dental hygienists need to commit to lifelong learning to maintain competence in an evolving health care system.[5]

Chapter Summary Statement

Best practice is a process of care with the goal of achieving consistent, superior patient outcomes. Best practice is founded on evidence-based data. The highest ranked level of evidence today is the systematic review, which is an evaluation of a body of research on a treatment or device through rigorous scientific methods to determine the overall validity and clinical applicability of that treatment or device. In addition to scientific data, best practice incorporates sound clinical judgment and patient values into the process. Achieving best practice requires that dental hygienists question and think about what they are doing and be open to learning new techniques. By using this approach to periodontal care, hygienists can meet the challenges of continuing to provide quality care in a rapidly changing field of dental health care.

Section 5

Focus on Patients

Clinical Patient Care

CASE 1

You have just started working in a new office and find that the other dental hygienist in the practice "doesn't believe in" using the ultrasonic equipment. The established dental hygienist feels that practicing for 20 years along with what was learned in school and "seeing good results with hand scalers" is evidence enough. It is a little intimidating because you have less experience (only 5 years), but you have routinely used ultrasonic instruments and mention "that is what you learned in school." For a while you pass it off as no big deal, a difference of opinions, but the equipment in the office is old and does not function at the level it should. You speak to your employer about getting a new machine, but he asks, "Why do you need this equipment?"

1. How would you answer your employer?
2. What types of evidence would you try to locate to justify your position?
3. Where would you search?
4. What types of key words would you use?
5. How would you manage your conflict with the established hygienist?

CASE 2

Your patient is a healthy, nonsmoking 30-year-old. The medical history reports no use of prescription drugs, only a daily multivitamin. The patient has been coming in for regular maintenance every 6 months and has a homecare routine of brushing two times per day and flossing perhaps once a week because "flossing is difficult and hurts." The exam shows some 4-mm probing depths and significant bleeding. As you have done several times in the past, you show the patient how to use the manual brush and floss and really "lay it on the line" about improving oral health and warning about coming in more frequently if the patient's habits do not improve. The patient promises to try but seems visibly upset when leaving. Although you hate upsetting a patient, you hope it helps improve the homecare situation.

About a month after the visit, you get a message that the patient would like to talk with you. When you connect, the patient tells you that a national newscast has just reported that "flossing has no proven medical benefits." The patient reports also reading about it on the Internet and talking with a relative who is a dental hygienist. The patient has read about automatic toothbrushes, interdental brushes, and oral irrigators and how they can help improve oral health. In fact, the patient has purchased one of everything, and feels improvements already. Not only that, the oral irrigating device makes the task so much easier and the convenience of those tiny brushes after lunch when at a work is perfect. The patient exclaims, "Why didn't you tell me about this? I am unhappy and going to have my records transferred elsewhere!"

1. What are some of the reasons you might have for not telling the patient about these products?
2. Ethically, is not telling a patient about all self-care products that have evidence to support their use the same as not telling a patient about all available professional treatment options? Why or why not?
3. What steps could you take to improve your knowledge of self-care products?

Evidence in Action

Using the Evidence to Communicate with Patients

Dental hygienists have an ethical obligation to recommend treatments based on the best available evidence. It is essential that clinicians learn how to effectively communicate these recommendations to patients. Patients will often have questions that reflect their own values, expectations, and perceived oral health needs. Acknowledging and respecting patient concerns is a key part of responding to questions and counseling patients about preferred treatments and any evidence-based alternatives.

It is common for patients to question why nonsurgical periodontal therapy is recommended. When practitioners are up to date and aware of current evidence, locating that evidence and providing an answer that knowledgably addresses the patients concerns and needs is much easier.

For example, a clinical practice guideline from the American Dental Association on nonsurgical treatment of periodontitis provides practitioners with a third-party, evidence-based recommendation on the treatment of choice for nonsurgical periodontal therapy.[14] Knowledge of documents that support everyday practice also helps align all members of the dental team to support a best practice treatment and provides strong documentation to the patient.

Ethical Dilemma

You have been working as a hygienist in the same periodontal practice for the last 10 years and are quite happy there. The practice has four dentists, so you have a very full schedule, which allows you to work four 10-hour days. The practice is very flexible and generous with their resources and benefits. They pay for continuing education courses for all staff members. Unfortunately, you haven't taken any courses since you have worked there, even though you know you are in violation of your state practice act. You have been very busy in your personal life over the last number years, as you got engaged, married, had two children within the span of 2 years, and are now caring for your aging parents. You just can't squeeze anything else into your life.

Your next patient is a 63-year-old real estate agent, who was referred to your practice for a periodontal evaluation 3 months ago. The patient comes armed with questions, articles, and information downloaded from the Internet. Although Dr. Willis, the periodontist, recommended traditional periodontal surgery, the patient is inquiring about laser procedures as well as bone and tissue regeneration, and is interested in your opinion on the articles and studies or if you think there are superior options to "gum surgery."

Although you want the patient to be an active partner in the treatment decision-making process, you feel very uncomfortable discussing this because you know you are not current in your dental knowledge and do not know how to accurately interpret the literature presented to you. Unfortunately, you have not updated or adapted your own treatment strategies, and still rely on what you learned in school over 10 years ago.

1. What ethical principles are in conflict in this dilemma?
2. What is the best way for you to handle this ethical dilemma?

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Recommended Readings

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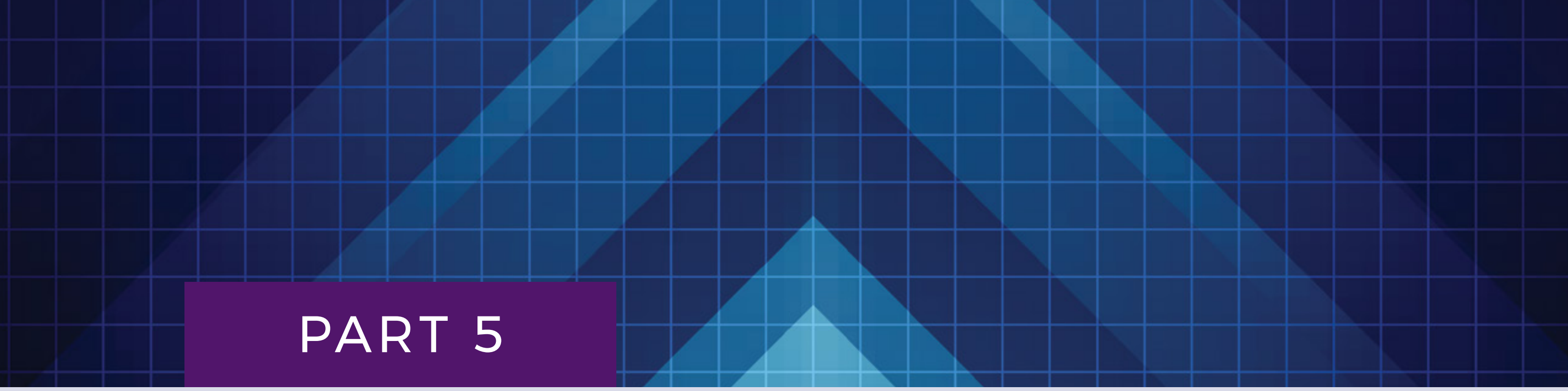

PART 5

Implementation of Therapy for Patients with Periodontal Diseases and Peri-Implant Diseases/Conditions

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CHAPTER 25

Nonsurgical Periodontal Therapy

- Section 1 Overview of Nonsurgical Periodontal Therapy
  - Understanding Nonsurgical Periodontal Therapy
  - Goals of Nonsurgical Periodontal Therapy
  - Types of Procedures Included in Nonsurgical Therapy
  - Nonsurgical Therapy Related to the Periodontal Diagnosis
  - Typical Treatment Plans for Nonsurgical Periodontal Therapy
- Section 2 Mechanical Nonsurgical Therapy
  - Overview of Instrumentation in Nonsurgical Therapy
  - End Point for Instrumentation During Nonsurgical Therapy
  - Dentinal Hypersensitivity Associated with Nonsurgical Therapy
- Section 3 Decisions Following Nonsurgical Therapy
  - The Reevaluation Appointment
  - The Relationship of Nonsurgical Reevaluation to Other Steps in Treatment
  - The Relationship Between Nonsurgical and Surgical Therapy
  - Recognizing the Need for Referral to a Periodontist
- Section 4 Focus on Patients
  - Clinical Patient Care
  - Ethical Dilemma

CLINICAL APPLICATION

Treating patients with periodontal diseases is a dynamic process that requires constant monitoring and continual reassessment of ongoing therapy. Periodontal therapy is often divided into phases and one of the first phases of therapy following comprehensive periodontal assessment is the nonsurgical therapy phase. Members of the dental team need to have a clear understanding of the importance of nonsurgical periodontal therapy and the decisions that are required following therapy. This chapter provides an overview of nonsurgical periodontal therapy, discusses some of the common unintended challenges that can arise during or after therapy, and outlines the decisions needed following reevaluation of the nonsurgical therapy.

LEARNING OBJECTIVES

- Define the term and list four goals of *nonsurgical periodontal therapy*.
- Explain the role of interdisciplinary collaborative care in nonsurgical periodontal therapy.
- Write a typical treatment plan for nonsurgical therapy of (1) a patient with dental biofilm–induced gingivitis and (2) a patient with generalized Stage I, Grade A periodontitis.
- Describe the type of healing to be expected following instrumentation of root surfaces.

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- Explain strategies for managing dental hypersensitivity.
- Explain why reevaluation is an important step during nonsurgical therapy.
- List the steps a clinician should perform at the periodontal reevaluation appointment.
- Discuss the rationale and indications for referring a patient to a periodontist.

KEY TERMS

Nonsurgical periodontal therapy
Interdisciplinary care
Periodontal instrumentation
Long junctional epithelium
Dentinal hypersensitivity
Reevaluation
Nonresponsive disease sites
Comanagement

Section 1

Overview of Nonsurgical Periodontal Therapy

Understanding Nonsurgical Periodontal Therapy

1. **Definition of Nonsurgical Periodontal Therapy.** **Nonsurgical periodontal therapy** describes the many nonsurgical steps taken by the clinician to eliminate the source(s) of inflammation in a patient with periodontal disease. The goal is to return the periodontium to a healthy state that can then be maintained by a combination of both professional care and patient self-care.
    a. **The Gold Standard**
        1. Nonsurgical periodontal therapy is the cornerstone of periodontal therapy and the first recommended approach to control periodontal infections.
        2. Although, nonsurgical periodontal therapy has evolved over the years, it is still the gold standard to which other treatment methods are compared.[1–4]
    b. **Alternate Terminology**
        1. Many terms have been used to describe nonsurgical periodontal therapy, and this fact may create some confusion. Some of the other terms that have been used to describe this same phase of treatment include *initial periodontal therapy*, *initial therapy*, *hygienic phase*, *anti-infective phase*, *cause-related therapy*, *phase I treatment*, and *soft tissue management*.
        2. *Nonsurgical periodontal therapy*, however, is one of the most frequently used terms for this phase of periodontal care.
2. **What Does Nonsurgical Periodontal Therapy Include?** Nonsurgical periodontal therapy includes self-care education, periodontal instrumentation, and the use of chemical agents to prevent or control periodontal disease. It is convenient to view nonsurgical periodontal procedures as the initial step in the periodontal treatment for a patient. However, for many patients, nonsurgical periodontal therapy alone is all that is needed to bring periodontal disease under control.
3. **What Are the Broad Objectives of Nonsurgical Periodontal Therapy?** Ideally, nonsurgical periodontal therapy should (1) remove both living microorganisms within the plaque biofilm and calcified nonliving microorganisms embedded within dental calculus that is adhering to the tooth surface, (2) eliminate, or at least minimize, periodontal inflammation and (3) return the periodontium to a healthy state that can then be maintained by a combination of patient self-care and professional care.
4. **Philosophy for Nonsurgical Periodontal Therapy**
    a. The basic tenants to successful nonsurgical periodontal therapy centers around controlling, managing, or eliminating (1) primary etiologic factors for periodontal disease, (2) local risk factors for periodontal disease, and (3) systemic risk factors for periodontal disease.
    b. Treatment included in a plan for nonsurgical periodontal therapy should be selected to meet the needs of each individual patient. Therefore, nonsurgical therapy plans can vary from patient to patient.

5. **General Principles of Controlling Periodontal Infection**
    a. **Patient Self-Care**
        1. Patients can remove supragingival plaque biofilm with daily brushing and use of interdental aids and/or irrigation devices.
        2. Unfortunately, studies show that many patients lack the motivation or skills to sustain a biofilm-free dentition over a substantial period of time.
        3. Clinical trials indicate that self-care alone, without the routine professional care, usually does not provide for long-term success in reversing gingivitis.[5–8]
    b. **Professional Care**
        1. Ramseier and colleagues examined a group of subjects first in 1970 and again in 2010 to assess long-term attachment loss and periodontitis-related tooth loss. The results of this study concluded that calculus removal, biofilm control, and the control of gingivitis are essential in preventing disease progression, further loss of attachment, and ultimately tooth loss.[9]
        2. Routine periodontal instrumentation is required to keep the subgingival microbial biofilm under control.[3]
        3. Adequate removal of subgingival biofilm is virtually impossible for patients to achieve on their own.[10,11]
        4. Studies indicate that periodontal instrumentation is needed approximately every 3 months to disrupt the harmful biofilm and to prevent or reverse any damage to the periodontal attachment apparatus.[2] Another reason for a 3-month recall is the need for continuous self-care counseling and patient motivation.
        5. Over time, maintenance intervals may be extended in patients with good compliance and with demonstrably stable supra- and subgingival environments.
6. **Factors Influencing Nonsurgical Therapy Outcomes.** Not all patients respond favorably to nonsurgical periodontal therapy.
    a. **Patient Compliance Factors.** Poor patient compliance with daily self-care and irregular professional maintenance appointments are common reasons for unfavorable treatment outcomes.
    b. **Lifestyle or Systemic Factors.** Uncontrolled systemic diseases/conditions and local factors may affect the outcome of nonsurgical therapy.[12,13]
    c. **Disease Factors.** Some cases of periodontitis are refractory and do not respond favorably in spite of well-executed nonsurgical periodontal therapy and patient self-care. In such a scenario, referral to a periodontist is prudent.

Goals of Nonsurgical Periodontal Therapy

Nonsurgical periodontal therapy constitutes the first step in controlling periodontal infections. The goals of nonsurgical periodontal therapy are discussed below and summarized in **Box 25-1**.

Box 25-1 Goals of Nonsurgical Periodontal Therapy

Goal 1: To mechanically (physically) remove the bacterial challenge to the patient
Goal 2: To eliminate or control local environmental risk factors for periodontal disease
Goal 3: To minimize the impact of systemic risk factors for periodontal disease
Goal 4: To stabilize the attachment level by eliminating inflammation

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1. **Goal 1: To Mechanically (Physically) Remove the Bacterial Challenge to the Patient**
    a. Control of the bacterial challenge usually involves intensive training of the patient in appropriate self-care techniques in combination with frequent professional care for the mechanical removal of plaque biofilm and calculus deposits from tooth surfaces.[1]
    b. Mechanical removal of plaque biofilm and calculus deposits contaminating the tooth surfaces are important steps in achieving control of the bacterial challenge. Calculus deposits are especially insidious because they are always covered with a layer of viable plaque biofilm which contributes to continuing inflammation if not removed.[14]

2. **Goal 2: To Eliminate or Control Local Contributing Factors for Periodontal Disease**
    a. Local contributing factors can increase the risk of developing periodontitis in localized sites. For example, defective restorations with overhangs can lead to plaque biofilm retention in a localized area. Local contributing factors are discussed in Chapter 16.
    b. Biofilm retention at a site over time allows periodontal pathogens to live, multiply, and lead to periodontal inflammation.
    c. A thorough plan for nonsurgical periodontal therapy will always include minimizing the impact of local contributing factors.
3. **Goal 3: To Minimize the Impact of Systemic Factors for Periodontal Disease**
    a. It is apparent that there are certain systemic diseases or conditions that can increase the risk of developing periodontitis or can increase the risk of developing more severe periodontitis where periodontitis already exists.[13]
        1. Systemic diseases or conditions such as diabetes mellitus may have a significant impact on long-term treatment outcomes.[15–19] Genetic susceptibility to periodontal disease is responsible for at least some disease occurrence.[20,21]
        2. Smoking is a highly significant risk factor for periodontitis.[12,22]
            a. Smokers exhibit compromised healing following nonsurgical and surgical therapy compared with nonsmokers or former smokers.
            b. Because most patients who do not respond well to therapy are smokers, smoking education and cessation programs should be included as part of a comprehensive periodontal treatment plan when indicated.
    b. A thorough plan for nonsurgical therapy always includes measures to minimize the impact of systemic risk factors. For example, a periodontitis patient with a family history of diabetes mellitus should be evaluated to rule out undiagnosed diabetes as a contributing systemic factor to the periodontitis. A medical evaluation should be included as part of the nonsurgical periodontal therapy. Another example would be a patient who smokes should receive smoking cessation counseling.
4. **Goal 4: To Stabilize the Attachment Level**
    a. The ultimate goal of nonsurgical periodontal therapy in periodontitis patients is to stabilize the level of attachment by eliminating the source of inflammation.
    b. Stabilization of the attachment level by eliminating the source of inflammation involves control of all the factors listed in the other goals of nonsurgical periodontal therapy.

Types of Procedures Included in Nonsurgical Therapy

Box 25-2 lists some of the nonsurgical therapy procedures that the dental team may utilize for patients. The list of procedures done as part of nonsurgical therapy is lengthy, but it should be evident that while some of the procedures are included in the treatment of all patients, other procedures are utilized only rarely. Because each patient presents unique treatment challenges, members of the dental team will need to customize the selection of the nonsurgical procedures included for each individual.

Box 25-2 Procedures That May Be Included in Nonsurgical Periodontal Therapy

1. Customized self-care instructions, including:
    a. Mechanical plaque biofilm control
    b. Chemical plaque biofilm control
    c. Adjunctive supragingival and subgingival oral irrigation
2. Periodontal instrumentation of tooth surfaces
3. Use of antimicrobial agents
4. Correction of local contributing factors
5. Interdisciplinary care for correction/control of systemic risk factors
6. Modulation of host defenses

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1. **Customized Patient Education and Self-Care Instructions**
    a. Patient education is a vital component of nonsurgical periodontal therapy. The dental team should ensure, from the onset of treatment, that the patient understands the long-term implications of periodontitis, both in terms of the benefits of treatment and the risks of nontreatment. Patients should be made aware that once the active phase of treatment is completed, they will require regular maintenance appointments to keep their periodontal condition stable.
    b. Another important element of nonsurgical periodontal therapy is effective daily self-care by the patient. Thus, patients need to be taught self-care skills and also be motivated to use those skills. These important topics are discussed in Chapters 26 to 28.
2. **Instrumentation of Tooth Surfaces.** Periodontal instrumentation is an essential component of nonsurgical periodontal therapy. The thoroughness of periodontal instrumentation determines the overall success of nonsurgical periodontal therapy in most patients.
3. **Use of Antimicrobial Agents.** A variety of antimicrobial agents are available in the form of irrigation solutions and gel applications. Antimicrobial agents are discussed in Chapter 28.
4. **Use of Adjunctive Supragingival and Subgingival Irrigation.** Some patients may benefit in supplementing their routine home oral hygiene regimen with an oral irrigation device. The rationale and the indications/contraindications for oral irrigation devices are discussed in Chapter 27.
5. **Correction of Local Contributing Factors.** Correction of local contributing factors, such as overhanging restorative margins or control of food impaction, is a critical component of nonsurgical periodontal therapy. In some cases, surgical therapy is necessary to gain better access to the local contributing factor so as to completely eliminate it, as in the case of a large subgingival restorative overhang. Local contributing factors are discussed in Chapter 16.
6. **Interprofessional Collaboration for Correction of Systemic Risk Factors**
    a. The American Dental Hygienists' Association defines **interdisciplinary care** as "[t]wo or more health care providers working within their respective disciplines who collaborate with the patient and/or caregiver to develop and implement a care plan."[23] Interprofessional collaboration values a team-based approach that emphasizes responsibility, accountability, communication, and cooperation between medical professionals, dental professionals, and allied health professionals to promote patient-centered care and positive outcomes.
    b. Correction of systemic risk factors such as undiagnosed diabetes is a critical component of nonsurgical periodontal therapy in some patients. This component often demands careful coordination of care with other health care providers such as physicians in light of the close interrelationship between oral health and systemic health. Systemic conditions that amplify susceptibility to periodontal diseases are discussed in Chapter 14. Conversely, the impact of periodontal disease on systemic health is discussed in Chapter 15.
7. **Modulation of Host Defenses.** When indicated, modulation of host defenses can also be a component of nonsurgical periodontal therapy. Modulation of host defenses is discussed in Chapter 29.

Nonsurgical Therapy Related to the Periodontal Diagnosis

Nonsurgical periodontal therapy is indicated for many patients, if not all patients. It may be helpful to view some common periodontal diagnoses and to see how nonsurgical periodontal therapy normally relates to these diagnoses.

1. **A Diagnosis of Dental Biofilm–Induced Gingivitis**
    a. Nonsurgical periodontal therapy is the primary type of care provided for most patients with dental biofilm–induced gingivitis.
    b. Thorough nonsurgical therapy can normally bring dental biofilm–induced gingivitis under control and bring it to a point that periodontal health can be maintained by a combination of both professional care and patient self-care.
    c. This general relationship between nonsurgical therapy and a diagnosis of dental biofilm–induced gingivitis should not be interpreted to mean that patients with this diagnosis never need periodontal surgery because periodontal surgery is sometimes needed to correct other existing problems (i.e., gingival recession or gingival overgrowth) in gingivitis patients.

2. **A Diagnosis of Stage I or II with Grade A Periodontitis**
    a. Thorough nonsurgical therapy can also bring many cases of incipient or moderate periodontitis under control and bring it to a point that periodontal health can be maintained by a combination of both professional care and patient self-care.[24–27]
    b. Note that some patients with this type of periodontitis diagnosis may require periodontal surgery if the periodontium does not respond favorably to standard nonsurgical treatment principles.[28]
    c. Periodontal surgery is sometimes used to correct other problems (i.e., gingival recession) and may indeed be indicated in any patient.
    d. A patient with Grade A periodontitis who exhibits few risk factors is more responsive to standard periodontal therapy than a patient with Grade C periodontitis. Nonetheless, it is important to periodically monitor and continually reassess an individual's periodontitis grade at each recall visit to identify any potential future risk factors (i.e., patient resumes smoking habit) that may alter the estimate of the future course of the disease.
3. **A Diagnosis of Stage III or Stage IV with Grade B or C Periodontitis**
    a. For most patients with Stage III or Stage IV periodontitis, control of the periodontitis requires thorough nonsurgical periodontal therapy and more advanced periodontal procedures, such as periodontal surgery. As discussed in Chapter 6, both Stage III and Stage IV represent a periodontitis that is characterized by significant periodontal breakdown that severely jeopardizes the support and function of the dentition.
    b. Although periodontal surgery is frequently indicated for patients with more advanced periodontitis, all periodontitis cases, regardless of severity, must undergo nonsurgical therapy prior to surgical intervention.
    c. Nonsurgical periodontal therapy is frequently successful in minimizing the extent of any surgery subsequently needed and may be needed to improve the outcomes of that periodontal surgery.
    d. A patient with Grade B or C periodontitis exhibits several known risk factors (i.e., smoking factors, systemic factors, etc.) that increase the likelihood of the disease progressing at a faster rate than is typical for most of the population and are less responsive to standard periodontal therapies. Thus, such a patient will require more intensive management of their condition and will need to be continually reassessed to identify any newly emerging future risk factors that may alter the estimate of the future course of the disease.

Typical Treatment Plans for Nonsurgical Periodontal Therapy

A treatment plan for nonsurgical therapy is a list of nonsurgical procedures or interventions that addresses a patient's periodontal health needs as identified during the periodontal assessment. Although it is critical that a nonsurgical treatment plan meets the needs of each individual patient, new clinicians often find it helpful to view examples of typical treatment plans. A properly designed treatment plan for nonsurgical periodontal therapy would include procedures to be carried out by the dental hygienist, by the dentist, and by the patient. It is important to keep in mind that the examples of the typical treatment plans provided below are intended to augment, *not replace*, the sound clinical judgment of the dental provider. In turn, the obligation of the practitioner is to design an individualized treatment plan that is tailored to specifically address the needs of an individual patient.

1. **Examples of Typical Treatment Plans**
    a. **Dental Biofilm–Induced Gingivitis.** A typical plan for nonsurgical therapy for a patient with moderate dental biofilm-induced gingivitis might include the following:
        1. Customized self-care instructions, including patient education and motivation.
        2. Periodontal instrumentation (typically a *dental prophylaxis* in American Dental Association [ADA] terminology).
        3. Elimination of plaque-retentive factors such as overhanging restorations, caries, or ill-fitting dental prostheses.
        4. Reevaluation of the patient's periodontal status.
            a. Response to nonsurgical therapy is normally delayed because it takes some time for the body's defense mechanisms to respond to individual treatment steps.
            b. Because of this delay time in healing, the dental team is obligated to reevaluate the results of nonsurgical periodontal therapy after a period of healing to ensure that all appropriate measures have been included and to identify any other measures that might be needed.

        c. It is wise for the dental team to include this reevaluation step in the plan for nonsurgical therapy so that the patient has a clear understanding from the outset how future treatment decisions will be made.

b. **Stage I or Stage II with Grade A Periodontitis.** As already discussed, it is always important to customize nonsurgical therapy to the needs of each patient. But again, it may be helpful to look at a typical nonsurgical plan for a patient with incipient or moderate periodontitis. This typical plan might include the following:
    1. Customized self-care instructions including patient education and motivation.
    2. Periodontal instrumentation (typically *scaling and root planing* in ADA terminology).
    3. Control of local risk factors to include steps such as removal of overhanging restorations, restoration of caries, or minimizing excessive occlusal forces.
    4. Correction of systemic risk factors to include steps such as smoking cessation counseling or referral for control of diabetes.
    5. Reevaluation of patient's periodontal status.

c. **Stage III or Stage IV with Grade B or C Periodontitis.** Most patients with Stage III or Stage IV periodontitis exhibit complicating factors such as advanced attachment loss, deep probing depths, advanced alveolar bone loss, furcation involvements, or mucogingival problems that require more advanced treatment procedures later in therapy.
    1. A typical treatment plan for nonsurgical therapy for a patient with Stage III or Stage IV periodontitis may require periodontal surgery as part of the treatment plan.
        a. Members of the dental team should be aware of the possible need for periodontal surgical intervention in patients with more advanced periodontitis.
        b. As the severity of periodontitis increases, it becomes more likely that periodontal surgery will be needed to bring the disease under control.
        c. The need for periodontal surgical therapy should be reevaluated after the completion of nonsurgical periodontal therapy. Surgical periodontal therapy is discussed in Chapter 30.

Section 2

Mechanical Nonsurgical Therapy

Overview of Instrumentation in Nonsurgical Therapy

1. **Periodontal Instrumentation During Nonsurgical Therapy.** It is well-established that removal of plaque biofilms and calculus is the major goal of periodontal instrumentation.
    a. **Plaque Biofilm**
        1. Because of the biofilm structure, mechanical removal of plaque biofilm is the most effective mechanism of control, though chemical agents are sometimes used to improve the patient response to these procedures.
        2. Most subgingival biofilm within pockets cannot be reached by brushes, floss, or antibacterial rinses.
            a. For this reason, frequent periodontal instrumentation of subgingival root surfaces to mechanically remove or disrupt plaque biofilms is an essential therapeutic component for most patients with periodontitis.
            b. In fact, periodontal instrumentation is likely to remain an important component of nonsurgical periodontal therapy for the foreseeable future.
    b. **Calculus Deposits**
        1. Removal of calculus deposits from tooth surfaces is a critical component in any plan for nonsurgical periodontal therapy.
        2. Calculus deposits harbor living bacterial biofilms. If calculus remains, so do the bacteria, making it impossible to reestablish periodontal health. Calculus removal is always a fundamental part of periodontal therapy.

2. **Preservation of Root Cementum During Periodontal Instrumentation**
   a. **Cementum's Role in the Periodontium**
      1. Cementum is a key component of periodontal tissues, and its preservation is of paramount importance for the quality of healing at the completion of both nonsurgical and surgical treatment modalities.
      2. Cementum can influence the activities of periodontal cells and may play an important regulatory role in periodontal treatment. The ideal outcome for periodontal therapy hinges on the complete removal of supragingival and subgingival plaque biofilm and calculus while preserving cementum.
      3. Periodontal reattachment or new attachment as the end result of therapy strongly relies on the presence of cementum after root instrumentation. Improper or aggressive mechanical instrumentation may reduce the thickness or eventually remove all the cementum over the root surface. This will result in exposure of dentin to the oral environment and make the tooth more susceptible to root caries or dentinal hypersensitivity.
   b. **Historical Perspectives Regarding Cementum Removal**
      1. Previously, it was postulated that bacterial endotoxins or bacteria penetrate the cementum of periodontally diseased root surfaces.
      2. This concept resulted in the removal of all or most of the cementum as a primary end point of periodontal healing.[29,30]
      3. More specifically, the goal of periodontal therapy was to obtain a treated root surface with smooth and hard surface characteristics that was free of endotoxins.[29,31]
   c. **Current Evidence-Based Perspectives Regarding Cementum Removal**
      1. In contrast, recent studies report that endotoxins are not located within cementum[20,32] and that removal of cementum is not necessary for a successful periodontal treatment.[33]
      2. The preservation of cementum on the root surface was further supported by Saygin et al. who reported that cementum was necessary for new attachment and as a source of growth factors.[34,35]
      3. Furthermore, Grzesik and Narayanan suggested that cementum plays an important regulatory role in periodontal regeneration.[36]
      4. From these studies, it can be concluded that conservation of cementum is necessary for optimal periodontal health as well as for periodontal regeneration.
      5. During periodontal instrumentation, the extent of instrumentation should be limited to that needed to obtain a favorable tissue response. Root surfaces should be instrumented only to a level that results in resolution of tissue inflammation in the periodontal tissues.
3. **Evolving Instrumentation Terminology.** The terminology for periodontal instrumentation has evolved over the past several years. The careful reader will be wise to note the terminology that appears in many dental hygiene journals and textbooks compared to the terminology in dental journals and textbooks. The differences in this terminology are described below.
   a. **Traditional Instrumentation Terminology.** Traditional instrumentation terminology uses the terms *scaling* and *root planing*. As traditionally defined, *scaling* is instrumentation to remove plaque biofilm and calculus off the root surface, whereas *root planing* is a treatment procedure designed to remove diseased cementum that is contaminated with toxins or microorganisms.
   b. **Emerging Instrumentation Terminology**
      1. Recently in the dental hygiene literature, increasing numbers of authors are using new terminology that reflects modern therapy better than the traditional terminology. The term *periodontal instrumentation* or *periodontal debridement* is suggested to replace the older terms *scaling* and *root planning*.
      2. **Periodontal instrumentation** (periodontal debridement) is defined as the supragingival and subgingival removal or disruption of plaque biofilm, its byproducts, and calculus deposits to the extent needed to reestablish periodontal health and restore a balance between the bacterial flora and the host's immune response.
   c. **Considerations Regarding Emerging Terminology and Insurance Codes**
      1. Insurance codes are numeric codes to identify different dental procedures. The most important use of codes is for insurance billing purposes. Insurance codes are entered on insurance forms indicating the dental treatment listed by the appropriate procedure code number. In the United States, insurance codes are published in the ADA Current Dental Terminology. These codes are very specific and should be reviewed carefully before specific dental treatment is coded.

2. The ADA Current Dental Terminology continues to use the traditional terms *prophylaxis* and *scaling* and *root planing* to describe periodontal instrumentation. This difference in terminology can be confusing to clinicians.
   a. Although the term *periodontal instrumentation/debridement* as defined in the dental hygiene literature may describe modern periodontal therapy better than older terms, this terminology has not yet replaced the older terms as recognized by the ADA. For this reason, many dentists have been reluctant to embrace the new terminology without changes in ADA codes.
   b. Some authors and clinicians have redefined the term *root planing* so that its meaning is similar to that of *periodontal instrumentation/debridement*. This approach of redefining the term *root planing* can be confusing, however, because it is difficult to determine which definition of root planing any one person is using.
   c. At this time, dental team members should use the currently accepted insurance codes when filling out insurance forms and in communications with insurance companies or other third-party payers.

End Point for Instrumentation During Nonsurgical Therapy

The end point of periodontal instrumentation is to return the tissues of the periodontium to a state of health. In this context, *health* means periodontal tissues that are free of inflammation.

1. **Healing Following Nonsurgical Instrumentation**
   a. After thorough periodontal instrumentation, some healing of the periodontal tissues will normally occur.
      1. The primary type of healing in a site of attachment loss after periodontal instrumentation is through the formation of a long junctional epithelium.
         a. As inflammation in the periodontium resolves, epithelial cells can readapt to the root surface, as shown in **Figure 25-1**.

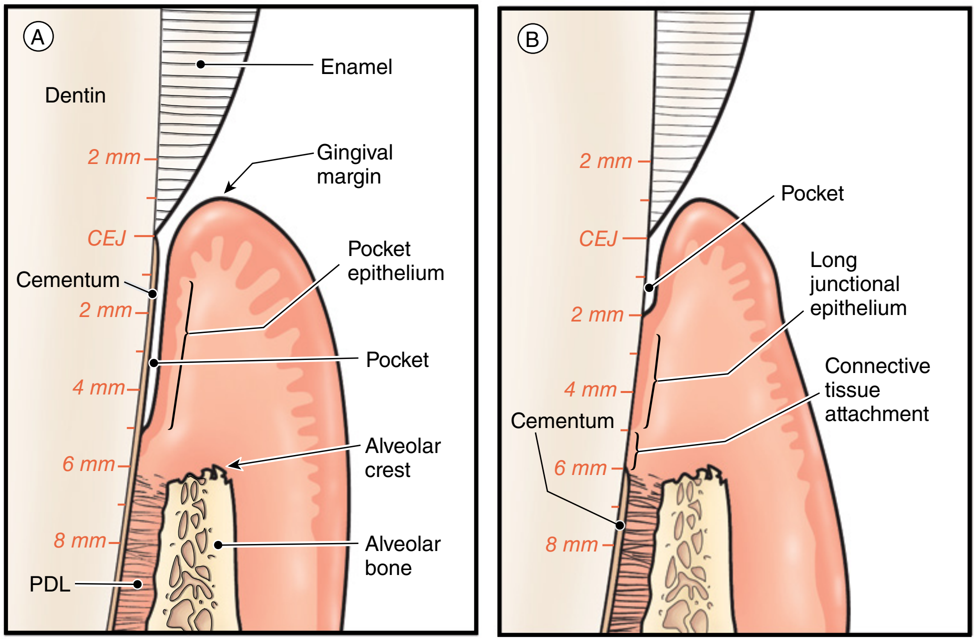

Figure 25-1 Healing After Nonsurgical Periodontal Instrumentation. The drawing on the left depicts the tissue before therapy; the periodontal pocket has a probing depth of 6 mm. The drawing on the right depicts the tissue after periodontal therapy; the tissue healing is through the formation of a long junctional epithelium. This results in a probing depth of 3 mm. Note that there is no formation of new bone, cementum, or periodontal ligament during the healing process that occurs after nonsurgical periodontal instrumentation.

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         b. This adaptation of the epithelial cells to the root surface is referred to as a **long junctional epithelium**.

2. Note that new alveolar bone, new cementum, or new periodontal ligament normally do not form following periodontal instrumentation. In other words, nonsurgical periodontal instrumentation does not stimulate periodontal regeneration.

b. Clinically, nonsurgical periodontal therapy, including instrumentation, may certainly result in reduced probing depths. **Figure 25-2** shows examples of various soft tissue responses to thorough periodontal instrumentation.

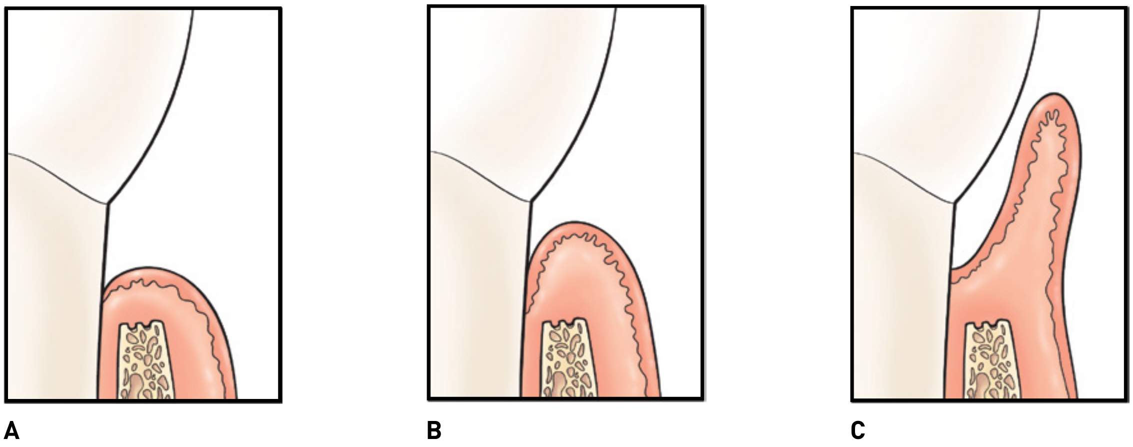

Figure 25-2 Soft Tissue Responses to Thorough Periodontal Instrumentation. The following panels show some of the possible tissue changes that can occur following thorough periodontal instrumentation of a root surface: **A.** There can be complete resolution of the inflammation resulting in shrinkage of the tissue and a shallow probing depth. **B.** There can be readaptation of the tissues to the root surface with a long junctional epithelium resulting in a shallow probing depth. **C.** There can be very little change in the level of the soft tissues resulting in a residual periodontal pocket.

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1. The reduced probing depths result from the formation of a long junctional epithelium combined in many instances with resolution of gingival edema that is usually a part of the inflammatory process. **Figure 25-3** shows how some of this reduction in probing depth can occur at the base of a periodontal pocket.

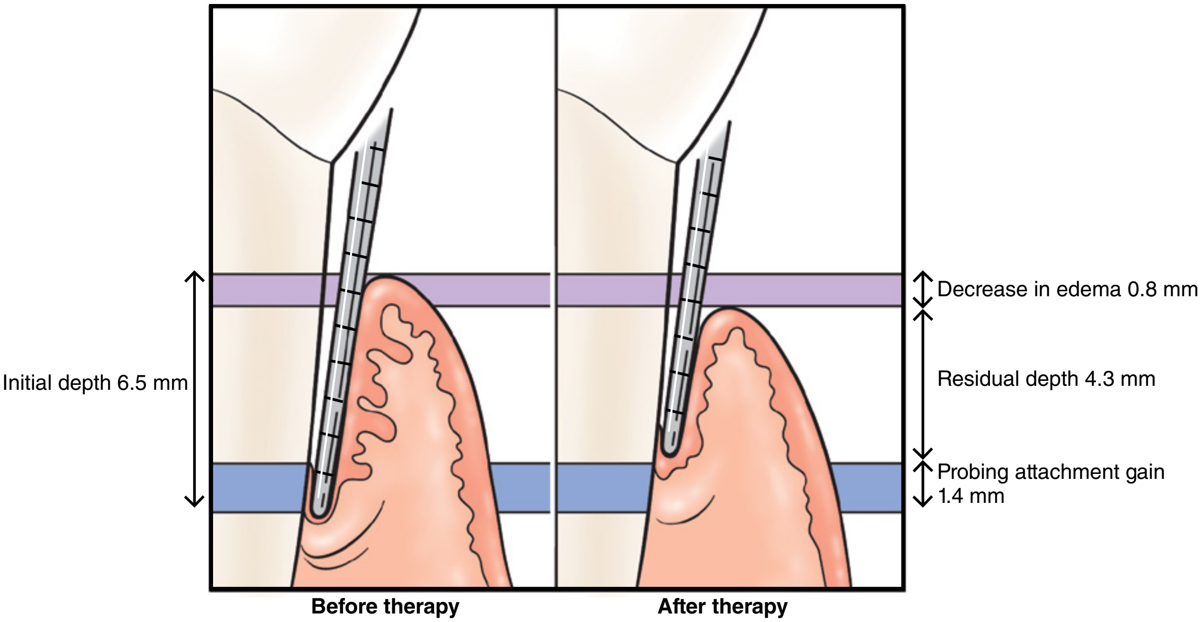

Figure 25-3 Details of Healing at the Base of a Pocket Following Periodontal Instrumentation. Given that the tip of a periodontal probe can easily penetrate inflamed soft tissue in the base of periodontal pocket, the probing depth may decrease following instrumentation because the tissues at the base of the pocket can be much more resistant to deeper penetration by the probe. This figure also depicts a slight decrease in gingival edema resulting from resolution of inflammation in the tissue.

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    2. Thus, one important clinical feature to monitor following periodontal instrumentation, in addition to clinical attachment loss, is the probing depths.
    3. Note at this point that there are other clinical signs that can correlate with inflammation that can be expected to change following periodontal instrumentation (i.e., bleeding on probing).
2. **Assessing Tissue Healing.** Tissue healing does not occur overnight, and in many patients, it is not possible to assess the true tissue response until at least 1 month after the completion of periodontal instrumentation. Assessing tissue healing during and following nonsurgical therapy is discussed in Section 3 of this chapter.

Dentinal Hypersensitivity Associated with Nonsurgical Therapy

Dentinal hypersensitivity, as described in this section, is not really a periodontal disease or a periodontal condition. It is not even considered a pathologic condition because "sensitive" dentin histologically looks the same and physiologically behaves the same as "nonsensitive" dentin. In fact, some clinicians believe that the sharp pain characteristic of dentinal hypersensitivity may be the normal pulpal response when the dentin is exposed to the oral environment. However, dentinal hypersensitivity appears so frequently following nonsurgical periodontal therapy that clinicians need to be aware of this condition, understand its origin, and understand therapies for the condition. However, keep in mind that not all teeth with exposed dentin are sensitive to mechanical, chemical, osmotic, tactile, or thermal stimuli; in such situations, the open dentinal tubule is covered by a protective smear layer protecting it from the external stimuli.

1. **Introduction to Dentinal Hypersensitivity**
    a. **Dentinal hypersensitivity** (also referred to as dentinal sensitivity) is a short, sharp, painful reaction that occurs when areas of exposed dentin are subjected to mechanical, thermal, or chemical stimuli. For example, an individual might experience heightened sensitivity while brushing, when eating cold foods (such as ice cream), or when eating sweet, sour, or acidic foods (such as grapefruit). In some patients, simply breathing in cold air while walking outside on a frigid day can trigger this painful reaction.
    b. **Anatomy of the Dentino–Pulpal Complex**
        1. Throughout the thickness of the dentin, there are long, thin branching tunnels known as dentinal tubules. The dentinal tubes are typically oriented perpendicular to the long axis of the tooth (**Figure 25-4**).

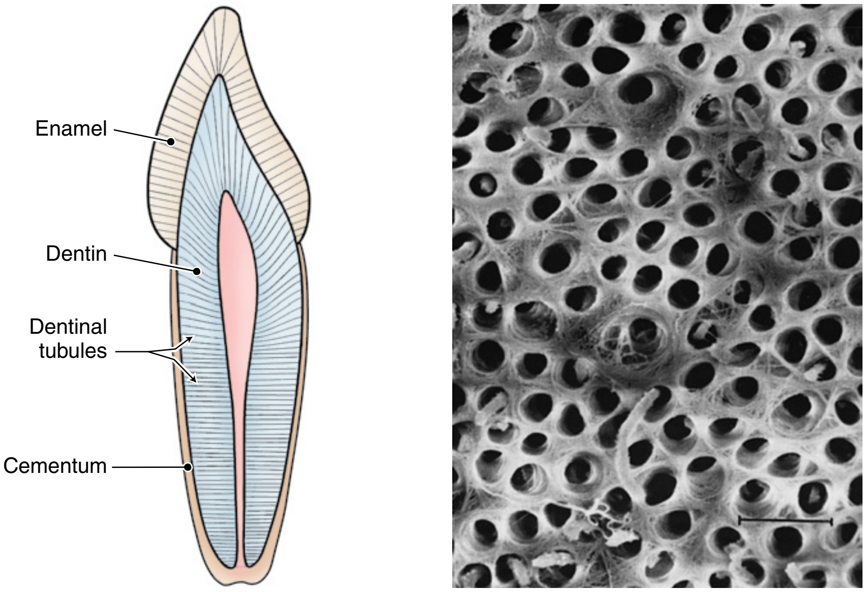

Figure 25-4 **Dentinal Tubules.** The diagram on the left shows the many dentinal tubules that run perpendicular to the outer surface of the dentin to the pulp. On the right, a scanning electron micrograph of the cross section of dentinal tubules adjacent to the pulp chamber of a human tooth. The black line engraved in the lower right is 10 microns long.

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2. Within each dentinal tubule is an odontoblastic process, which is a direct extension of a cell in the tooth pulp known as an odontoblast (**Figure 25-5**), and dentinal fluid, which is extracellular fluid that surrounds the odontoblastic process.

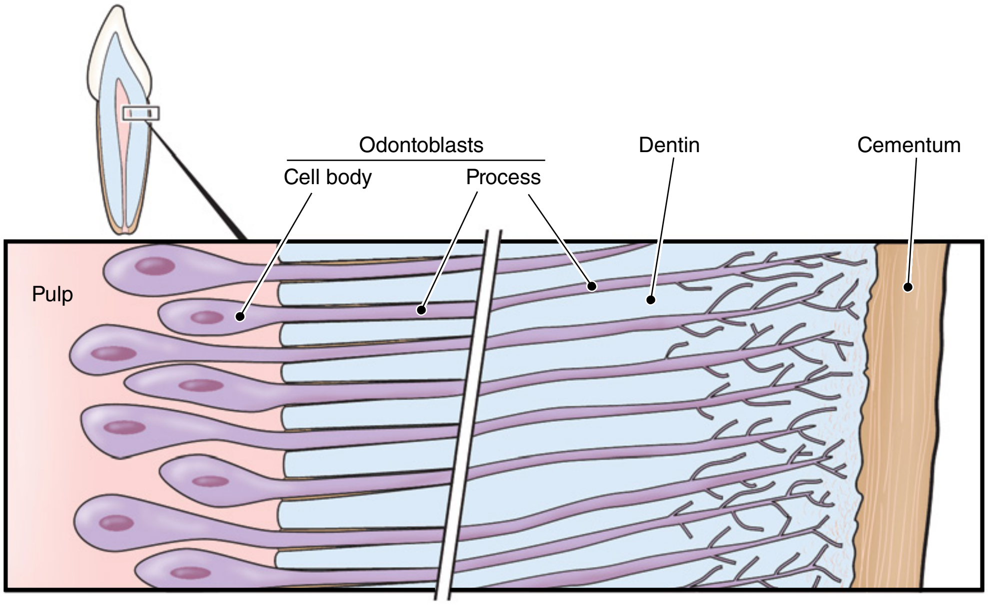

Figure 25-5 **Odontoblastic Process.** The odontoblastic cell process in the dentinal tubule often fills the part of the dentinal tubule closest to the pulp but can extend farther from the pulp toward the junction of the dentin with the enamel or cementum.

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3. One of the functions of cementum is to protect the underlying radicular dentin. As long as the cementum remains intact and covers the radicular dentin, the underlying dentin will be protected from any external stimuli that could potentially trigger the pain associated with dentinal hypersensitivity.

c. **Mechanism of Dentinal Hypersensitivity**
   1. Dentinal hypersensitivity develops in two phases. First, the dentin loses its protective cementum covering (lesion localization). This leads to exposure of the dentinal tubules to the oral environment (lesion initiation).
   2. Three theories have been proposed to explain the mechanism behind dentinal hypersensitivity.
      a. **Direct Stimulation Theory**
         1. This theory postulated that the pulpal nerve could extend through the entire extent of the dentinal tubule. As such, any external stimulus that acts upon the exposed dentinal tubules could potentially activate the nerve ending.
         2. However, this theory lost much credence as it was later found that pulpal nerve endings do not traverse through the entire length of the dentinal tubule and innervate the outer dentin, which is the most sensitive part of the root.[37]

      b. **Odontoblastic Transducer Mechanism Theory**
         1. This theory postulated that the odontoblasts themselves act as receptor cells that relay signals to a nerve terminal. This, in turn, would stimulate the pain fibers in the pulp and trigger the sudden sharp pain sensation characteristic of dentinal hypersensitivity.[38]
         2. This theory, however, lost much support when subsequent microscopic studies failed to show synapses between odontoblasts and nerve terminals.[39]

      c. **Hydrodynamic Theory**
         1. This theory proposes that movement of the dentinal fluid in the tubules activates nerve cell endings that initiate the pain response at the dentin–pulpal complex.[40]

2. Stimuli—such as cold thermal stimulus, hot thermal stimulus, or evaporative stimulus—cause the dentinal fluid to shift back and forth within the tubule. This movement of the fluid, in turn, excites the nerve cell endings and triggers the sharp pain response characteristic of dentinal hypersensitivity.
3. The hydrodynamic theory would explain why some patients with dentinal hypersensitivity may experience sharp pain when inhaling cold air through the mouth. Currently, the hydrodynamic theory is the most widely accepted theory to explain dentinal hypersensitivity.

2. **Precipitating Factors for Hypersensitivity**
   a. Dentinal hypersensitivity is usually associated with exposure of dentin as a result of loss of either enamel or cementum. Factors that can contribute to dentinal hypersensitivity include apical displacement of the gingival margin (caused by traumatic toothbrushing, dental erosion, and periodontitis) or incisal (or occlusal) attrition, abrasion, or erosion that leads to loss of enamel. Even nonsurgical periodontal instrumentation can inadvertently contribute to recession because of the resolution of swelling of the gingiva. In fact, approximately half of patients who undergo scaling and root planing experience post-treatment dentinal hypersensitivity.[41]
   b. **Dentinal Hypersensitivity Following Periodontal Instrumentation**
      1. Note that most dentinal hypersensitivity following periodontal instrumentation is mild and resolves within a few weeks if the exposed root surfaces are kept biofilm-free by thorough self-care.
      2. In its more severe forms, however, dentinal hypersensitivity can result in so much discomfort that it can prevent a patient from performing thorough self-care. It is fortunate that most instrumentation of root surfaces does not result in the development of dentinal hypersensitivity.
      3. Sensitivity may not occur in some instances because instrumentation of root surfaces can result in a smear layer of dentin that covers the root surfaces and blocks the tubules. This so-called smear layer refers to crystalline debris from the tooth surface that covers or plugs the dentinal tubules during the instrumentation; this smear layer blocks the tubules, inhibits fluid flow, and prevents the development of sensitivity.
3. **Strategies for Managing Patients with Dentinal Hypersensitivity.** A number of strategies have been recommended for dealing with dentinal hypersensitivity (**Figure 25-6**). These strategies range from simply allowing the open dentinal tubules to seal themselves to performing periodontal surgery. Each of the strategies has proved successful for some patients. **Table 25-1** provides an overview of chemical agents that have been investigated for the control of dentinal hypersensitivity by occluding dentinal tubules.

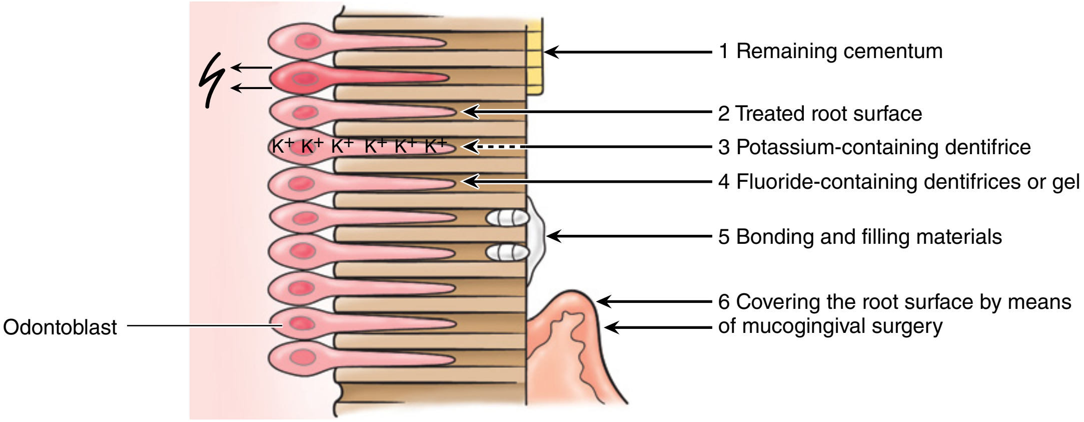
1 Remaining cementum
2 Treated root surface
K+ K+ K+ K+ K+ K+
3 Potassium-containing dentifrice
4 Fluoride-containing dentifrices or gel
5 Bonding and filling materials
6 Covering the root surface by means of mucogingival surgery
Odontoblast

Figure 25-6 **Treatment Management Options to Alleviate Dentinal Hypersensitivity.** Chemicals can be used to occlude the dentinal tubules and to eliminate or minimize associated dentinal sensitivity. Another treatment modality is mucogingival surgery to cover the exposed dentin on the root surface. Soft tissue grafts will also provide a more esthetic outcome as it reestablishes the natural contours and harmony of the gingival margin of the affected tooth with adjacent teeth.

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Table 25-1 Overview of Agents That Have Been Investigated for the Control of Hypersensitivity by Occluding Dentinal Tubules

Agents that can precipitate proteins that can occlude dentinal tubules:
- Glutaraldehyde
- Zinc chloride
- Silver nitrate
- Strontium chloride

Agents that can precipitate minerals that can occlude dentinal tubules:
- Sodium fluoride
- Calcium phosphate
- Stannous fluoride
- Calcium carbonate
- Strontium chloride
- Arginine
- Potassium oxalate
- Bioactive glass
- Silver diamine fluoride

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a. **Periodontal Instrumentation in Patients with Existing Dental Hypersensitivity**
   1. For teeth with existing dentinal hypersensitivity, instrumentation of hypersensitive root surfaces can elicit sharp pain during a clinical procedure such as periodontal instrumentation or drying the tooth with a compressed stream of air.
   2. Because many patients have existing dentinal hypersensitivity, it is critical for dental hygienists to ask patients about hypersensitive teeth prior to attempting a clinical procedure such as periodontal instrumentation.
   3. For patients with existing hypersensitivity, local anesthesia can be used to control any discomfort that might arise during thorough instrumentation.

b. **Patient Education—A Critical Element in Patient Management.** In the patient's eyes, tooth sensitivity may simply be a result of nonsurgical treatment. However, in reality, the heightened sensitivity results from areas where clinical attachment loss has previously occurred as a result of existing periodontitis. An appropriate strategy for managing patients undergoing nonsurgical periodontal therapy would include educating patients about the possibility of developing hypersensitivity following the procedure. This warning should be given before beginning any treatment and should be part of the discussion preceding informed consent. It may be helpful to provide the patient with the following facts before initiating instrumentation:
   1. Sensitivity to cold can increase following periodontal instrumentation.
   2. If sensitivity resulting from periodontal instrumentation occurs, it will usually gradually disappear over a few weeks.
   3. Thorough daily plaque biofilm removal during self-care is one of the most important factors in the prevention and control of sensitivity. Note that without meticulous self-care, treatments for dentinal hypersensitivity are usually not successful.
   4. If dentinal hypersensitivity becomes an ongoing problem, recommendations for in-home therapies can be made that can enhance its resolution, but immediate results should not be expected.

c. **Occluding Patent (Open) Dentinal Tubules with In-Home Therapies**
   1. One important strategy for managing patients with dentinal hypersensitivity involves applying chemicals to the exposed root surface that can help occlude (or block) the openings of the dentinal tubules.
   2. The chemicals used to occlude the dentinal tubules often eliminate or minimize associated sensitivity. The mechanism of action of these chemicals has been reported to be precipitating minerals or precipitating proteins within open dentinal tubules resulting in sealing the openings of the tubules.

   3. Many of these chemical agents are available in special formulations of toothpastes, and a variety of toothpastes have been specifically formulated to aid in desensitizing teeth.[42–45] Many chemical agents have been reported to help in some patients. Examples of some of the more common chemical agents in toothpaste formulations for dentinal hypersensitivity are potassium nitrate, strontium chloride, sodium citrate, and fluorides. Table 25-1 includes an overview of some of the chemical agents that have been investigated for control of hypersensitivity by blocking dentinal tubules.
   4. The reported efficacy of the chemical agents in controlling dentinal hypersensitivity varies, but this strategy remains the most commonly utilized treatment for this condition.

d. **Occluding Patent Tubules with In-Office Therapies**
   1. Professionally applied in-office products are also available that contain chemicals that also have been reported to decrease dentinal hypersensitivity.
   2. A variety of chemical agents have been utilized in in-office remedies for dentinal hypersensitivity. Examples of chemicals that have been reported to decrease hypersensitivity following in-office applications are potassium oxalate, ferric oxalate, and fluoride solutions or fluoride varnishes.
   3. Table 25-1 lists an overview of some of the chemical agents that have been investigated for control of hypersensitivity by blocking dentinal tubules.

e. **Desensitizing Nerves Associated with the Tubules.** It has also been reported that applying certain chemicals to the root surface can block the nerve receptors in or near the tooth pulp from activating the painful response.
   1. The chemical agents that can be used to block the nerve receptors from activating a painful response include potassium salts (such as potassium nitrate); potassium nitrate is the active ingredient found in some desensitizing toothpastes and has been shown to be effective in decreasing sensitivity in some patients.
   2. It is thought that potassium depolarizes the nerve fibers associated with the odontoblastic processes, thus preventing pain signals from traveling to the brain.

f. **Treating Exposed Dentin Surfaces with Lasers**
   1. Lasers have been used to treat exposed hypersensitive dentin surfaces with some success.
   2. The mechanism of action of lasers in decreasing dentinal hypersensitivity is not clear at this point.

g. **Blocking Dentinal Tubules with Restorative Materials**
   1. One strategy for blocking some dentinal tubules is to cover the exposed dentin surface with a bonding agent—the same kinds of materials that can sometimes be used to restore caries.
   2. Because the exposure of dentin at some sites can be associated with missing tooth structure resulting from abrasion or erosion of the root surface, bonding a restorative material over that surface can not only block exposed dentinal tubules, but it can also restore any missing tooth structure.
   3. Examples of restorative materials that have been used to block dentinal tubules include oxalic acid and resin, glass ionomer cements, composites, and dentin-bonding agents.

h. **Blocking Dentinal Tubules with a Periodontal Surgical Procedure**
   1. Another strategy for blocking the open dentinal tubules is to cover the root surface with a gingival grafting material.
   2. This approach can result in rebuilding the gingiva to a more natural level that covers an exposed tooth root, covering the dentinal tubules.

Section 3

Decisions Following Nonsurgical Therapy

The Reevaluation Appointment

Reevaluation refers to a formal step after the completion of nonsurgical therapy. During the reevaluation appointment, the members of the dental team perform another periodontal assessment to gather information about the patient's periodontal status and determine how well the periodontium has responded to initial therapy. After comparison with the periodontal status at the time of the initial assessment, the team members make several critical clinical decisions regarding management of the patient's periodontal condition. The reevaluation is described in detail below.

1. **The Reevaluation Appointment**
    1. **Timing of a Reevaluation**
        1. Periodontal tissue healing does not occur immediately, and in most cases, it is not possible to determine the true tissue response for at least 1 month after the completion of nonsurgical periodontal therapy.
        2. Members of the dental team should usually schedule a reevaluation appointment of a periodontitis patient 4 to 6 weeks after completion of nonsurgical therapy.
    2. **Understanding the Steps in a Reevaluation.** The steps in a typical reevaluation appointment usually include those listed below (**Box 25-3**):
        1. The first step in the reevaluation appointment is to review and update the patient's medical history. Of course, this is the first step in any patient appointment.
        2. The second step is to perform a thorough clinical periodontal assessment. The nature of a clinical periodontal assessment has already been described in Chapter 19.
        3. The third step is to compare the results of the patient's initial periodontal assessment with the results from the patient's reevaluation assessment.
        4. The fourth step is to make appropriate decisions related to the next steps in therapy.

Box 25-3 Steps in a Typical Reevaluation Appointment

1. Update the medical status of the patient.
2. Perform a periodontal clinical assessment.
3. Compare the initial periodontal assessment with the reevaluation assessment.
4. Make decisions related to the next steps in periodontal therapy.

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2. **Options for Treatment Following Reevaluation**
    1. **Managing Nonresponsive Disease Sites.** During the reevaluation, members of the dental team may identify nonresponsive disease sites.
        1. **Nonresponsive disease sites** are areas in the periodontium that continue to exhibit persistently deep or even deeper probing depths, continued loss of attachment, or continued clinical signs of inflammation in spite of the nonsurgical therapy previously provided. This would be consistent with the characteristics of a refractory periodontitis (see Chapter 6).
        2. Nonresponsive sites should be carefully reassessed for thoroughness of self-care and evaluated for the presence of residual or re-formed subgingival plaque biofilm and calculus deposits.
        3. If plaque biofilm or calculus deposits are discovered at a nonresponsive site, the site should be mechanically instrumented with an ultrasonic scaler (unless ultrasonics are contraindicated for this patient) and/or hand instruments. Then, the patient should receive additional self-care motivation and training.
        4. When nonresponsive sites are encountered, members of the dental team should also consider the possibility that other factors might be contributing to the disease process, such as undiagnosed diabetes or smoking.
        5. In some patients, the nonresponsive disease sites may also require the use of antimicrobial agents.
        6. Nonresponsive sites with more severe periodontal destruction will require more advanced treatment, such as periodontal surgery.
    2. **Performing Additional Nonsurgical Therapy.** At the reevaluation stage, a clinician must come to a decision as to whether additional nonsurgical periodontal therapy is warranted. This decision is based on several factors:
        1. Self-care efforts by the patient, though improved, may not be adequate for control of inflammation.
        2. Subgingival plaque biofilm and/or calculus deposits are difficult to remove, especially in the presence of inflammation resulting in edematous tissue.
        3. An unsuspected systemic condition may be contributing to the disease process.

c. **Establishing a Program for Periodontal Maintenance**
   1. When the patient's periodontitis condition has been brought under control, all patients should be enrolled in a periodontal maintenance program.
   2. Periodontal maintenance includes all measures used by the dental team and the patient to keep periodontitis under control. Periodontal maintenance is discussed in Chapter 31.
   3. The goal of periodontal maintenance is to prevent the recurrence of periodontal diseases.
   4. At each periodontal maintenance recall visit, the dental practitioner performs a clinical reassessment of the periodontium to determine if the periodontitis is controlled. If the periodontium appears healthy and the periodontitis remains arrested, periodontal maintenance should be performed. However, if the periodontium is inflamed and there are signs of disease reoccurrence, then site-specific or full-mouth periodontal instrumentation should be performed. If the disease has severely deteriorated, then referral to a periodontist for evaluation is warranted.

d. **Recognizing the Need for Periodontal Surgery**
   1. The need for some types of periodontal surgery can often be determined at the time of the initial periodontal assessment.
   2. However, the decision to proceed to periodontal surgery is frequently made at the time of the reevaluation. Periodontal surgery and its indications are discussed in Chapter 30.

The Relationship of Nonsurgical Reevaluation to Other Steps in Treatment

Figure 25-7 illustrates how reevaluation of nonsurgical periodontal therapy relates to the other steps in the overall management of a periodontal patient.

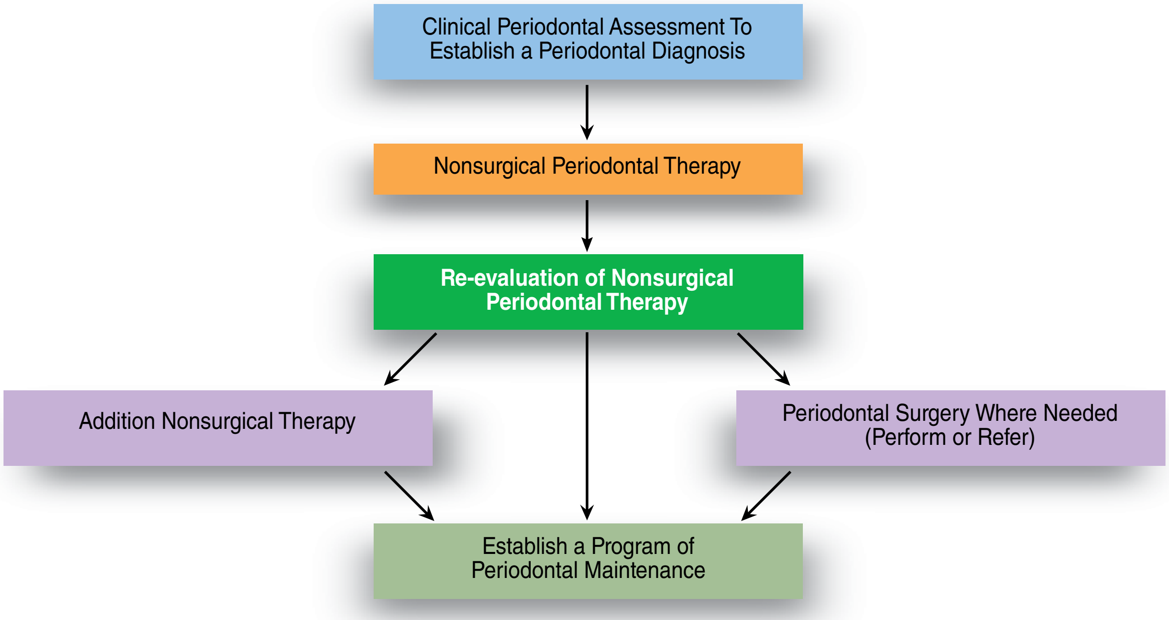
Clinical Periodontal Assessment To Establish a Periodontal Diagnosis
Nonsurgical Periodontal Therapy
Re-evaluation of Nonsurgical Periodontal Therapy
Addition Nonsurgical Therapy
Periodontal Surgery Where Needed (Perform or Refer)
Establish a Program of Periodontal Maintenance

Figure 25-7 How Reevaluation of Nonsurgical Periodontal Therapy Relates to Other Steps in the Overall Management of a Periodontal Patient. Reevaluation of nonsurgical therapy would be performed following the therapy. Decisions made at the reevaluation will be 1) nonsurgical *re*-instrumentation, 2) periodontal surgery, or 3) periodontal maintenance.

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The Relationship Between Nonsurgical and Surgical Therapy

Surgical therapy (also known as periodontal surgery) can play an important role in therapy for certain patients. This topic will be discussed in-depth in Chapter 30. However, at the time of reevaluation, members of the dental team should be aware of the general relationship between nonsurgical periodontal therapy and surgical periodontal therapy. Further, all team members should be able to explain to the patient the rationale and indications for periodontal surgery.

1. **Indications for Surgical Therapy.** As a general rule, periodontal surgery will be needed for patients with more advanced periodontal conditions. **Table 25-2** shows an overview of the general relationship of nonsurgical periodontal therapy to surgical periodontal treatment for patients with various periodontal conditions.

Table 25-2 Indications for Nonsurgical and Surgical Therapy

Disease Status	Nonsurgical Therapy	Surgical Therapy
Dental biofilm–induced gingivitis	Always indicated	Usually not indicated
Stage I periodontitis	Always indicated	Usually not indicated
Stage II periodontitis	Always indicated	Usually not indicated
Stage III periodontitis	Always indicated	Sometimes indicated
Stage IV periodontitis	Always indicated	Usually indicated

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   a. **Patients with Dental Biofilm–Induced Gingivitis.** For most patients with dental biofilm–induced gingivitis, the gingivitis can be controlled solely with nonsurgical therapy and reinforcement of oral hygiene instructions alone; only rarely will periodontal surgery be a part of the treatment recommended.
   b. **Patients with Stage I or Stage II Periodontitis.** For most patients with Stage I or Stage II periodontitis, the periodontitis can be controlled with nonsurgical therapy alone and reinforcement of oral hygiene instructions; only occasionally will periodontal surgery be a part of the treatment recommended.
   c. **Patients with Stage III Periodontitis.** For some patients with Stage III periodontitis, the periodontitis can be controlled with nonsurgical therapy alone. For other patients, control of the periodontitis will require thorough nonsurgical periodontal therapy followed by periodontal surgery.
   d. **Patients with Stage IV Periodontitis.** For most patients with Stage IV periodontitis, control of the periodontitis will require thorough nonsurgical periodontal therapy followed by periodontal surgery.
2. **Exceptions.** There will always be exceptions to the general guidelines described above.
   a. One example of an exception would be a patient with moderate dental biofilm–induced gingivitis where the gingivitis has resulted in disfiguring gingival enlargement. It is common for some types of gingival enlargement to require periodontal surgery to eliminate the disfigured enlargement, improve esthetics, or provide for improved access for effective self-care. This type of surgical therapy is known as a gingivectomy. A patient with drug-induced gingival enlargement, for instance, would benefit from undergoing a gingivectomy. However, as all periodontal therapies, long-term benefit can only be sustained with effective patient motivation and continuous reinforcement of patient self-care.
   b. Another example of an exception to the guidelines would be a patient with Stage I, Grade A periodontitis accompanied by severe gingival recession on a tooth. Although surgery is not normally needed in patients with Stage I periodontitis, periodontal surgery may well be needed to correct the mucogingival deformity (i.e., gingival recession defect) and/or address the patient's chief complaints (typically related to gingival esthetics or thermal sensitivity).

Recognizing the Need for Referral to a Periodontist

Although the general dental team can and should treat most patients with dental biofilm–induced gingivitis and most patients with periodontitis, the need for referral of some patients from a general dental office to a periodontist is always a possibility that should be considered. Making the important decision of where the patient should receive periodontal therapy is not always easy. The members of the team should discuss this issue in detail to determine the comfort level for treating periodontitis patients within the general dental practice. There are occasions when, due to the extent and complexity of a case, a referral to a periodontist is indicated. **Box 25-4** lists some of the primary reasons to refer to a periodontist.[46–51]

Box 25-4 Clinical or Risk Factors Indicating Patients Who Would Likely Benefit from Comanagement by a Periodontist

- Unresolved inflammation or continued loss of bone and/or attachment, despite nonsurgical periodontal therapy
- Managing a Stage III or Stage IV periodontitis that exhibits severe periodontal destruction
- Intensive management of a Grade B or Grade C periodontitis
- Gingival conditions that are not dental biofilm–induced and require specialist care, such as treatment of gingival recession defects attributed to nonperiodontitis reasons or cases that involve mucocutaneous disorders such as pemphigus vulgaris or lichen planus
- Patients requiring surgical procedures involving tissue augmentation or regeneration, including management of mucogingival problems, infrabony defects, and furcation invasions
- Patients requiring surgery involving bone removal (e.g., crown lengthening)
- Patients requiring surgery associated with osseointegrated implants
- Patients with medical history that significantly affects medical management (e.g., head/neck radiotherapy, intravenous bisphosphonate therapy, bleeding disorders)
- Patients that the general dental office team does not feel comfortable treating, for any reason

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Certain patients may benefit from comanagement by a general dental practice and periodontal office. **Comanagement** is defined as the shared responsibility and accountability for the care of a patient. Referral of patients with periodontal problems to a periodontist depends on several factors, including:

- The severity of disease and complexity of treatment required
- The patient's desire to see a specialist or undergo specialist treatment
- The general practitioner's knowledge, experience, and training to treat patients with a range of periodontal problems
- The presence of other complicating factors, such as the patient's medical history

Chapter Summary Statement

Nonsurgical periodontal therapy refers to all the initial steps used by the dental team to bring gingivitis and periodontitis under control. The goals of nonsurgical periodontal therapy are to control the bacterial challenge to the patient, to minimize the impact of systemic risk factors on the periodontium, to eliminate or control local intraoral factors, and to stabilize the attachment level. The precise steps included in nonsurgical periodontal therapy should depend on the specific needs of each individual patient.

A vital component of a plan for nonsurgical periodontal therapy is periodontal instrumentation. Since biofilms are resistant to topical chemical control, mechanical periodontal instrumentation of subgingival root surfaces is an essential component of successful nonsurgical periodontal therapy. Dentinal hypersensitivity may occur following nonsurgical periodontal therapy. Thorough daily self-care is the most important factor in the prevention and control of hypersensitivity, but some patients with dentinal hypersensitivity will need additional measures.

Reevaluation is an important step in nonsurgical periodontal therapy. During reevaluation the dental team determines the patient's need for additional nonsurgical therapy, referral of the patient for periodontal surgery, or enrollment of the patient in a periodontal maintenance program.

Section 4

Focus on Patients

Clinical Patient Care

CASE 1

Mr. H just returned for a 6-week reevaluation appointment following nonsurgical periodontal therapy for periodontitis. Clinically, Mr. H exhibits poor tissue healing and generalized plaque biofilm.

Although the patient denies having diabetes, the patient does report having several close family members with this disease.

Make a list of steps your dental team might include in an appropriate plan for nonsurgical periodontal therapy for this new patient.

CASE 2

At the time of reevaluation for a patient with a diagnosis of periodontitis, your dental team identifies a few sites of residual subgingival calculus deposits and documents totally ineffective patient self-care. How should the members of your dental team manage the oral health needs of this patient?

CASE 3

One of your patients who has been undergoing nonsurgical periodontal therapy seems a bit upset when he comes in for another appointment. After being seated in your treatment room, he states that he gets a sharp pain in his teeth when he tries to eat his favorite desert, ice cream. How should you proceed based upon the patient's complaint?

CASE 4

Mr. B is a 63-year-old patient who presents to your office for a comprehensive periodontal evaluation. He reports a history of leukemia that he is currently managing with his physician. During your examination, you note that the gingiva generally appears swollen with spontaneous hemorrhaging. What steps would you take with his physician to coordinate proper management of his dental care?

Ethical Dilemma

You are a retired librarian who just recently lost your husband. You grew up, went to college, married and raised a family all in the same town in which you were born. You are fortunate to be surrounded by many family members and long-time friends.

You are currently a patient in Dr. Jay Mack's periodontal practice, and have been for the last 15 or so years. You see Evelyn, the older of the two hygienists in the practice, every 3 months. The only supplemental aids she has suggested that you use, in addition to tooth brushing, are "tufted floss and a tongue scraper."

You are very active in your church and community. At your weekly bridge club gathering, several other participants mention that they also are patients in Dr. Mack's practice. Further discussions revealed that all the patients who saw Evelyn were instructed to use tufted floss and tongue scrapers for their self-care. However, those patients who saw Anne, the younger hygienist, were instructed to use a variety of self-care aids. It sounds like Anne recommends different aids based on each individual patient's needs. Your bridge partner, Florence who also sees Evelyn, the hygienist, says her teeth are very sensitive to hot, cold, and sweets, yet has only been instructed and educated in the use of tufted floss and a tongue scraper as well.

You wonder why so many patients with differing needs receive the same instructions from the dental hygienist, Evelyn.

1. What ethical principles are in conflict in this dilemma?
2. Should you meet with Dr. Mack to discuss your concerns?
3. How should this ethical dilemma be handled?

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CHAPTER 26

Patient's Role in Nonsurgical Periodontal Therapy

- Section 1 Patient Self-Care in Nonsurgical Therapy
- Section 2 Types of Toothbrushes
  - "Which Type of Toothbrush Should I Use?"
  - "How Should I Brush My Teeth?"
- Section 3 The Effectiveness of Various Interproximal Cleaning Modalities
- Section 4 Tongue Cleaning as an Adjunct
- Section 5 Focus on Patients
  - Clinical Patient Care
  - Evidence in Action
  - Ethical Dilemma

CLINICAL APPLICATION

In all cases, patient self-care is paramount to not only attaining good oral hygiene for the *short term*, but also preserving good oral hygiene for the *long term*. In a highly motivated patient who is periodontally healthy *and* fully compliant to adhering to daily self-care measures, this is an achievable task. In an individual with periodontal disease, however, considerable challenges and barriers may arise that thwart the patient's best self-care efforts. Some of these challenges include open embrasure spaces, exposure of root concavities, and attachment loss, as well as lack of patient ability and motivation. This chapter introduces the oral health care provider to many of the well-accepted patient self-care regimens and cleaning modalities, along with guidance to planning and tailoring a customized self-care approach that targets the specific characteristics of an individual patient to ensure the best response with the highest margin of success.

LEARNING OBJECTIVES

- In a classroom or laboratory setting, explain how to select and demonstrate the use of the following to an instructor: manual toothbrush, power toothbrush, and various interdental aids.
- Explain why interdental care is of utmost importance for any patient.
- In a clinical setting, recommend, explain, and demonstrate appropriate interdental aids to a patient with Type III embrasure spaces. Assist the patient in selecting an appropriate interdental aid that the patient is willing to use daily.
- Explain how the presence of exposed root concavities in a dentition would influence your selection of effective self-care aids.
- State the rationale for tongue cleaning and in the clinical setting recommend and teach tongue cleaning to an appropriate patient.

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KEY TERMS

Partnering with the patient
Gingival embrasure space
Root concavity
Volatile sulfur compounds

Section 1

Patient Self-Care in Nonsurgical Therapy

Nonsurgical periodontal therapy includes all nonsurgical treatment and educational measures used to help control gingivitis and periodontitis, such as patient self-care, periodontal instrumentation, and chemical plaque control.

1. **Partnering with the Patient.** Because periodontal disease is a plaque biofilm-induced disease, a key element of nonsurgical periodontal is meticulous daily self-care by the patient.
    a. Successful nonsurgical periodontal therapy always involves the patient in a collaborative coaching program on self-care techniques.
    b. The patient's efforts at self-care are essential to disease control and the outcome of therapy. **Partnering with the patient** can help enhance compliance and motivation. Some dental teams refer to the patient as having the role of co-therapist in the process of nonsurgical periodontal therapy.
        1. The concept of partnering with the patient underscores the vital role the patient plays in the daily self-management of biofilm and the stabilization of periodontitis.
        2. Partnering ensures that the patient is actively involved in making decisions about his or her own health care and is willing to make a long-term commitment to meticulous self-care and regular professional care.
    c. Mechanical biofilm removal includes daily self-care efforts by the patient and supragingival/subgingival periodontal instrumentation by the dental provider at regular intervals.
2. **Self-Care**
    a. **What Is Self-Care?** There is no single accepted definition of self-care. One that has been developed for people with chronic diseases is "the actions individuals take to lead a healthy lifestyle; to meet their social, emotional, and psychological needs; to care for their long-term condition and prevent further illness or accident."[1] A recent systematic review found that periodontitis was associated with having a negative impact on the quality of life. The more severe the disease, the greater the impact, thus underscoring the value of daily self-care.[2]
        1. Self-care education involves instruction on the use and frequency of a product; for example, suggesting the addition of a product such as an interdental aid to the daily routine.
        2. The self-care plan should involve collaboration between the dental health care provider and patient.
            a. The dental hygienist needs to provide input and support while at the same time take into consideration the patient's interest and abilities. Optimally, the final decision on self-care devices is made collaboratively.
            b. Recommending a device that the clinician likes but that the patient dislikes will not contribute to patient motivation and compliance.
            c. Because collaboration hinges on good communication, the dental hygienist needs to provide education and instruction using empathy and words that the patient understands.
    b. **Goals of Self-Care.** The goal of any self-care regimen is to enhance oral health via optimal biofilm removal and the elimination of inflammation.
    c. **Self-Care Aids Employed in Biofilm Control**
        1. Toothbrushing is the most commonly used aid for biofilm removal. Generally, brushing is the only daily form of self-care performed by most patients. Interdental aids are necessary for most patients and critical for periodontal maintenance patients because the interdental area is generally not accessible to toothbrushing.
        2. Tongue cleaning on a daily basis helps control halitosis and may contribute to a healthy periodontal environment.

Section 2

Types of Toothbrushes

When it comes to self-care, a motivated patient will commonly ask the oral health care provider two questions: (1) "Which type of toothbrush should I use?" and (2) "How should I brush my teeth?" This section will tackle each of these questions so that the oral health care provider can rely on a sound basis to respond to these pertinent patient questions.

"Which Type of Toothbrush Should I Use?"

When it comes to responding to the question of which type of toothbrush the patient should use, there is no clear-cut winner. A 2014 systematic review that compared manual versus power toothbrushes gave the edge to the latter with respect to plaque removal over a 3-month period; however, the investigators were unsure of the clinical importance of these findings due to the lack of standardization of study design among existing research trials investigating this question.[3] In fact, time and research have shown that when it comes to plaque biofilm removal, both manual and power toothbrushes (also known as power-assisted, automatic, electrical, or mechanical toothbrushes) are equally effective when implemented correctly and on a daily basis. The American Dental Association's own recommendation reflects this balanced approach to the question at hand: when implemented correctly and on a routine basis, *both* manual and power toothbrushes are equally comparable at removing plaque.[4] Thus, the decision of which toothbrush to use should not be made without fully understanding the advantages and disadvantages of manual versus power toothbrushes and without fully taking account of individual patient-specific needs. Further, although many types of power toothbrushes are offered directly to consumer, the oral health care provider should convey to the patient that not all power brushes are alike. Below is a review of the different types of toothbrushes commonly available to the patient, grouped into two general categories: (1) manual toothbrushes and (2) power toothbrushes (**Figure 26-1**). For the sake of discussion, this section will solely focus on the four types of power toothbrushes and the modes of action for each.

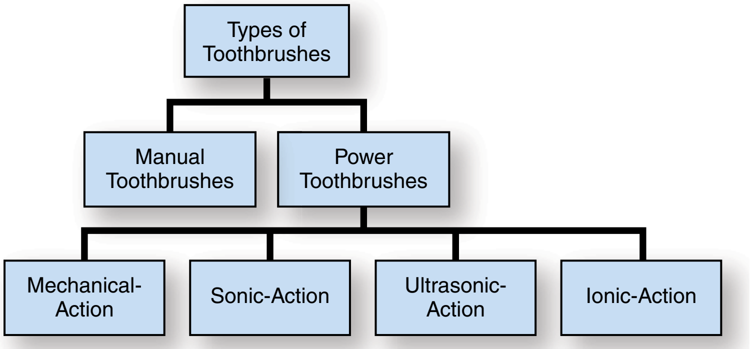

Figure 26-1 **Toothbrush Categories.**

1. A mechanical-action power toothbrush is a power-assisted (via electricity or an internal rechargeable battery) self-care device that comes equipped with a small head composed of nylon bristles or tufts. When activated, the bristles on the head rotate and/or oscillate like the spinning brushes in a car wash.
   a. The head of the mechanical toothbrush is attached to a handle containing a battery-powered electric motor that provides mechanical rotational/oscillation movement of the brush head with filaments moving rapidly.
   b. A mechanical-action toothbrush rotates at 1,000–8,000 movements per minute (**Figure 26-2A**).
2. A sonic-action power toothbrush is a type of power-assisted self-care device that emits sound waves. These sound waves produce a characteristic audible "humming" sound that is well within the hearing range of the human ear (at 20–20,000 Hz).
   a. These sound waves, in turn, induce the bristles on the head to vibrate rapidly in a side-to-side manner, thereby creating high-speed scrubbing strokes when engaging the surface of a tooth.

   b. In contrast to a mechanical-action toothbrush, conventional sonic-action power toothbrushes vibrate much more rapidly and range anywhere from 25,000–100,000 movements per minute, depending on the type of sonic toothbrush used (**Figure 26-2B**).
   c. One of the first sonic brushes introduced in the U.S. market was launched by Phillips Sonicare in the early 1990s.
3. An ultrasonic-action power toothbrush is another power-assisted self-care device that, from the outside, looks like a sonic-action toothbrush.
   a. However, unlike a sonic-action toothbrush, an ultrasonic-action toothbrush does not rely on the physical side-to-side motion of the bristles to dislodge the plaque biofilm or food debris. Rather, the handle of the ultrasonic-action toothbrush comes equipped with an embedded piezoelectric device that converts electrical energy to mechanical energy when activated. This mechanical energy, in turn, creates a high-frequency vibration called ultrasound, which breaks up plaque biofilm without physically moving the brush head.
   b. Similar to an ultrasonic scaler, cavitational effect, fluid streaming, and acoustic vibrations may also aid in loosening plaque biofilm from tooth surfaces.
   c. Compared to sonic-action toothbrushes, ultrasonic toothbrushes function at much higher frequency (20 kHz to 10 MHz), which is well beyond what humans can hear.
   d. Further, compared to mechanical-action and sonic-action toothbrushes, an ultrasonic-action vibrates close to a whopping 200 million movements per minute (**Figure 26-2C**)!

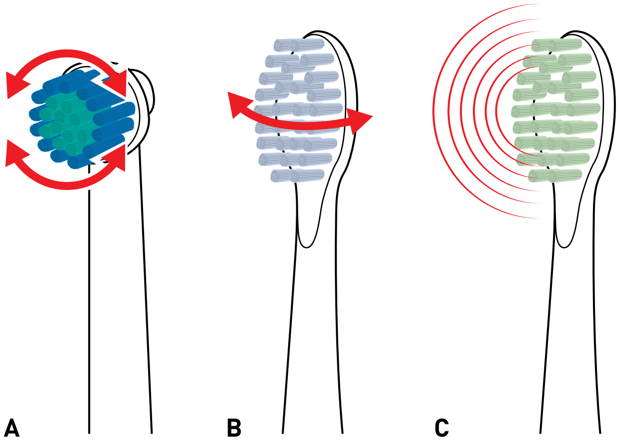

Figure 26-2 Key Differences Between Various Types of Power Toothbrushes. A. Mechanical-action toothbrush. The head rotates or oscillates to physically dislodge plaque biofilm and extraneous debris from the tooth surface. **B. Sonic-action toothbrush.** Instead of rotating or oscillating, the bristles move in a side-to-side manner and vibrate at high speeds. The more vibrations a toothbrush makes, the more brush motions the bristles make. **C. Ultrasonic-action toothbrush.** This brush emits a much higher frequency of vibration called ultrasound to loosen plaque biofilm and extraneous food debris from the tooth surface. Unlike the other two types of power-action brushes, an ultrasonic toothbrush does not rely on the physical movement of the head (like the rotating/oscillating movement of a mechanical-action toothbrush) or the bristles (like the bristles of a sonic-action toothbrush).

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4. The ionic toothbrush is a newer type of power-action toothbrush that functions on the fundamental physics principle that positive and negative charges attract (opposites attract) (**Figure 26-3**).
   a. Teeth naturally have a negative ionic charge, whereas plaque biofilm and food particles naturally have a positive ionic charge. This is why plaque biofilm and food particles cling so tightly to a tooth.

b. Embedded within the handle of the ionic toothbrush is a titanium battery that, when powered on, reverses the polarity of the tooth surface by temporarily altering the tooth's negative ionic charge to a positive charge. This change in polarity repels the positively charged plaque biofilm and food debris away from the positively charged tooth surface.
c. The bristles of the ionic toothbrush are positively charged, and thus attract the negatively charged plaque biofilm and food particles toward it. The mechanical action of the bristles contributes to loosening plaque biofilm and food away from the tooth surface.

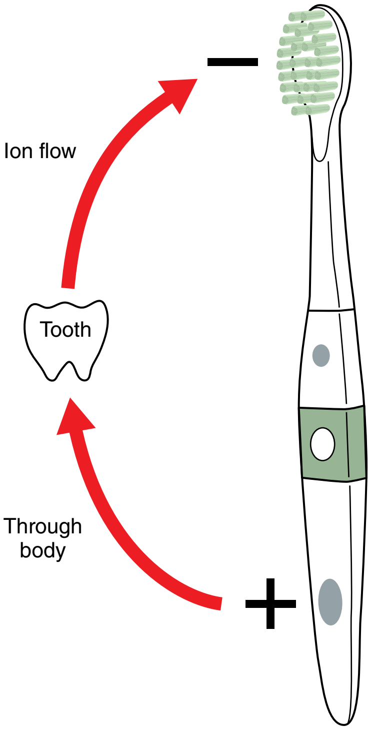

Figure 26-3 **Ionic Toothbrush.** When activated, an ionic toothbrush releases many positive charges which travel through the human body (since the individual is grasping onto the handle when the ionic toothbrush is in use), then affects the tooth by altering the polarity of its surface from a negative charge to a positive charge.

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5. Despite much research, results are still contradictory with respect to which type of toothbrush is superior. In fact, all toothbrushes effectively remove plaque biofilm and extraneous food debris *when carried out correctly by the patient*. In some cases, however, the patient and oral health care provider may decide that a power toothbrush is more user-friendly for individuals who have limited manual dexterity and/or motor skills or individuals who are cognitively impaired. In the end, regardless of which type of toothbrush is selected, the key to effective patient self-care may hinge on the patient's own actions in staying engaged in the self-care process and adhering to the recommended self-care regimen.

"How Should I Brush My Teeth?"

Although several well-established toothbrushing techniques are available to patients, there is currently no conclusive scientific evidence to support the superiority of one technique over the others. This is because no two patients and no two periodontal disease cases are alike. Thus, the "best" toothbrushing technique is often contingent on an array of factors unique to each patient. Hence, it is incumbent on the health care provider to identify these factors and implement a suitable toothbrushing strategy that is best customized to an individual patient's case. Included in this section is a list of various toothbrushing techniques. Toothbrushing techniques can be grouped into four general categories: (1) horizontal reciprocating motion, (2) vibratory motion, (3) vertical sweeping motion, and (4) rotary (circular) motion (**Figure 26-4**). The reader may refer to a textbook such as *Wilkins' Clinical Practice of the Dental Hygienist* for a thorough review of toothbrushing techniques.

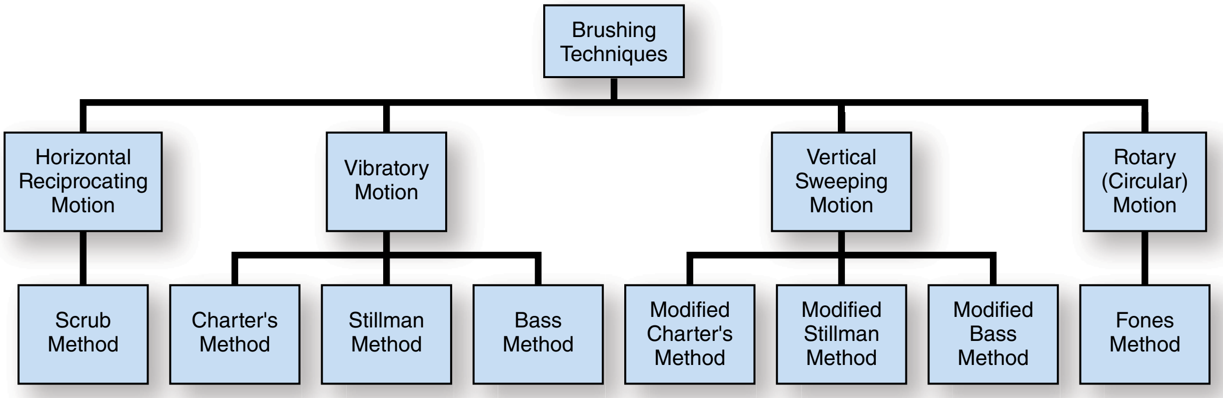
Brushing Techniques
Horizontal Reciprocating Motion
Vibratory Motion
Vertical Sweeping Motion
Rotary (Circular) Motion
Scrub Method
Charter's Method
Stillman Method
Bass Method
Modified Charter's Method
Modified Stillman Method
Modified Bass Method
Fones Method

Figure 26-4 **Four General Categories of Toothbrushing Techniques.**

Section 3

The Effectiveness of Various Interproximal Cleaning Modalities

Compared to self-care measures that aim to remove plaque biofilm off facial and lingual surfaces, interproximal self-care methods can pose significant challenges for some patients. Below is an outline of some of the challenges a patient may encounter when carrying out interproximal self-cleaning measures. Additionally, various types of interproximal self-cleaning instruments will be reviewed.

1. **Anatomical Challenges for the Patient with Periodontitis.** Due to attachment loss, the dentition of an individual with periodontitis often presents anatomical challenges to effective biofilm control, such as recession of the gingival margin, type of embrasure spaces, and exposed root concavities.
    a. **Gingival Embrasure Spaces.** The **gingival embrasure space** is the small triangular space (apical to the contact area) between the curved proximal surfaces of two teeth. The three types of embrasure spaces are shown in **Figures 26-5** to **26-7**.

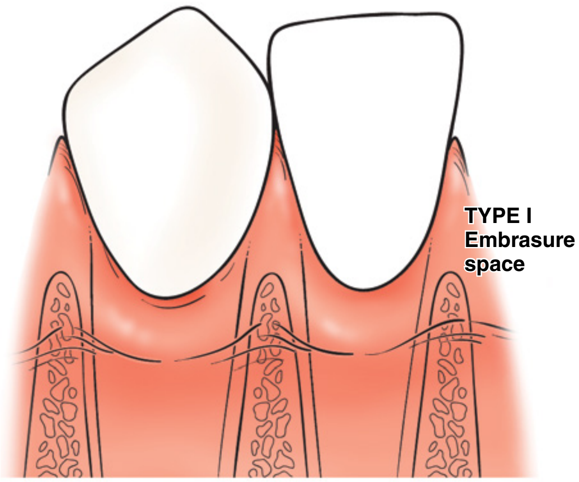
TYPE I
Embrasure
space

Figure 26-5 **Type I Embrasure.** With Type I embrasure, the interdental papilla completely fills the embrasure space. For the patient with excellent self-care compliance and good manual dexterity, dental floss is an effective means of biofilm control.

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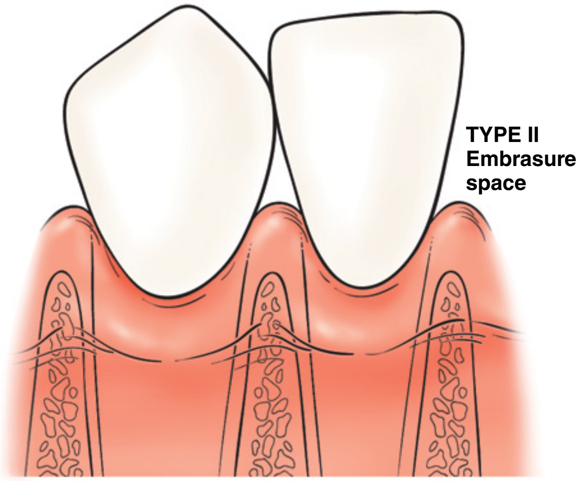
TYPE II
Embrasure
space

Figure 26-6 **Type II Embrasure**. With Type II embrasure, the interdental papilla partially fills the embrasure space. The loss of the papillary tip may be due to periodontitis or mechanical trauma. Interdental brushes, wooden toothpicks, or power-driven flossers (like water flossers and air flossers) are effective means of biofilm control.

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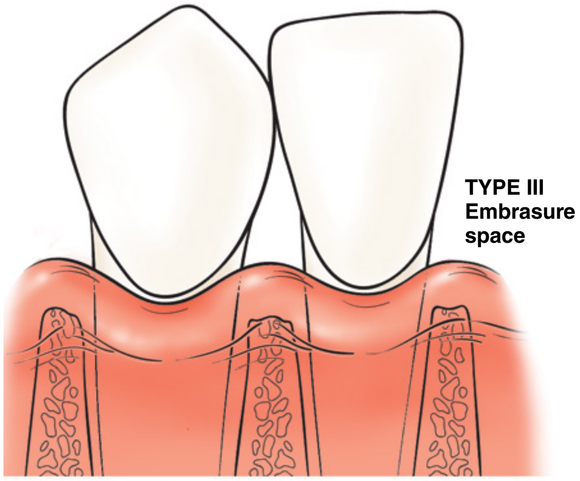

Figure 26-7 Type III Embrasure. Type III embrasure is characterized by complete loss of the interdental papilla due to periodontitis or mechanical trauma. Interdental brushes, end-tuft brushes, or power-driven flossers (like water flossers and air flossers) are effective means of biofilm control.

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1. In health, the interdental papilla fills the gingival embrasure space. Dental floss is most effective in areas of normal gingival contour.
2. The tissue destruction characterized by periodontitis usually results in an interdental papilla that is reduced in height or missing, resulting in an open embrasure space. Mechanical trauma that traumatizes the interdental papilla can also result in a Type II or Type III embrasure. Dental floss is not effective in areas with open embrasure spaces.
3. Analysis of the gingival embrasure spaces is critical in determining which interdental aid is likely to be most effective in control of plaque biofilms. **Table 26-1** summarizes aids for interdental biofilm removal.

Table 26-1 Aids for Interdental Plaque Biofilm Removal

Interdental Aid	Description/Example	Indications for Use
Dental floss	Unwaxed or waxed thread made of silk, nylon, or plastic monofilament fibers	A patient with Type I embrasure spaces and excellent compliance to self-care regime
Floss holders	Handheld device to hold the floss or single-use device containing a segment of dental floss (Reach Access Flosser, Glide Floss Picks)	A patient with Type I embrasure spaces who is motivated but has dexterity issues
Tufted dental floss	Thickened yarn-like dental floss (J&J Super Floss)	Type II embrasure spaces, fixed bridges, distal surface of last tooth in the arch, proximal surfaces of widely spaced teeth
Interdental brush	Tiny nylon brushes on a handle (Butler GUM Proxabrush) Some brands come in varying sizes (TePe Interdental Brushes)	Type II or Type III embrasure spaces, distal surface of last tooth in the arch, exposed furcation areas that permit easy insertion of the brush, embrasure spaces with exposed proximal root concavities
End-tuft brush	Small bristle tuft on a toothbrush-like handle (Butler GUM End-Tuft Toothbrush)	Type III embrasure spaces, distal surface of the last tooth in arch, lingual surfaces of mandibular teeth, crowded or misaligned teeth, exposed furcation areas

(*continues*)

Table 26-1 **Aids for Interdental Plaque Biofilm Removal** *(continued)*

Interdental Aid	Description/Example	Indications for Use
Pipe cleaner	Standard pipe cleaner cut into 3-inch lengths	Type III embrasure spaces, exposed furcation areas that permit insertion
Toothpick in holder	A round toothpick in a plastic handle (Marquis Perio-Aid)	Type II or III embrasure spaces, biofilm removal at gingival margin, furcation areas or root concavities
Triangular wooden wedge	A triangular-shaped toothpick generally made of basswood (J&J Stim-U-Dent)	Type II or III embrasure spaces
Power-driven water irrigation device (also known as a water flosser)	A power device that uses a combination of pulsation and pressure to create shear hydraulic forces for plaque removal (see Chapter 27)	Type I, II, and III embrasures
Power-driven air floss (also known as an air flosser)	Similar to a water flosser, but instead of solely relying on water, an air flosser emits small spurts of compressed air in between teeth interspersed by short bursts of microdroplets of water or mouth rinse	Types I, II, III embrasures

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b. **Exposed Root Concavities.** A **root concavity** is a trench-like depression on the root surface. Root concavities commonly occur on the proximal surfaces of anterior and posterior teeth and the facial and lingual surfaces of molar teeth.

1. In health, root concavities are covered with alveolar bone and help to secure the tooth in the bone.
2. Periodontitis results in the apical migration of the junctional epithelium, loss of connective tissue, and destruction of alveolar bone. This tissue destruction results in the exposure of root concavities to the oral environment (either in the presence of tissue recession or, frequently, within a periodontal pocket).
3. **Figure 26-8** shows the root surface of a mandibular canine covered in a colored powder; the colored powder represents plaque biofilm. **Figure 26-9** demonstrates the ineffectiveness of dental floss in cleaning the root concavity. **Figure 26-10** demonstrates the effectiveness of an interdental brush in cleaning the root concavity. Note that only the interdental brush is effective in reaching the concave surface of the root concavity.

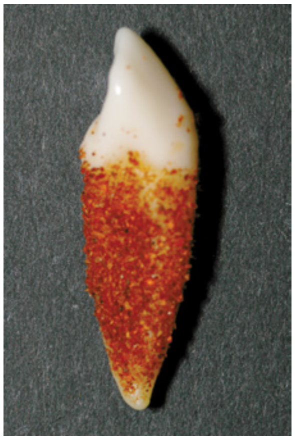

Figure 26-8 **Anatomical Challenge: Biofilm Removal from the Root Concavity.** The proximal surface of this mandibular canine is covered with colored powder representing plaque biofilm. Figures 26-9 and 26-10 compare the effectiveness of dental floss and an interdental brush in removing biofilm from an exposed root cavity.

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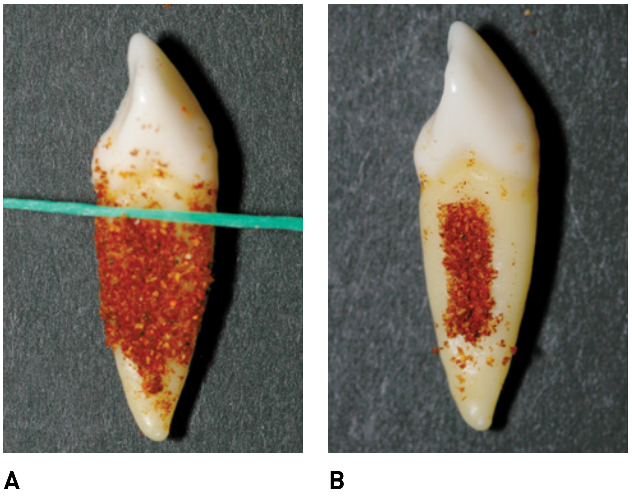

Figure 26-9 Use of Dental Floss for Biofilm Removal. As seen in panels **A** and **B**, dental floss is not effective in removing the powder (simulated biofilm) from the exposed root concavity.

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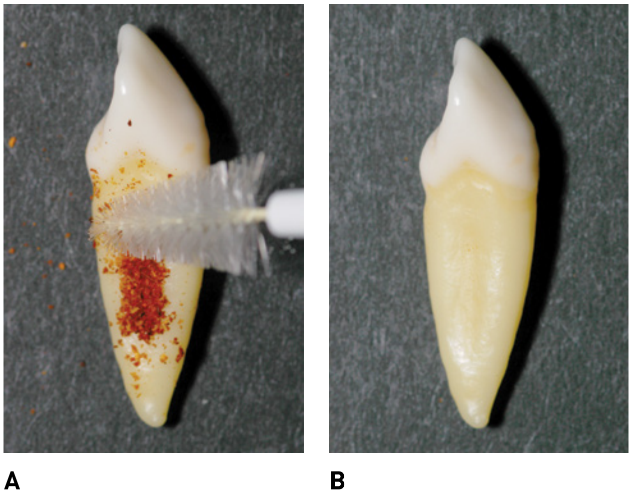

Figure 26-10 Use of an Interdental Brush for Biofilm Removal. As seen in panels **A** and **B**, an interdental brush effectively removes the powder (simulated biofilm) from the root concavity.

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2. **Selecting Interdental Aids for the Patient with Periodontal Disease**
   a. According to a 2015 online survey conducted by the American Academy of Periodontology involving over 2,000 adults in major U.S. markets, more than a quarter of the respondents admitted to being dishonest with their oral health provider about flossing habits.[5] Interestingly, respondents in this study reported that they would rather do an unpleasant activity over daily flossing such as cleaning the toilet (14% of respondents), washing a sink (18% of respondents), or waiting in a long checkout line (14% of respondents).
   b. A clinical practice guideline developed by the European Federation of Periodontology on treating periodontitis concluded that flossing should not be recommended as the first choice for periodontal maintenance patients. Hence, several studies suggest that interdental brushes and/or oral irrigators (Chapter 27) might be a better choice in reducing inflammation due to their ease of use.[6,7]
   c. Recently, a new type of power-driven interdental device, known as an air flosser, has been introduced in the market. This device emits rapid burst of air interspersed with microdroplets of water to physically disrupt the plaque biofilm and loosen it from the surfaces of teeth. In a head-to-head comparison of two leading types of powered interproximal devices (air flosser vs. water flosser), a water flosser was shown to be more effective than an air flosser in reducing gingival inflammation and plaque biofilm.[8] However, additional investigations are ongoing to determine whether one type of power-driven interdental device is superior to the others. Thus, the decision to use one type of interdental aid over the other should be made when taking other patient-related factors such as patient preference, gag reflex, dexterity, compliance, and cognitive abilities into account.
   d. **Interdental Brush**
      1. **Description**
         a. An interdental brush is a tiny cylindrical or conical "pine tree"–shaped nylon bristle brush attached to a handle (**Figure 26-11**). Brushes are available in different diameters so that the best size can be selected.
         b. The size of the embrasure space determines the type of interdental brush that should be used.
         c. There should be a slight bit of resistance as the interdental brush is moved back and forth between the teeth. Often, it is necessary to use different size interdental brushes due to varying sizes of embrasure spaces within the same mouth.

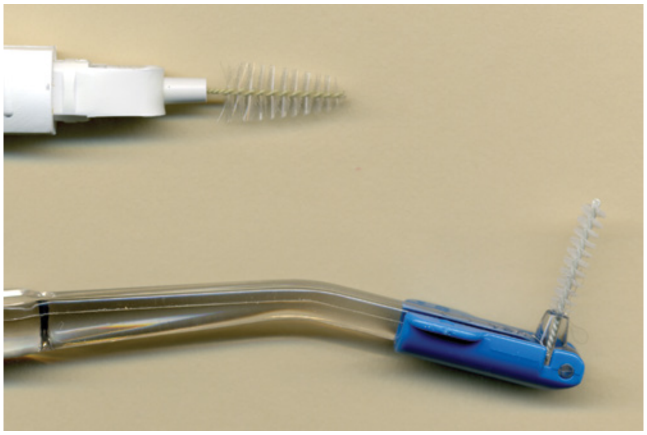

Figure 26-11 **Interdental Brushes.** Interdental brushes are one of the most useful aids for cleaning root concavities.

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2. **Indications**
   a. A systematic review of interdental brushes found that interdental brushes are more effective than dental floss in reducing plaque and gingival inflammation in periodontal maintenance patients.[9,10]
   b. A cylindrical brush has been shown to be more effective for plaque removal than a conical shaped brush.[10]
   c. Interdental brushes are recommended for biofilm removal in Type II and III embrasure spaces (**Figure 26-12**).
   d. The bristles of an interdental brush are very effective at cleaning root concavities.
3. **Technique.** The brush is inserted into the open interdental space, then gently slid in and out of the embrasure space with several strokes. *Always use the interdental brush without toothpaste. Toothpaste has abrasive ingredients, such as chalk and silica, that could traumatize the fine, delicate interdental papilla.* **Figure 26-13** shows techniques for use of interdental brushes. In a Type II embrasure space, care should be exercised to avoid vigorous brushing so as to avoid further traumatizing the interdental papilla.

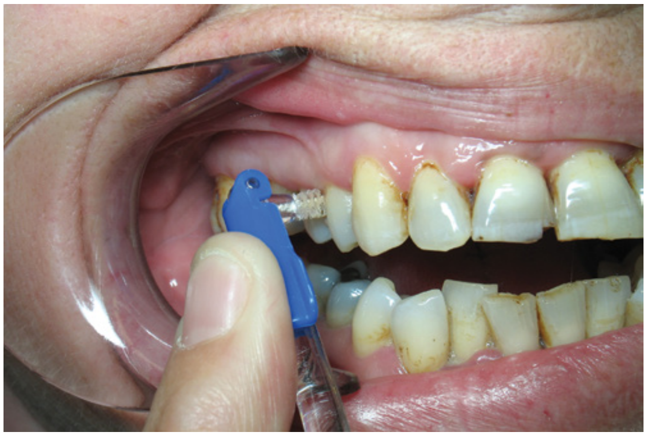

Figure 26-12 **Use of Interdental Brush.** The brush is inserted into the open interdental space, then gently slid in and out of the embrasure space for several strokes. Always use the interdental brush without toothpaste.

Courtesy of Dr. Deborah Milliken, South Florida State College.

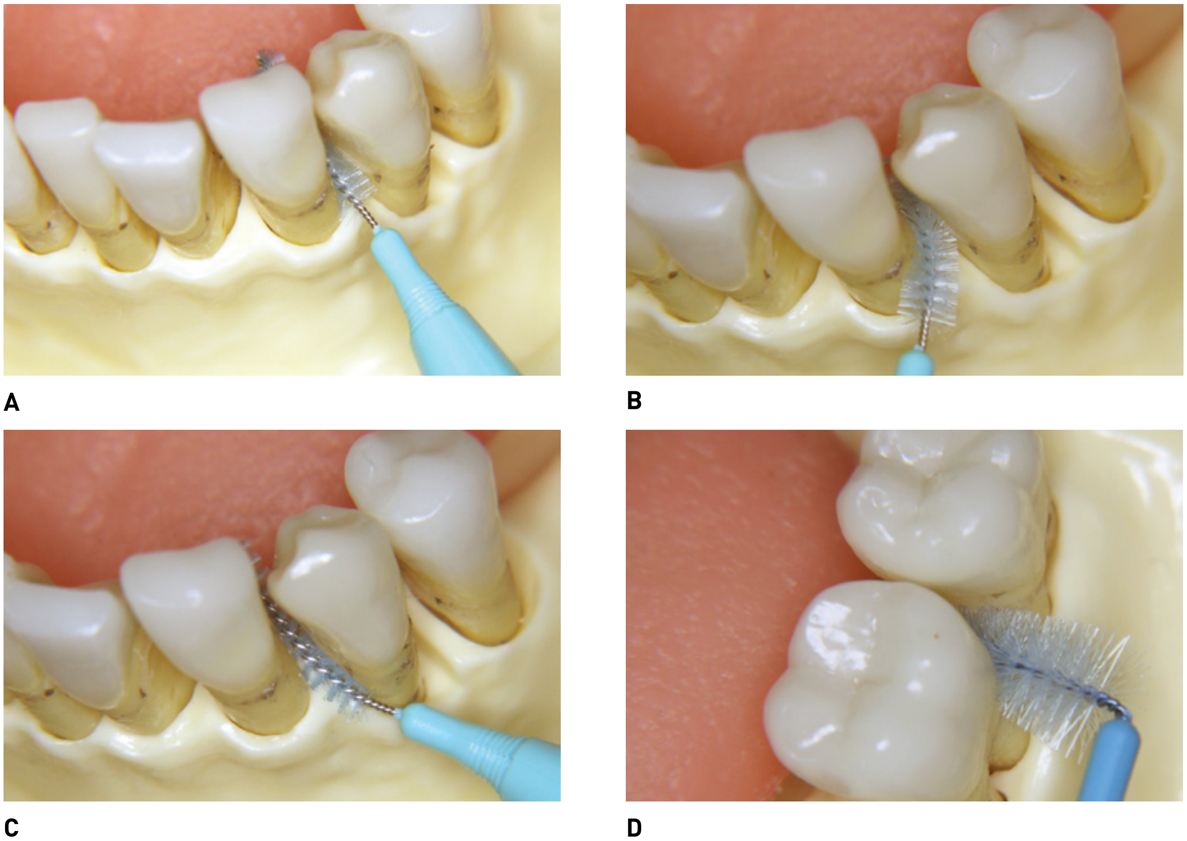

Figure 26-13 **Steps for Use of an Interdental Brush. A.** The brush handle is held between the thumb and index finger and the brush gently inserted between the teeth. The brush should be maintained at a 90-degree angle to the long axis of the tooth. **B.** The bristles are adapted to the interproximal surfaces with slight pressure and varying the angle of insertion. For optimal biofilm removal, the brush is slid in and out of the space using the entire length of the bristle part of the brush. **C.** By changing the angle of insertion, the bristles can be adapted to the mesial surface of the first premolar. Slight pressure with the brush against the gingiva allows the bristles to clean slightly beneath the gingival margin. **D.** For posterior areas, advise the patient to close his or her mouth slightly to relax the cheek. The brush may be bent to facilitate insertion between posterior teeth.

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e. End-Tuft Brush

1. **Description.** An end-tuft brush is like a standard toothbrush except that the brush head has only a small tuft of bristles (**Figure 26-14**). A standard toothbrush easily can be modified to create a customized end-tufted brush by removing some of the bristles.

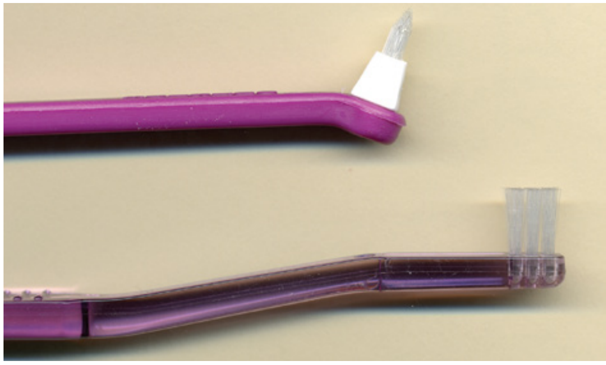

Figure 26-14 **End-Tuft Brushes.** End-tuft brushes are used to clean areas that are difficult to access with a standard brush.

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2. **Indications**
    a. End-tuft brushes can effectively reach sites around teeth that are difficult for patients to clean, such as the distal surface of the last tooth in the arch, lingual surfaces of mandibular teeth, and crowded or misaligned teeth (**Figure 26-15**).

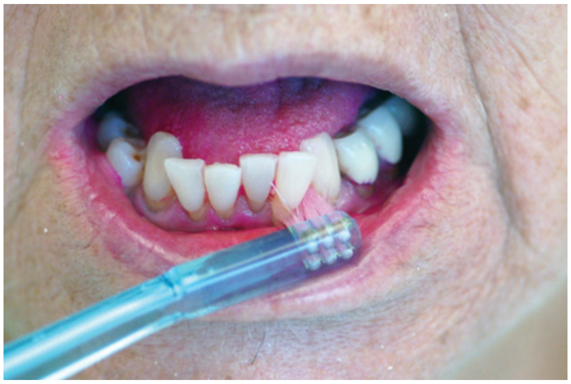

Figure 26-15 Use of End-Tuft Brush. An end-tuft brush used around crowded anterior teeth.

Courtesy of Dr. Deborah Milliken, South Florida State College.

    b. Works well to remove biofilm from Type III embrasure spaces. Useful in removing biofilm from an exposed furcation area because the small size of the bristle tufts allows them to partially enter the furcation site.
3. **Technique**
    a. The end tuft is directed into the embrasure space or furcation area. Gentle circular strokes are used to clean the area.
    b. For difficult-to-reach mandibular lingual tooth surfaces, the brush is used like a standard brush with a sulcular brushing technique.

f. **Dental Floss**
1. **Description.** Dental floss is unwaxed or waxed thread made of silk, nylon, or plastic monofilament fibers used to remove dental plaque biofilm from the proximal surfaces of teeth.
2. **Effectiveness.** Although floss tends to be the primary recommendation of most dental hygienists, floss works better for patients with healthy gingiva versus patients with periodontitis or maintenance patients.[6] Furthermore, when a patient flosses, the effectiveness of dental floss is low because of its difficulty to perform—in fact, a significant number of those who do floss are not able to perform the function effectively.[11]
    a. Evidence indicates that a variety of alternative interdental aids are as effective or more effective than dental floss in removing biofilm and in reducing bleeding and gingivitis.[6,7,9]
    b. When added to toothbrushing, a 14-day study found that dental floss has been shown to significantly reduce bleeding compared to toothbrushing alone.[12]
    c. A systematic review of dental floss added to manual toothbrushing found that in most cases the addition of dental floss does not increase biofilm removal or reduce gingival inflammation. The authors conclude that practitioners need to determine on an individual basis whether effective flossing is a reasonable and achievable goal.[13]
    d. A systematic review looked at the ability of dental floss to reduce interproximal caries. Even though conventional wisdom has long advocated for flossing, the review found mixed evidence on the ability of flossing to reduce decay in children. The review also found that there have been no scientific studies conducted on adults to evaluate the ability of dental floss to reduce or prevent interproximal decay. The absence of this evidence does not mean flossing may not have an effect on interproximal decay. Rather, it means that flossing has not been proven to prevent decay.[14]
    e. It may be easier for some individuals to use floss via a floss holder. Flossing via floss holder has been shown to be as effective as handheld floss due to its ease-of-use.[15] Many variations of floss holders are available. Some require manually wrapping the floss onto the holder. Others may be single-use devices with the floss already in place (**Figure 26-16**).

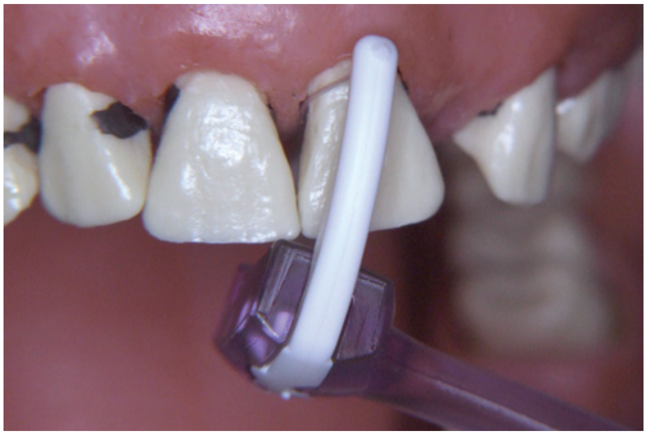

Figure 26-16 Technique for Use of Floss Holder. Flossing via a floss holder is as effective as handheld flossing, provided the patient uses proper technique. The dental floss should be wrapped around the proximal tooth surface in a similar manner to the technique employed with handheld floss.

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    f. When given a choice, patients often prefer interdental cleaning aids that are easier to use than dental floss.
    g. Flossing may not be as effective for certain periodontal patients; recession, attachment loss, and size of the gingival embrasure space are limiting factors.[6]
    h. A water flosser has also been shown to be an effective alternative to dental floss, appropriate for patients with many types of oral anatomy and conditions.[6,7] Irrigation is discussed in detail in Chapter 27.
    i. Some mouth rinses have been shown to be an effective alternative to dental floss. Mouth rinses are discussed in detail in Chapter 28.
  3. **Indications for Recommendation of Dental Floss**
    a. **Type I Embrasures.** Dental floss is effective in removing biofilm from tooth crowns and the convex root surfaces in the region of the cementoenamel junction (CEJ). Dental floss is not effective in removing biofilm from root concavities and grooves.
    b. **Excellent Self-Care.** Dental floss is recommended for patients with excellent compliance with self-care. Unfortunately, patient compliance with dental flossing is low, with many patients being unable or unwilling to perform daily flossing.[6]
    c. Patients who participate in crafting hobbies such as knitting, crocheting, needlepoint or woodworking may be good candidates for dental floss as they are likely more adept and comfortable at using their hands.

g. **Tufted Dental Floss**
  1. **Description.** Tufted dental floss is a specialized type of dental floss that has a segment of ordinary floss attached to a thicker, fluffy, yarn-like segment of floss (**Figure 26-17**).
  2. **Indications**
    a. For Type II embrasures.
    b. To clean under the pontic of a fixed bridge.
    c. To clean the distal surface of the last tooth in the arch.
    d. To remove plaque biofilm from the proximal surfaces of widely spaced teeth.
  3. **Technique**
    a. For interdental proximal surfaces, the fluffy part of the floss is used interdentally in a C-shape against the tooth, applying light pressure with a slight sawing motion against first one proximal surface and then the adjacent proximal tooth surface.
    b. For fixed bridges, the tufted floss is threaded under the pontic and used to clean the undersurface of the pontic. Next, the distal surface of the mesial abutment tooth and the mesial surface of the distal abutment tooth are cleaned using the tufted floss.

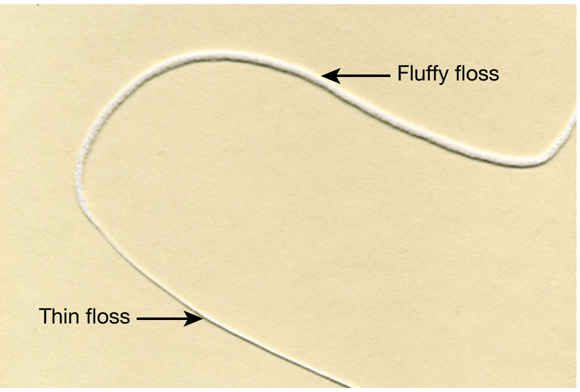

Figure 26-17 Tufted Dental Floss. This aid is a specialized type of floss consisting of a fluffy segment of yarn-like floss attached to a segment of thin floss.

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h. **Wooden Toothpick in a Holder**
   1. **Description.** This device consists of a round toothpick in a plastic handle.
   2. **Indications**
      a. The toothpick in a holder has been shown to reduce biofilm and bleeding as effectively as dental floss.[16]
      b. It can be used gently along or slightly below the gingival margin or directed into exposed furcation areas for biofilm removal.
      c. Effective in Type II embrasures, if the toothpick is easily inserted between the teeth; however, this is not an effective aid in cleaning root concavities unless the teeth are widely spaced.
   3. **Technique for Use of a Wooden Toothpick in a Holder**
      a. A toothpick is secured in the holder and the long end is broken off flush with the holder so that it will not scratch the inside of the cheek (**Figure 26-18**).
      b. The end of toothpick is moistened with saliva.
      c. The toothpick tip is applied at right angles to the tooth or directed just beneath the gingival margin at a less than 45-degree angle. The tip should not be directed against the epithelial attachment. The tip is used to trace the gingival margin around each tooth.
      d. Where space permits, the tip is angled into embrasure spaces or exposed furcation areas and moved gently back and forth to remove accumulated biofilm.

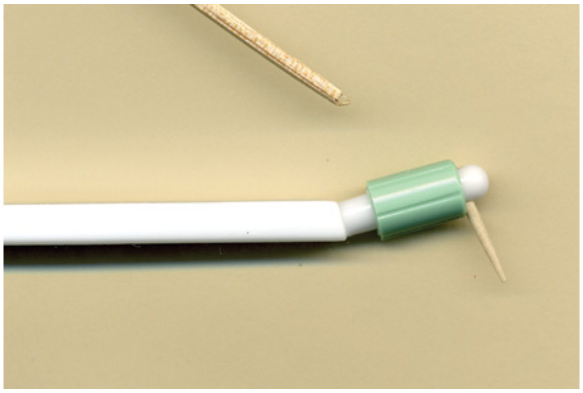

Figure 26-18 Toothpick Holder. To prepare this aid for use, secure a toothpick in the holder and break off the long end so that it is flush with the plastic holder.

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i. **Wooden Wedge**
   1. **Description.** This is a short wooden stick usually made of soft wood. These wedges are triangular, wedge-shaped sticks and should not be confused with round or rectangular toothpicks.
   2. **Indications.** A systematic review of triangular wooden wedges found they did not increase the amount of visible biofilm removal beyond toothbrushing but they did improve interdental inflammation better than brushing alone.[17] To use a wedge, there must be sufficient interdental space to allow easy placement of the wooden wedge. Long-term use of wooden wedges in Type I embrasures may cause a permanent loss of the papillae.
   3. **Technique for Use of Wooden Wedge**
      a. The wooden wedge should be moistened thoroughly in the mouth to soften the wood prior to use.
      b. The wedge is then gently inserted between the teeth with the flat side next to the gums (**Figure 26-19**).
      c. The wedge is used with a gentle in and out motion to clean between the teeth. The wedge should not be forced into tightly spaced teeth. The wedge should be discarded as soon as the first signs of splaying are evident.

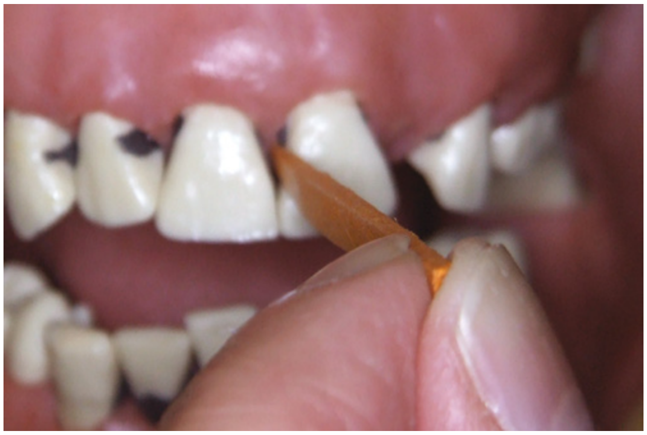

Figure 26-19 **Use of Wooden Wedge.** The wedge is held between the thumb and index finger with the flat side toward the gingiva. In the upper arch, the flat surface faces up, while in the lower arch the flat surface faces down.

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Section 4

Tongue Cleaning as an Adjunct

Many patients have coated tongues that make it difficult to maintain fresh breath and diminishes the sense of taste. Periodontal patients have been shown to have significantly higher prevalence of tongue coating.[18] Daily tongue cleaning controls halitosis and may help to maintain a healthy periodontal environment. A more in-depth discussion on oral malodor (halitosis) can be found in Chapter 37 in the Online Resources section.

1. **Tongue Coating and the Role of Volatile Sulfur Compounds in Halitosis.** The tongue coating is made up of bacteria and other putrefied debris that produces distinct, foul-smelling hydrogen sulfide and methyl mercaptan.
   a. **Volatile sulfur compounds (VSCs)** are a family of gases that are responsible for halitosis.
      1. Two members of the VSC family of gases, hydrogen sulfide and methyl mercaptan, are principally responsible for mouth odor. Methyl mercaptan is produced primarily by periodontal pathogens. Some studies have suggested that low concentrations of these gases may be toxic to tissues. However, the research in this area is limited.[19]
      2. Most patients are concerned about controlling halitosis and therefore are receptive to the introduction of tongue cleaning to their self-care routine.
      3. Tongue coating can contribute to a diminished sense of taste. Daily tongue cleaning should be recommended to geriatric patients who have a low desire to eat due to diminished taste sensation.

   b. Tongue cleaning is recommended because the bulk of bacteria and debris—especially the periodontal pathogens that produce methyl mercaptan—accumulate mostly within the filiform papillae and on the posterior portion of the dorsal surface of the tongue. Routine tongue cleaning may not only make a patient feel more confident, but may help maintain a healthy periodontal environment.

2. **Technique for Use of Manual Tongue Cleaners.** Manual tongue cleaners come in a variety of styles. The two most common types are specialized toothbrushes and tongue scrapers (**Figure 26-20**).

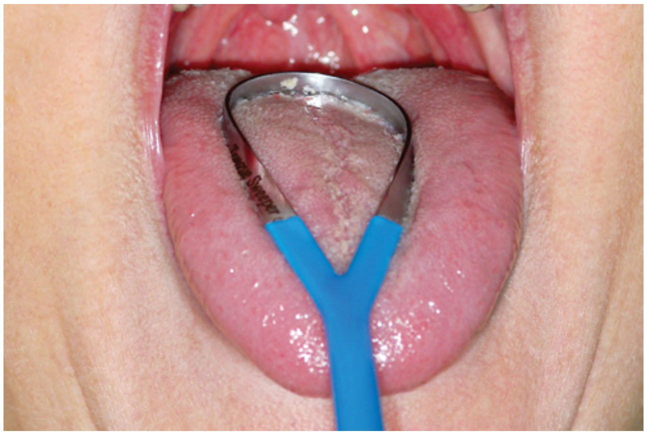

Figure 26-20 **Manual Tongue Scraper.** Daily tongue cleaning controls halitosis and may help maintain a healthy periodontal environment.

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   a. The tongue brush (or scraper) is positioned as far back on the tongue as possible.
   b. Once the tongue brush is in position, it is pulled forward gently over the tongue. This procedure is repeated two or three times or until the tongue is clean.
   c. When first learning how to tongue clean, some individuals gag and find the process unpleasant. In the beginning, encourage the patient to place the cleaner wherever it is most comfortable on the tongue. With regular use, most patients become accustomed to the sensation of the tongue cleaning device and are able to clean further back on the tongue. Over time, most patients become skilled at tongue cleaning.

Chapter Summary Statement

This chapter introduces many of the well-established conventional self-care regimens and new oral health devices available to the patient. Nevertheless, although it is important to be well-versed in all aspects of patient self-care, it is equally essential to understand that there is no one-size-fits-all self-care approach. Rather, the clinician should identify case-specific factors, then work with the patient to map out an appropriate self-care regimen that is customized to the specific needs of the individual. The oral health care practitioner should also fully recognize that although there are studies that favor one type of toothbrush or interproximal modality over others, there is currently no conclusive scientific evidence that clearly substantiates the superiority of one type of self-care regimen over the others. As such, opinions are divided, and a consensus has not been reached. However, one thing is loud and clear: *patient motivation and compliance to a patient-friendly self-care regimen that is easy to do and entails active removal of plaque biofilm on a routine daily basis—rather than the type of self-cleaning device, technique, or method—is the key to success*. Hence, the oral health care provider should be prepared to carefully assess each individual patient's needs and preference, then recommend an appropriate customized self-care modality for that patient.

Section 5

Focus on Patients

Clinical Patient Care

CASE 1

A patient with periodontitis has generalized recession of the interdental gingival papillae. What options would you have for training this patient in interdental plaque biofilm control? What options would you have for training the patient to toothbrush?

CASE 2

You are discussing self-care for biofilm removal with a patient with periodontitis. You point out to the patient how the biofilm control on the facial and lingual surfaces of his teeth is greatly improved and praise him for this success. The patient comments that he likes using his powered toothbrush and has been brushing longer. Unfortunately, you note that there is heavy plaque biofilm on the proximal surfaces of most teeth. The patient tells you that there is "No way that I am going to use that string. It is just too hard to use." The patient has Type II embrasure spaces throughout his mouth. What suggestions might you make for interdental biofilm control?

CASE 3

A patient with periodontitis has generalized bone loss and gingival recession so that the cervical-thirds of the roots are exposed to the oral cavity. What interdental aid would you recommend to clean interproximally (between the roots)?

Evidence in Action

You recently began working as the dental hygienist in an established periodontal practice. The previous hygienist retired after a 20-year career in dental hygiene.

Today, you are seeing for the first time a patient with a history of periodontal surgery, Type III embrasures throughout the dentition, and extensive restorative work. According to the patient record, at the past several maintenance visits there has been very little plaque biofilm on the facial and lingual surfaces of her teeth, but moderate biofilm accumulation on the mesial and distal surfaces of her teeth. Today, your assessment reveals a similar pattern of plaque biofilm formation.

You ask the patient about her current self-care program, and you learn that she has been instructed to use an electric toothbrush and dental floss daily. The patient likes the electric toothbrush, but simply cannot use the floss because it breaks between the "fillings" and that it is just simply too frustrating to use.

Based on what you know about plaque biofilm control with Type III embrasure spaces what suggestions might you offer to this patient as other options for her self-care regimen?

Ethical Dilemma

You have been referred to Dr. Rogers's periodontal practice by your general dentist Dr. Patel. You now alternate your periodontal maintenance appointments between the two offices, with appointments every 3 months.

Six months ago, the hygienist at Dr. Rogers's office, a recent graduate, recommended that you should use an interdental brush, due to the recession and large spaces between your teeth. The hygienist spent much time educating you in the proper technique, and even watched you use it effectively in your own mouth. However, at home, you find it difficult to use, and due to the osteoarthritis in your fingers, find changing the small nylon brushes on the handle almost impossible. As a result, you have not been using the aid.

At your periodontal maintenance visit today, you explain that it has been difficult for you to use the interdental brush due to your arthritis and ask if there is an alternative that would be easier to use. In reply, the hygienist emphatically states interdental brush is the best device for mechanical biofilm control in your situation and seems irritated by your request.

You feel very frustrated and do not think that the hygienist is being sensitive or responsive to your physical limitations and personal needs.

1. How should this ethical dilemma be handled?
2. What ethical principles are in conflict in this dilemma?

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CHAPTER 27

Supragingival and Subgingival Irrigation

- Section 1 Patient-Applied Power-Driven Irrigation Devices
- Section 2 Professional Subgingival Irrigation
- Section 3 Focus on Patients
  - Clinical Patient Care
  - Evidence in Action
  - Ethical Dilemma

CLINICAL APPLICATION

Routine use of a manual interdental cleaning aid (like floss or an interdental brush) as an adjunct to conventional toothbrushing (either a manual or power toothbrush) is typically recommended as part of the self-care regimen. Unfortunately, based on patient surveys, manual interdental cleaning aids are neither popular nor well-accepted by most patients. In fact, a considerable number of patients admit to either infrequently using manual interdental cleaning aids or completely omitting this step as part of their home care regimen. To increase patient acceptance, oral health care companies have developed numerous types of *power*-driven irrigation devices which have been subsequently marketed directly to the consumer. Many of these power-driven irrigation devices range in price and mode of action. Moreover, various power-driven irrigation devices work in different ways (such as solely using water or using both a combination of water and air) and drastically differ in price. Thus, it is important to know whether power-driven irrigation devices are more effective at removing plaque when used in conjunction with a regimen that involves conventional toothbrushing and interdental cleaning aids, and whether their use reduces the gingival inflammation (gingivitis) and prevents or slows the progression of periodontitis. Hence, this chapter addresses the role of patient-applied and professional power-driven irrigation in the preservation of periodontal health. The primary objective of patient-applied home power-driven irrigation is to increase patient acceptance and enhance ease-of-use so that plaque biofilm can be easily removed daily. The goal of professional irrigation in the dental office is to enhance the outcome of periodontal instrumentation and home care by reducing the number of bacteria in the periodontal pocket.

LEARNING OBJECTIVES

- Discuss the oral health benefits of a power-driven irrigation device for the patient with periodontal disease.
- Distinguish the depth of the delivery between the power-driven water irrigation device and a power-driven air floss device, a toothbrush, dental floss, and other interdental aids.
- Name the types of agents that can be used in a power-driven water irrigation device.
- In a clinical setting, be able to educate a patient how to use a power-driven water irrigation device.
- Summarize research findings that relate to using professional irrigation to deliver chemicals to periodontal pockets.

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KEY TERMS

Power-driven irrigation device
Power-driven water irrigation device
Hydrokinetic activity
Impact zone
Flushing zone
Power-driven air floss
Standard irrigation tips
Subgingival irrigation tips
Orthodontic irrigation tips
Filament-type irrigation tips
Professional subgingival irrigation

Section 1

Patient-Applied Power-Driven Irrigation Devices

1. **What Is a Power-Driven Irrigation Device?** A **power-driven irrigation device** cleans teeth by gently delivering:
    a. A pulsating stream of water or other solution (like an antimicrobial solution) around and between teeth above the gingival margin and into the gingival sulcus or periodontal pocket, *OR*:
    b. A stream of compressed air interspersed with microdroplets of water (or an antimicrobial solution) around and between teeth above the gingival margin and into the gingival sulcus or periodontal pocket.
    c. These units are commonly referred to as home or oral irrigation devices.
2. **Types of Power-Driven Irrigation Devices.** Since their launch six decades ago, two types of power-driven irrigation devices have emerged:
    a. **Power-Driven Water Irrigation Device**
        1. **Description.** A **power-driven water irrigation device** (also known as an oral irrigator, water flosser, or power-driven water floss) emits a pulsating stream of water or antimicrobial agent (e.g., Listerine or chlorhexidine) to flush out plaque biofilm and other extraneous debris (like food) from the tooth surface and the gingival sulcus. The Water Flosser developed and manufactured by Waterpik® is classified as a power-driven water irrigation device (**Figure 27-1**).
        2. **Mechanism of Action**
            a. The pulsating fluid delivered by the power-driven water irrigation device uses an alternating compression and decompression phase that efficiently displaces biofilm, bacteria, and debris.[1,2]
            b. The pulsating fluid creates two zones of fluid movement termed **hydrokinetic activity** (**Figure 27-2**).
                1. The area of initial fluid contact to the portion of the tooth near the gingival margin is called the **impact zone**.
                2. The depth of fluid penetration within a subgingival sulcus or pocket is called the **flushing zone**.[3]
            c. Hydrokinetic fluid movement results in movement (or penetration) of the fluid into the subgingival and interdental areas.
            d. The combination of pulsation and pressure produces shear hydraulic forces that have been demonstrated to significantly remove biofilm from treated areas.[4]
        3. **Fluid Penetration**
            a. A power-driven water irrigation device produces subgingival fluid penetration regardless of the type of tip or attachment used.[5,6]
            b. A power-driven water irrigation device has the greatest potential for reaching deeper into a sulcus or pocket compared to other types of conventional oral hygiene aids such as toothbrushes and manual interdental devices.[5,6] **Table 27-1** compares the depth of delivery of common self-care products.

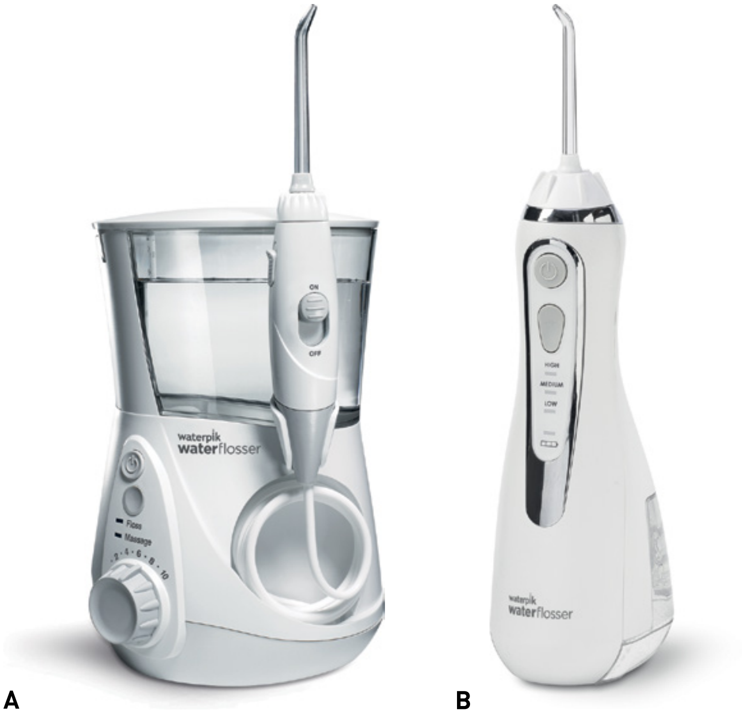

Figure 27-1 Power-Driven Water Irrigation Devices. A. A countertop power-driven water irrigation device that plugs into an electrical outlet (these are corded models that requires electricity from an external source). **B.** A handheld, portable power-driven water irrigation device that works off an internal rechargeable battery (these are cordless models that do not require external power). All power-driven water irrigation devices share four main components: the water reservoir or water tank, pressure control knob or button, various irrigation tips, and a handle. Note that the water reservoir (tank) in a handheld, portable device is built in the back of the handle.

Courtesy of Water Pik, Inc., Fort Collins, CO, USA.

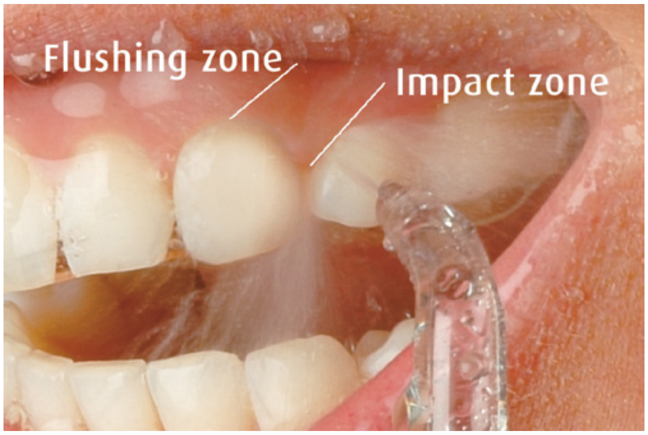

Figure 27-2 The Impact and Flushing Zones. A power-driven water irrigation device creates a pulsating stream of water to flush an area with fluid. The area of the tooth and gingival margin where the fluid initially contacts is called the impact zone. The depth of fluid penetration within the subgingival sulcus or pocket is called the flushing zone.

Courtesy of Water Pik, Inc., Fort Collins, CO, USA.

Table 27-1 Depth of Delivery of Various Self-Care Products

Oral Hygiene Aid	Penetration	Comments
Toothbrush	1–2 mm	No manual or power toothbrush has clinically proven subgingival access
Oral rinsing	2 mm	Can reach less accessible areas, but penetrates subgingival areas minimally.[5]
Toothpick/wooden wedge	Depends on embrasure size	Effectiveness depends on sufficient interdental space
Interdental brush	Depends on embrasure size	Most effective with an open interdental space
Dental floss	3 mm	Cannot reach into deeper pockets
Waterpik water flosser	6 mm and beyond[5,6]	Clinically proven to remove plaque biofilm and bacteria[3,4,15,18,23]

Data from Cobb CM, Rodgers RL, Killoy WJ. Ultrastructural examination of human periodontal pockets following the use of an oral irrigation device in vivo. *J Periodontol*. 1988;59(3):155–163; Gorur A, Lyle DM, Schaudinn C, Costerton JW. Biofilm removal with a dental water jet. *Compend Contin Educ Dent*. 2009;30 Spec No 1:1–6; Braun RE, Ciancio SG. Subgingival delivery by an oral irrigation device. *J Periodontol*. 1992;63(5):469–472. Drisko CL, White CL, Killoy WJ, Mayberry WE. Comparison of dark-field microscopy and a flagella stain for monitoring the effect of a Water Pik on bacterial motility. *J Periodontol*. 1987;58(6):381–386; Goyal CR, Lyle DM, Qaqish JG, Schuller R. Evaluation of the plaque removal efficacy of a water flosser compared to string floss in adults after a single use. *J Clin Dent*. 2013;24(2):37–42; Sharma NC, Lyle DM, Qaqish JG, Galustians J, Schuller R. Effect of a dental water jet with orthodontic tip on plaque and bleeding in adolescent patients with fixed orthodontic appliances. *Am J Orthod Dentofacial Orthop*. 2008;133(4):565–571.

b. **Power-Driven Air Floss (Air Flosser)**

1. **Description.** In 2011, Phillips introduced a new type of power-driven irrigation device branded as Sonicare AirFloss and advertised as an easier way to get “a deeper clean between teeth.” Interestingly, Phillips specifically targeted this product to individuals who are “inconsistent flossers” (**Figure 27-3**).

PHILIPS
sonicare

Figure 27-3 Power-Driven Air Floss Device. From its outward appearance, this power-driven air floss device looks like a conventional power-driven water irrigation device. However, the key difference is that the device emits a burst of compressed air containing microdroplets of water when activated, whereas the water irrigation device releases a focused stream of fluid to dislodge plaque biofilm and food.

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2. **Mechanism of Action.** Unlike a power-driven water irrigation device, the **power-driven air floss** does not release a continuous stream of fluid at low velocity. Rather, the air floss emits a microburst of high-velocity compressed air interspersed with microdroplets of liquid (e.g., water or mouth rinse) to create enough shear stress on the surface to loosen plaque biofilm deposits[7] (**Figure 27-4**).

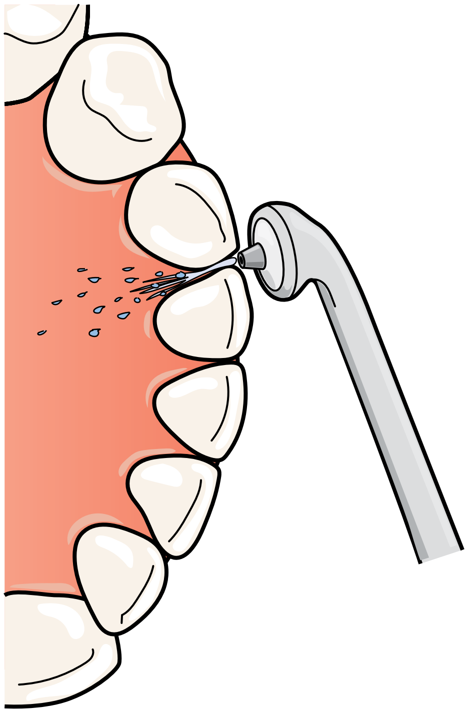

Figure 27-4 Power-Driven Air Floss. The circular nozzle of the power-driven air floss is designed to fit in between teeth and as close to the gingival margin as possible without contacting it. When activated, the device releases bursts of compressed air and microdroplets of water (or mouth rinse) to reach in between teeth and underneath the gumline to physically disrupt and dislodge plaque biofilm and food.

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c. **Head-to-Head Comparison of Power-Driven Water Irrigation Versus Power-Driven Air Floss**
   1. In a randomized controlled study comparing the reduction of the clinical signs of inflammation by two power interdental cleaning devices used in combination with manual toothbrushing, the power-driven water irrigation device was found to be significantly more effective than the power-driven air floss in reducing the clinical signs of inflammation.[8]
   2. In another randomized control clinical trial, the power-driven water irrigation device was found to remove significantly more plaque from all tooth surfaces compared to the air flosser when used in conjunction with manual toothbrushing.[9]
   3. Although existing studies seem to give the edge to power-driven water irrigation devices, many of these clinical trials were sponsored by the company manufacturing the power-driven devices used in the study. As such, the present studies do not allow for an unbiased head-to-head comparison between the two different power-driven irrigation devices. Hence, future appropriately designed, industry-independent trials comparing both types of power-driven devices is warranted to ascertain the clinical efficacy and limitations of each device.

3. **Benefits of Home Power-Driven Irrigation.** Studies have shown that a power-driven irrigation device can remove plaque biofilm and reduce bleeding, gingival inflammation, periodontal pathogens, and inflammatory mediators when paired with a daily toothbrushing regimen.[5–28]
   a. **Removal of Biofilm.** A power-driven irrigation device used in combination with manual toothbrushing has been shown to remove more biofilm than conventional brushing and flossing.[18,27,28]

b. **Reduction in Bleeding.** Studies consistently show that the power-driven irrigation device is a valuable tool for helping patients reduce bleeding.[10–14,16,17,19,20,22,23]
    1. Daily irrigation with a power-driven device significantly reduced bleeding in 14 days.[14]
    2. Daily irrigation with a power-driven device was significantly better than rinsing with 0.12% chlorhexidine at reducing marginal bleeding and bleeding on probing.[17]

c. **Reduction in Gingival Inflammation.** The power-driven irrigation device has been shown to reduce gingival inflammation.[7,8,10–14,16,17,20,22–28]
    1. A power-driven irrigation device used in combination with manual toothbrushing is more effective in the reduction of gingivitis than manual toothbrushing and flossing.[11]
    2. Daily use of a power-driven irrigation device coupled with either an oscillating power toothbrush or a manual toothbrush significantly reduces gingival inflammation.[27,28]

d. **Reduction of Periodontal Pathogens.** Studies show the fluid emitted (from a power-driven water irrigation device) or the combination of compressed air/fluid (from a power-driven air floss device) can reduce subgingival bacteria.[3,12,15,21,24]
    1. The power-driven irrigation device has demonstrated the ability to reduce periodontal pathogens up to a 6-mm level within a periodontal pocket.[3,15] Daily irrigation with either water or 0.04% chlorhexidine significantly reduces subgingival bacteria compared to toothbrushing and 0.12% chlorhexidine rinsing.[11]
    2. Daily irrigation with water for 30 days reduced the bacterial load inside periodontal pockets as effectively as a single administration of the antibiotic minocycline.[24]

e. **Reduction in Inflammatory Mediators and Destructive Host Response.** Recent studies show that home oral irrigation is effective in significantly reducing inflammatory cytokines interleukin-1 beta (IL-1β) and prostaglandin E_2 (PGE_2).[10,14] These cytokines have been implicated in attachment loss and alveolar bone loss.[29,30]

4. **Indications for Recommending Home Oral Irrigation**
    a. **Individuals with Gingivitis on a Prophylaxis Regimen or Successfully Stable Periodontitis Individuals on a Periodontal Maintenance Regimen.** Studies have shown that daily use of a power-irrigation device may be beneficial for patients with gingivitis or for those on a periodontal maintenance regimen.[10–14,16,17,20,23,28] Patients with 5 mm pockets and gingival bleeding who included daily oral irrigation as part of their home care regimen achieved significant reductions in gingival inflammation and bleeding on probing when compared to patients using only conventional self-care methods.[20]

    b. **Individuals Noncompliant with Dental Floss.** While previously considered an adjunctive to brushing and flossing, new information indicates that home power irrigation can be considered an effective alternative to daily flossing for those who cannot floss at a level to achieve a health benefit or who are not interested in using string floss.
        1. A systematic review of 10 oral hygiene aids found that water flossing was superior to string floss for reducing inflammation.[31]
        2. The addition of a power-driven irrigation device once daily with plain water to either a manual or power brushing routine was an effective alternative to dental floss for the reduction of bleeding, gingivitis, and biofilm.[11,19,22,23,28]
        3. The combined use of power-driven irrigation device and a manual toothbrush was 29% more effective at plaque removal than the combined use of a manual toothbrush and floss.[18]

    c. **Individuals with Special Needs.** The power-driven irrigation device may be safe and effective for patients with special needs. Use of a power-driven device may simplify interdental cleaning, especially in areas where access can be challenging and excellent manual dexterity is required.

    d. **Individuals with Dental Implants.** For improving the health of peri-implant tissues, manual toothbrushing paired with daily irrigation using a specialized tip with three soft filaments and water at medium pressure was significantly more effective at reducing bleeding than the combined use of manual brushing and flossing.[19]

    e. **Individuals with Diabetes.** For individuals living with diabetes, twice daily water irrigation using the soft rubber tip provided a 44% better reduction in bleeding over routine oral hygiene.[10]

    f. **Individuals with Orthodontic Appliances.** For those with orthodontic appliances, manual toothbrushing paired with a power-driven irrigation device with the orthodontic tip provided up to three times better biofilm removal and 26% better bleeding reduction than the combined use of a manual toothbrush and flossing using a floss threader.[23]

5. **Patient Instruction**
    a. **Considerations for Water Flosser Use: Product Safety**
        1. Water flossers have been extensively studied on thousands of people for more than six decades. More than 80 studies have been conducted on the water flosser and none have reported adverse effects. A recent review of the literature pertaining to the safety of the water flosser found no detrimental effects on the attachment, junctional epithelium, or pocket depth.[32]
        2. A 6-week study of water flossing with water pressure at the highest setting was found to promote stability of clinical attachment levels and probing pocket depths comparable to string floss and manual brushing or manual brush alone. There were no adverse events to the gingival tissue or epithelial attachment.[33]
        3. The risk of bacteremia from a power-driven water irrigation device is comparable to risks encountered in everyday toothbrushing, flossing, and mastication. Thus, because of the low risk of systemic bacteremia, power-driven water irrigation devices are deemed to be safe.[34]
            a. The American Heart Association's 2021 scientific statement reaffirmed the 2008 guidelines. The authors noted the incidence of bacteremia using a power-driven water irrigation device is similar to the use of other conventional self-care devices, such as wooden toothpicks, floss, toothbrushing, and even everyday routine mastication.[35]
            b. Maintenance of good daily self-care and regular access to professional dental care are considered more important than antibiotic prophylaxis for preventing infective endocarditis.[35]
    b. **Irrigant Solutions.** Most solutions can be used in a water flosser. The most effective agent is one that is acceptable to the patient.
        1. **Water**
            a. Simple tap water has been demonstrated as highly effective in numerous clinical trials.[10,11,13,14,18–20,22–27] Therefore, the addition of any antimicrobial agent for home oral irrigation should be considered carefully.
            b. Water has several advantages: it is readily available, cost-effective, and has no side effects.
        2. **Acceptable Antimicrobial Solutions to Use in a Power-Driven Irrigation Device**
            a. **Chlorhexidine**
                1. For home irrigation, chlorhexidine can be diluted with water. Use of diluted solutions of chlorhexidine has been studied in concentrations from 0.02% to 0.06%.[12,17,21]
                2. Because of better interproximal and subgingival penetration with irrigation compared to rinsing, a dilute solution of chlorhexidine is acceptable for daily irrigation. In some cases, dilution can minimize staining.
                3. Chlorhexidine is available by prescription only. In the United States, the maximum strength is 0.12%. In Europe, it is available at 0.2%.
            b. **Listerine**
                1. For home irrigation, the effectiveness of an essential oil mouth rinse has been demonstrated only when used at full strength.[13] *However, pure full-strength essential oils should be avoided for the reason discussed below.*
                2. Essential oil mouth rinses, such as Listerine, are available over the counter in both brand name and generic forms. *Note that although Listerine is classified as an essential oil, it is not a pure essential oil, because the essential oil content in Listerine (menthol, thymol, methyl salicylate, and eucalyptol) is diluted by other inactive ingredients*. Hence, using Listerine with a power-driven irrigation device is permissible.
                3. Listerine has been clinically tested for safe use in according to Waterpik® and may provide an added benefit of delivering an antimicrobial agent to the difficult-to-reach places like interproximal regions and below the gingival margin.
        3. **Unacceptable Antimicrobial Solutions to Use in a Power-Driven Irrigation Device.** Caution should be exercised when using pure full-strength essential oils and povidone iodine, because these solutions will leave a sticky residue within the power-driven irrigation device, thereby reducing the performance and shortening the life of the product. Further, using full-strength antimicrobial solutions should be avoided as they may lead to some unintended systemic side-effects in certain individuals which will be discussed in Chapter 28.

   a. **Criteria for Equipment Selection.** Selection of a power-driven irrigation device may be confusing because there are many types on the market. The commercial and scientific claims of some devices have yet to be evaluated. Only pulsating power-driven water irrigation devices have clinical research supporting safety and efficacy. Given that each device operates differently in respect to pressure and pulsation, outcomes from studies on one brand of product cannot be transferred to another product brand. Therefore, before recommending any device, it is important to evaluate the pertinent research unique to that brand of product. For the purposes of discussion, only the techniques pertaining to power-driven water irrigation device will be reviewed in this chapter. For a step-by-step description on how to use the power-driven air floss, the reader should refer to the instruction manual that accompanies the device.

6. **Technique for Use of Irrigation Tips**
   a. **General Instructions**
      1. It is important for both dental health care providers and patients to read all instructions thoroughly before using a water flosser.
      2. The fluid reservoir can be filled with water, a combined solution of water and mouthwash, or a solution of an antimicrobial agent and water. The irrigating solution should be at warm temperature for maximum patient comfort.
      3. The unit should be flushed after using any solution other than water. After using a diluted solution, such as diluted chlorhexidine, the unit is cleaned by filling the reservoir with warm water and running the unit while holding the handle in the sink until the reservoir is empty.
      4. Most patients seem to comply with recommendations to use a water flosser and find a standard irrigation tip easy to use for supragingival irrigation.[16]
      5. For subgingival irrigation, it is important to provide patients with clear instructions on its use including the specific areas where the tip should be used.
   b. **Irrigation Tips.** Full mouth water flossing for effective interproximal cleansing can be accomplished using a standard irrigation tip, a soft tapered brush orthodontic tip, or a tip with three fine filaments. **Figure 27-5** shows four different types of tips a patient can select from to best access and clean the surfaces of all teeth.

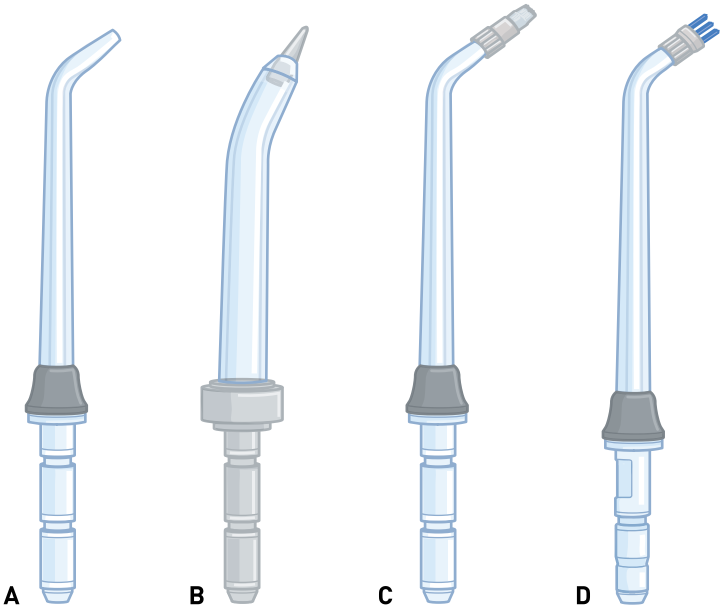

Figure 27-5 Four Types of Irrigation Tip Designs. A. Standard tip. **B.** Orthodontic tip. **C.** Filament-type tip. **D.** Subgingival tip.

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      1. **Standard irrigation tips** are usually made of a plastic material.
         a. This type of tip is recommended for generalized, full-mouth irrigation. It is the most evaluated tip and has been shown to improve oral health better than dental floss.[11,18,22]

    b. Water flosser devices with standard irrigation tips may deliver solution that penetrates a depth of 50% or more of the pocket.[6]

2. **Orthodontic irrigation tips** have a soft tapered brush end that enhances biofilm removal and provides for simultaneous irrigation.
    a. Orthodontic tips can be used for full-mouth irrigation and are recommended for people with orthodontic appliances, implants, or who need additional help with biofilm removal.
    b. The bristles lightly contact the tooth or orthodontic appliances to facilitate biofilm removal.
        1. The orthodontic tip when used in conjunction with toothbrushing was 3.76 times as effective at removing biofilm and 26% better at reducing inflammation than string floss.[23]
        2. When compared to toothbrushing only, the toothbrushing and orthodontic tip combination was 5.83 times as effective.[23]
3. **Filament-type irrigation tips** have three soft filaments surrounding a standard jet tip. Around a dental implant, manual toothbrushing paired with the use of an irrigation device with a filament tip is twice as effective as the combined use of manual brushing and flossing and better at reducing bleeding.[19]
4. **Subgingival irrigation tips** usually have a soft rubber-tipped end.
    a. Subgingival irrigation tips are not cleansing tips. They are to be used to deliver a medicament in areas such as deep pockets, furcation areas, dental implants, or areas that are difficult to access with a standard tip.
    b. The subgingival irrigation tip should be used after full-mouth irrigation with either the standard irrigation tip, orthodontic tip, or filament tip.
    c. Subgingival placement of the tip allows the water or antimicrobial agent to penetrate deeper into a pocket.
        1. In periodontal pockets 6 mm or less in depth, the subgingival tip may deliver water that penetrates up to 90% of the pocket depth.[5]
        2. In deeper pockets—7 mm or more—depth of penetration is somewhat less at 64% of the depth of the pocket.[5]

c. **Procedure for the Use of a Standard Tip**
    1. Initially, the pressure setting should be adjusted to its lowest setting. Over time, as the condition of the gingival tissue improves, pressure should be increased to at least the medium setting, because this setting is where clinical efficacy has been demonstrated.[1,2]
    2. The tip is used to "trace" along the gingival margin with the tip positioned at a 90-degree angle almost touching the gingiva (**Figure 27-6**). The tip should be held briefly at each interproximal area.

d. **Procedure for the Use of an Orthodontic and Filament Tips**
    1. Initially, the pressure setting should be adjusted to its lowest setting. Over time, as the condition of the gingival tissue improves, pressure should be increased to at least the medium setting, because this setting is where clinical efficacy has been demonstrated.[1,2]
    2. The tip is used to "trace" along the gingival margin with the tip positioned at a 90-degree angle touching the gingiva (**Figures 27-7** and **27-8**). The tip should be held briefly in each interproximal area.
    3. This tip can also be placed around orthodontic brackets or wires or implants to enhance cleaning.

e. **Procedure for the Use of a Subgingival Irrigation Tip**
    1. The dental clinician should instruct the patient on the areas of his or her mouth where use of a subgingival irrigation tip would be beneficial, such as pockets, dental implants, or furcation areas.
    2. The pressure setting is adjusted to its lowest setting. However, regardless of the pressure setting of the unit, due to the design of the tip, the exit pressure from the subgingival irrigation tip will always be 20 psi.
    3. The tip should be positioned in the site prior to starting the irrigation unit. The subgingival tip is directed at a 45-degree angle and placed at the gingival margin or slightly beneath the gingival margin as recommended by the manufacturer (**Figure 27-9**).
    4. Once the tip is in place, the irrigation unit is turned on and the fluid is allowed to flow briefly in the area.
    5. After a site has been irrigated, power to the unit is paused and the subgingival tip is repositioned in the next area of the mouth.

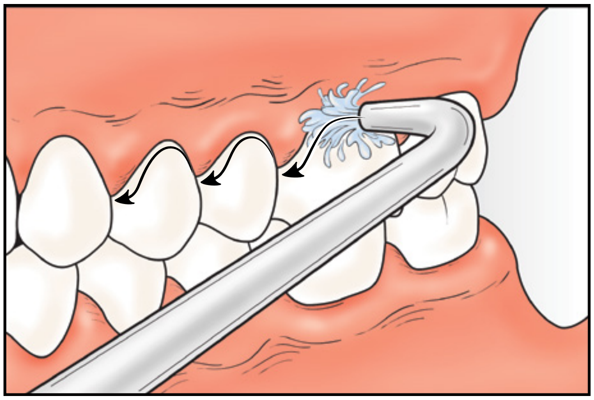

Figure 27-6 Standard Irrigation Tip. The tip is positioned perpendicular to the gingival margin without contacting the gingiva, then traced along the gingival margin. When activated, the device flushes out plaque biofilm and food debris from around the teeth and the gingiva.

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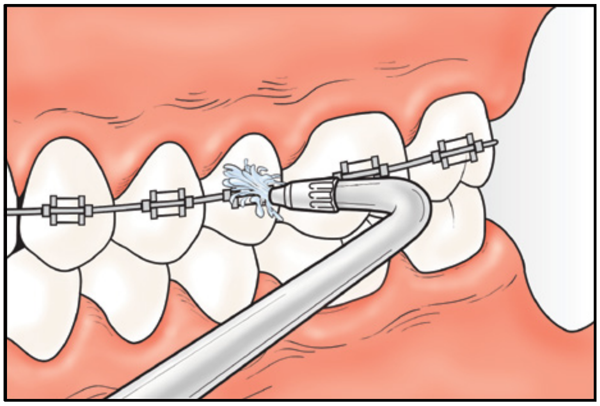

Figure 27-7 Orthodontic Irrigation Tip. The orthodontic tip is specially designed to clean around orthodontic appliances (e.g., an orthodontic wire or brackets). Similar to the use of a standard irrigation tip, the orthodontic tip is positioned perpendicular to the long axis of the tooth without contacting the tooth, orthodontic appliance, or the surrounding gingiva.

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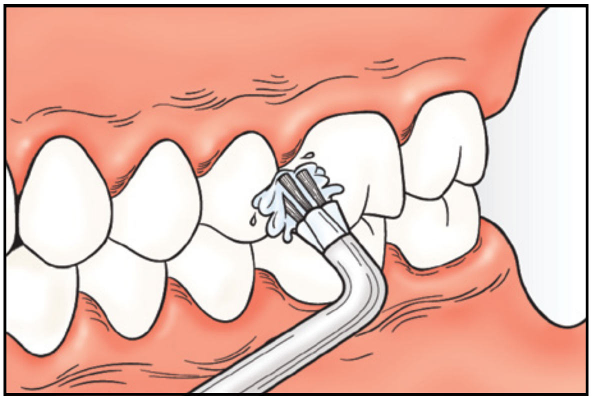

Figure 27-8 Filament-Type Irrigation Tip. At the end of each filament tip are three soft, thin tufts of bristles that gently clean around dental implants, crowns, bridges, and veneers. The filament-type tip is placed close to the teeth (or the implant) with the bristles gently touching the tooth (or the implant). With the device activated, the tip "traces" along the gingival margin.

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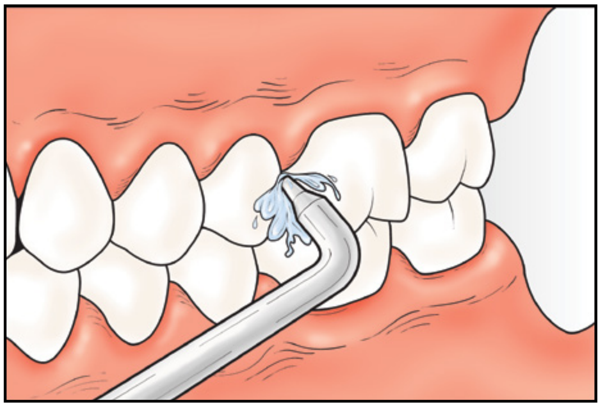

Figure 27-9 Placement of the Subgingival Irrigation Tip. Unlike other tips, this tip is designed to be placed into periodontal pockets. Prior to activating the irrigation device, the tip should be placed at the gingival margin and then directed at a 45-degree angle. The tip is subsequently advanced gently into the periodontal pocket, and the device is then activated.

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Section 2

Professional Subgingival Irrigation

1. **Introduction to Professional Irrigation**
    a. **Description of Subgingival Irrigation.** **Professional subgingival irrigation** is the in-office flushing of pockets performed by the dental hygienist or dentist using one of three systems:
        1. A blunt-tipped irrigating cannula that is attached to a handheld syringe (**Figure 27-10**).

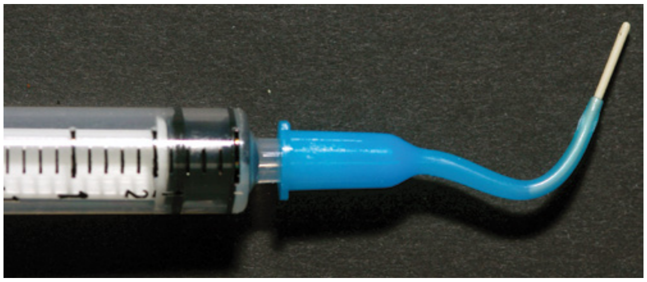

Figure 27-10 **Handheld Syringe.** Close-up view of the tip of a handheld syringe used for subgingival irrigation. The thin, long tip is gently inserted subgingivally for delivery of an antimicrobial solution.

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2. Ultrasonic unit equipped with a reservoir (**Figure 27-11**).

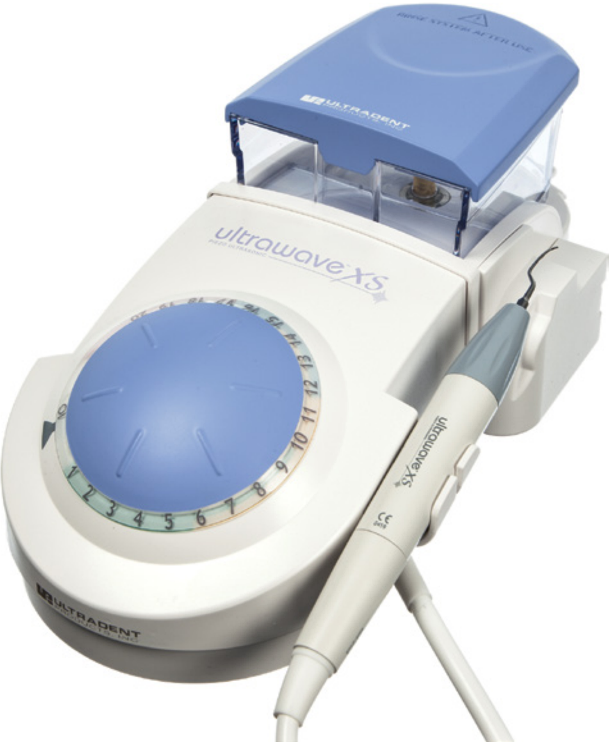

Figure 27-11 **Reservoir for Ultrasonic Unit.** This device has a reservoir system for dispensing irrigant solutions—such as chlorhexidine gluconate—through an ultrasonic tip.

Courtesy of Ultradent Products Inc.

3. A specialized air-driven handpiece that connects to the dental unit airline.

b. **Goal of Subgingival Irrigation.** The purpose of subgingival irrigation is to enhance the outcome of periodontal instrumentation by disrupting and diluting the bacterial biofilm and bacterial products from within the periodontal pocket.

c. **Irrigant Solutions.** Solutions used for subgingival irrigation include chlorhexidine gluconate, povidone-iodine and water, stannous fluoride oral rinse, tetracycline dilutions, Listerine ozonized water.

2. **Effectiveness of Professional Subgingival Irrigation**
   a. **Professional Application**
      1. A systematic review conducted by Hallmon et al. in 2003 found that the use of professional irrigation in addition to nonsurgical periodontal therapy provided no additional benefit to nonsurgical periodontal instrumentation alone.[36] This conclusion was attributed to the following reasons:
         a. There is questionable substantivity of the antimicrobial agent in the periodontal pocket due to the continuous flow of gingival crevicular fluid from the pocket and the presence of serum and proteins in the pocket. A substantive antimicrobial agent, such as chlorhexidine gluconate, would have to be retained in the pocket and be released slowly over a period of time to interfere with the repopulation of bacteria within the pocket. However, the rapid elimination of chlorhexidine gluconate from the periodontal pocket renders its antimicrobial substantivity ineffective.
         b. Depth of the periodontal pocket and presence of calculus may impede the depth of solution penetration.
      2. In their concluding remarks, Hallmon et al. judged that their systematic review should be viewed with caution due to the lack of study heterogeneity. Consequently, subsequent investigations continued to explore the role of professional subgingival irrigation when paired with nonsurgical periodontal instrumentation. To date, emerging evidence leans more favorably to the use of professional subgingival irrigation (when paired with nonsurgical instrumentation), especially in light of the improved clinical and microbiological parameters of periodontal disease following the combined therapy.[36–38]
   b. **Conclusions Regarding Professional Irrigation**
      1. If future studies further substantiate the effectiveness of professional subgingival irrigation, then this combined therapy may offer a safe, simple, and cost-effective means to nonsurgically treat mild (Stage I) and moderate (Stage II) forms of periodontitis. Moreover, this type of treatment modality may be an acceptable alternative to surgery in the medically compromised individuals with periodontitis, in patients with refractory forms of periodontitis, and in patients who have difficulty in maintaining areas such as orthodontic appliances, tooth malalignment, splinted prosthesis, inaccessible embrasure spaces, furcation invasions, and deep periodontal pockets. Nevertheless, further studies that utilize larger sample sizes and longer follow-up periods are needed to further ascertain the clinical and microbiological effectiveness of professional subgingival irrigation.
      2. Subgingival irrigation performed before periodontal instrumentation may reduce the incidence of bacteremia and reduce the number of microorganisms in aerosols.

Chapter Summary Statement

When used in conjunction with toothbrushing, a power-driven irrigation device may boost the effectiveness of the daily self-care regimen. Indeed, existing evidence casts a favorable light on the effectiveness of power-driven irrigation devices in the removal of plaque biofilm and in the reduction in inflammation when used as an adjunct to a commonly accepted self-care regimen. Moreover, power-driven irrigation devices seem to rate higher in patient satisfaction due to its ease-of-use compared to conventional manual interproximal devices. In some cases, patients with systemic conditions, cognitive impairments, or physical disabilities may benefit from using a powered-driven irrigation device over a manual interproximal cleaning aid.

When prescribed with nonsurgical periodontal instrumentation, professional subgingival irrigation therapy has the potential to open up new avenues in the treatment and management of periodontitis.

Section 3

Focus on Patients

Clinical Patient Care

CASE 1

You have recommended a power-driven water irrigation device to a patient. The patient is in periodontal maintenance, has two 5-mm pockets (the mesiobuccal site of the maxillary right first molar and the distolingual site of the mandibular left third molar), one 6-mm pocket (maxillary left first molar, mesial surface), a furcation on the mandibular right first molar, and an implant replacing the mandibular left first molar. What type of self-care instructions would you provide for this patient?

Evidence in Action

A new patient comes to your practice from another state. The patient history indicates that the patient has had type 2 diabetes for 7 years and periodontal therapy in the past. The patient uses a power toothbrush and flosses irregularly. Supragingival plaque biofilm control looks good but several areas of the mouth bleed upon probing. What would you recommend regarding a power-driven irrigation device for this patient?

Ethical Dilemma

Your next patient recently returned from a 3-year stint in the Peace Corps. Prior to the Peace Corps, the patient had received routine and regular dental care, but has not been to a dentist in the last 3 years. You start your periodontal assessment and discover periodontal probe readings have significantly increased from 1 to 3 mm from the last appointment to generalized 4- to 5-mm probe readings. The tissues appear moderately inflamed; there is substantial supragingival biofilm and subgingival calculus but no bone loss.

You recently attended a continuing education course on the use of "water flossers" and feel that this will be an ideal device to help the patient. As you start explaining the device and demonstrating its use, the patient becomes quite agitated and refuses to use it. While working in the Peace Corps, clean water was extremely scarce and considered a luxury. The patient states, "This is a ridiculous waste of water." You are not sure how to proceed.

1. What ethical principles are in conflict in this dilemma?
2. What is the best way for you to handle this ethical dilemma?
3. Do you have an ethical obligation to treat this patient?

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CHAPTER 28

Chemotherapeutics in Periodontal Care

- Section 1 Introduction to Chemical Agents in Biofilm Control
- Section 2 Use of Systemic Antibiotics to Control Biofilm
- Section 3 Use of Locally Delivered Chemical Agents
- Section 4 Topically Delivered Mouth Rinses and Toothpastes
- Section 5 Educating Patients on the Use of Complementary and Alternative Medicine Products/Remedies/Practices
- Section 6 Focus on Patients
  - Clinical Patient Care
  - Ethical Dilemma

CLINICAL APPLICATION

Thorough and meticulous mechanical instrumentation is the cornerstone of successful conventional periodontal therapy. In certain cases, however, a treatment strategy that combines the use of a chemical agent (known as a chemotherapeutic) in combination with conventional mechanical instrumentation is warranted. This chapter reviews common chemotherapeutic agents used to treat and manage periodontal diseases and highlights the role they play in periodontal therapy. This chapter will also examine the alleged claims and the current scientific evidence behind an emerging health care approach known as complementary and alternative medicine (CAM) that has recently garnered headlines in the media.

LEARNING OBJECTIVES

- Define *chemotherapy* and explain its use in the treatment and management of periodontal disease.
- Compare and contrast systemic delivery from topical delivery of chemical agents.
- Define the term *systemic antibiotics* and explain why they are not used routinely in the treatment of patients with plaque-induced gingivitis and patients with periodontitis.
- Describe three examples of mouth rinse ingredients that can help reduce the severity of gingivitis.
- List three antimicrobial agents that can be delivered using controlled-release delivery devices.
- Explain why toothpastes are nearly ideal delivery mechanisms for chemical agents.
- List two toothpaste ingredients that can reduce the severity of gingivitis.
- Explain the current scientific evidence behind charcoal-based dental products and oil pulling.

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KEY TERMS

Chemotherapy
Systemic delivery
Topical delivery
Microbial reservoir
Systemic antibiotics
Antibiotic resistance
Conventional mechanical periodontal therapy
Controlled-release chemotherapeutic agent
Non-controlled-release chemotherapeutic agent
Therapeutic mouth rinses
Substantivity
Complementary and alternative medicine (CAM)

Section 1

Introduction to Chemical Agents in Biofilm Control

Thorough and meticulous mechanical removal of supragingival and subgingival biofilm (along with all other systemic/local contributing factors) via a customized self-care regimen performed by a highly compliant patient *and* routine periodontal instrumentation performed by a fully competent oral health care provider remains the gold standard to treating and managing periodontal disease. In advanced cases, however, solely relying on mechanical instrumentation can be challenging, time consuming, and costly. It may also lead to unpredictable and ineffective outcomes, especially for sites with furcation defects; deep, tortuous periodontal pockets; or altered root surface irregularities. Hence, the adjunctive use of a chemotherapeutic agent (also known as a chemotherapeutic) combined with mechanical therapy may be indicated to reduce or eliminate pathogenic microorganisms that cannot be accessed by mechanical means alone. In this context, the term **chemotherapy** is a broad term that describes any type of therapy that utilizes a chemical agent to treat or control a disease. The term *chemotherapeutic* refers to the actual chemical agent used to treat and manage the disease. (In the field of oncology, a chemotherapeutic is any type of anticancer treatment that uses chemical agents to inhibit cancer cells from multiplying; likewise, in the context of periodontal disease, a chemotherapeutic is any chemical agent used to treat and manage periodontal disease by inhibiting and eliminating the microorganisms in the oral cavity.) The term *adjunctive* refers to supplementary therapies done concurrently with conventional periodontal therapy. Adjunctive chemotherapeutics include systemic antibiotics, locally delivered antimicrobials, or topically applied full-mouth mouth rinses, dentifrices, or gels.

1. **Delivery of Chemotherapeutics in Periodontal Patients.** When used in combination with conventional periodontal therapy, chemotherapeutics can be easily administered via systemic delivery or topical delivery.
    a. **Systemic Delivery**
        1. In dentistry, **systemic delivery** usually refers to administering chemotherapeutics in the form of a tablet or capsule. After a patient swallows a tablet (or capsule), the chemotherapeutic agent is released as the tablet (or capsule) dissolves in the stomach and intestines, and the agent subsequently enters the bloodstream. Thus, the chemotherapeutic is circulated "systemically" throughout the body.
        2. As the chemotherapeutic circulates throughout the body in the bloodstream, it enters the periodontal tissues and becomes part of the gingival crevicular fluid where it encounters the periodontal microorganisms invading the periodontal tissues.
        3. An example of systemic chemotherapeutic delivery would be a patient who takes the antibiotic penicillin in tablet or capsule form. The penicillin passes through the wall of the gastrointestinal tract and enters the body's tissues. In medicine, systemic delivery of chemical agents can also be administered via injection into a muscle (known as intramuscular delivery) or into a blood vessel (known as intravenous delivery); although in dentistry, these two types of delivery are not part of routine dental practice to control periodontal disease.
    b. **Topical Delivery**
        1. In dentistry, **topical delivery** (also referred to as local delivery) usually refers to the intraoral placement of a chemotherapeutic directly into a periodontal pocket where the chemical agent then contacts the biofilm covering the teeth or in the periodontal pocket.

2. Examples of topical delivery in dentistry would be using a mouth rinse or toothpaste that contains a chemical agent, which neutralizes bacteria growing in dental biofilm. In this instance, the chemical agent comes into direct contact with the teeth, oral mucous membranes, and dental biofilm.
3. When a topical chemical agent contacts oral mucous membranes, some of the agent crosses through the mucous membranes to enter the bloodstream. However, the bulk of the agent remains in the oral cavity. **Table 28-1** provides an overview of topical and systemic delivery mechanisms for chemotherapeutics used in dental biofilm control.

Table 28-1 Delivery Mechanisms for Various Types of Chemotherapeutics

Delivery Type	Specific Mechanism	Possible Patient Benefit(s)
Topical	Therapeutic mouth rinses	Reduction in the severity of gingival inflammation
Topical	Therapeutic dentifrices	Reduction in dentinal hypersensitivity Reduction in gingival inflammation Reduction of supragingival calculus Reduction in surface stains
Topical	Subgingival irrigation	Disruption and dilution of bacteria within the dental biofilm
Topical	Controlled-release delivery devices	Subject subgingival bacteria to therapeutic levels of a drug for a period of a week or longer
Systemic	Tablets, capsules	Help control more aggressive forms of periodontitis Fight acute oral infections

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2. **Considerations for Use of Chemotherapeutics in Periodontal Patients**
   a. **Resistance of the Biofilm to the Delivery of Chemotherapeutics.** The surface of dental plaque biofilm is covered by a layer known as the extracellular protective matrix. The extracellular protective matrix acts as a barrier preventing some chemical agents from contacting the bacteria in the plaque biofilm.
   b. **Microbial Reservoirs for Periodontal Pathogens.** The oral cavity contains a variety of microbial reservoirs that can lead to rapid repopulation of bacterial pathogens in a treated periodontal patient.
      1. A **microbial reservoir** is a niche or secure place in the oral cavity that allows live periodontal pathogens to remain undisturbed during routine therapy and subsequently repopulate periodontal pockets quickly.
      2. An example of a microbial reservoir would be a residual calculus deposit following periodontal instrumentation. Living bacteria found within the calculus deposit can reproduce in periodontal pockets and continue to promote disease even after what appears to be thorough nonsurgical therapy.
      3. **Box 28-1** provides an overview of some of the many microbial reservoirs for periodontal pathogens in the oral cavity.

Box 28-1 Microbial Reservoirs for Periodontal Pathogens

- Bacterial plaque biofilm in protected sites such as furcation areas
- Bacteria in residual calculus deposits that are not removed during nonsurgical therapy
- Bacteria living within the epithelial layers and connective tissue adjacent to a periodontal pocket
- Bacteria that have penetrated dentinal tubules
- Bacteria protected by irregularities in tooth surfaces after mechanical treatment
- Bacteria protected by poorly defined restoration margins

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3. **Criteria for Effective Chemotherapeutics.** For chemotherapy to be effective, it must meet three requirements: (1) reach the sites of disease activity, namely the base of the pocket; (2) be delivered at a bacteriostatic or bactericidal concentration; and (3) remain in place long enough to be effective.[1] See **Table 28-2** for a comparison of the effectiveness of common drug delivery systems for management of periodontitis.

Table 28-2 Comparison of Drug Delivery Systems for Management of Periodontitis

	Adequate Drug Concentration	Reaches Sites of Disease Activity	Adequate Time in Place to Be Effective
Mouth rinsing	Good	Poor	Poor
Subgingival irrigation	Good	Good	Poor
Systemic delivery	Fair	Good	Fair
Controlled-release delivery	Good	Good	Good

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4. **Chemical Agents Effective Against Periodontitis.** Periodontal instrumentation is an effective mechanical therapy for periodontitis.[2,3] In deep or tortuous pockets or sites that do not respond to nonsurgical periodontal therapy, however, it may be beneficial to use adjunctive chemical agents.[4,5] Nevertheless, while there is no chemotherapeutic that can halt periodontitis single-handedly, there are a number of different chemotherapeutics that can be used as part of comprehensive treatment for patients with periodontal diseases. An overview of chemical agents that have been suggested for use in biofilm control in periodontal patients is presented in **Table 28-3**.

Table 28-3 Overview of Some of the Chemical Agents Used in Biofilm Control

Type of Agent	Example of Agent	Means of Administration
Antibiotics	Tetracyclines	Tablet/capsule Subgingival delivery
Bisbiguanide antiseptics	Chlorhexidine	Mouth rinse Subgingival delivery
Fluorides	Stannous fluoride	Mouth rinse Toothpaste Gel
Metal salts	Tin/zinc	Mouth rinse Toothpaste
Oxygenating agents	Hydrogen peroxide Sodium hypochlorite	Mouth rinse Subgingival delivery
Iodophors	Povidone-iodine	Mouth rinse Subgingival delivery
Phenolic compounds	Essential oils	Mouth rinse
Quaternary ammonium	Cetylpyridinium chloride	Mouth rinse
Tertiary amine surfactant	Delmopinol	Mouth rinse

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Section 2

Use of Systemic Antibiotics to Control Biofilm

1. **Overview of Systemic Antibiotics**
    a. **Definitions**
        1. Antibiotics are antimicrobial agents used to help combat infections either because they kill bacteria (termed as *bactericidal*) or because they can inhibit the growth and multiplication of bacteria (termed as *bacteriostatic*).
        2. **Systemic antibiotics** are those antibiotics that can be taken orally or that can be injected. Worldwide, antibiotics are used to fight bacterial infections. In North America, health care providers, such as physicians and dentists, have access to a broad range of antibiotics for use in patients with infections. These antimicrobial agents have been used for many years to help the body fight certain bacterial infections and have undoubtedly been responsible for saving countless lives.
    b. **Systemic Antibiotics Studied for Use in Periodontal Diseases.** Systemic antibiotics have also been studied for their use in controlling periodontal diseases when used as an adjunct to mechanical instrumentation. **Box 28-2** lists examples of systemic antibiotics that have been studied by researchers for use in periodontal patients. Note that there is no one type of antibiotic that cures every periodontal infection. This is because of the diverse array of different types of microorganisms that inhabit the dental biofilm. Further, the overuse and overreliance of systemic antibiotics to control basic infections undermines our own efforts to combat antibiotic-resistant infections, such as methicillin-resistant *Staphylococcus aureus* (MRSA) infections, which can be fatal.

Box 28-2 Examples of Systemic Antibiotics Studied for Use in Periodontal Care

- Penicillin and amoxicillin
- Tetracyclines
- Azithromycin
- Erythromycin
- Metronidazole
- Clindamycin

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    c. **Plaque-Induced Gingivitis and Periodontitis**
        1. For most patients with the more common forms of periodontal disease, periodontal infections can be successfully managed by performing thorough and meticulous mechanical instrumentation and by a carefully supervised patient self-care regimen, rather than relying on the adjunctive use of systemic antibiotic drugs to control these diseases. There are two major reasons to avoid their routine use in these patients.
            a. The misuse or overuse of systemic antibiotics can lead to antibiotic resistance.[6–8] **Antibiotic resistance** refers to the ability of a bacterium to withstand the effects of an antibiotic by developing mechanisms that protects itself from the killing or inhibiting effects of the antibiotic. Most species of subgingival bacteria are considerably more resistant in biofilms than in planktonic cultures. Resistance appears to be age-related because biofilms demonstrated progressive antibiotic resistance as they matured with maximum resistance coinciding with the steady-state phase of biofilm growth.[9]
                1. Antibiotic resistance is a serious public health problem throughout the world, and the problem is increasing in scope. When antibiotic-resistant strains of bacteria develop, they are generally not affected by the antibiotic and continue to cause more damage.
                2. An example of how antibiotic resistance can be a public health problem is the high incidence of penicillin-resistant microorganisms that have already limited the usefulness of this important drug. As more and more bacteria develop resistance to the penicillin, the usefulness of this antibiotic as a life-saving drug will decline.

b. Another reason dentists avoid routinely using systemic antibiotics is because studies indicate that, in most patients, these diseases respond to conventional mechanical periodontal therapy just as well as they respond to the systemic administration of antibiotics. **Conventional mechanical periodontal therapy** refers to self-care, periodontal instrumentation, surgical interventions, and control of local contributing factors.

2. When antibiotics are being considered for use in periodontal patients, careful patient selection is necessary.
    a. When tempted to use systemic antibiotics to control either plaque-induced gingivitis or periodontitis, dental health care providers must weigh potential benefits and risks. At this point, most clinicians have decided that the potential risks outweigh the benefits.
    b. In patients with periodontitis, the efficacy of adjunctive antibiotic therapy is not completely clear, and antibiotic therapy in these patients should usually be limited to those patients who have continued periodontal breakdown after thorough conventional mechanical periodontal therapy.
    c. Note that even though systemic antibiotics are rarely indicated for routine treatment of patients with plaque-induced gingivitis or periodontitis, they are frequently indicated in the treatment of patients with more aggressive forms of periodontitis.
3. **Patient Education.** Even though systemic antibiotics are not normally used for patients with plaque-induced gingivitis or periodontitis, systemic antibiotics are discussed here because periodontal patients may ask why antibiotics are not being recommended for them.
    a. This question may arise when the patient learns that periodontitis is a bacterial infection. This is a natural question for a patient to ask given the widespread use of antibiotics in fighting infections of all sorts.
    b. When confronted with a question from a patient about why antibiotics are not being recommended, the dental hygienist can explain the following facts:
        1. Most cases of gingivitis and periodontitis can be readily controlled with conventional mechanical treatments that do not require the use of systemic antibiotics.
        2. The misuse and overuse of systemic antibiotics has greatly contributed to the development of antibiotic-resistant strains of bacteria. In fact, many antibiotics have been rendered ineffective for life-saving measures through the development of antibiotic-resistant strains of bacteria.
        3. Most antibiotics have accompanying adverse effects such as gastrointestinal discomfort, nausea, oral ulcers, and diarrhea. In some cases, an antibiotic can trigger a life-threatening anaphylactic reaction in individuals who are allergic to the prescribed medication (such as a hypersensitivity reaction to amoxicillin in penicillin-allergic individuals). If not managed immediately, this can be fatal.

d. **More Aggressive Forms of Periodontitis**

1. The use of antibiotics in conjunction with mechanical therapy was the recommended treatment regimen for what was previously known as aggressive periodontitis.[10]
2. However, because the 2017 AAP/EFP Disease Classification scheme eliminated aggressive periodontitis as a distinct periodontal disease entity, knowing when to combine systemic antibiotics with the standard conventional treatment regimen is now unclear. Thus, the dental clinician must rely on his or her own experience, judgment, and treatment philosophy when assessing each individual periodontitis case to determine if systemic antibiotics is warranted as part of the individualized periodontal therapy.
3. When antibiotics are being considered for use, it is prudent to perform a microbiologic analysis. Microbiologic analysis involves sampling the bacteria associated with the disease process and testing cultures of the bacteria for specific antibiotic susceptibility. Utilizing microbiologic analysis can avoid prescribing inappropriate antibiotics that yield a poor outcome.
4. The flowchart in **Figure 28-1** can assist the clinician in evaluating which type of periodontitis case would benefit from systemic antibiotics. However, before reviewing the diagram, note that the recommendations presented in Figure 28-1 are neither mandatory rules nor part of the standards of care. Rather, this figure is designed to illustrate the thought processes that should be in a clinician's mind when evaluating whether a specific periodontitis case warrants systemic antibiotics as an adjunct to conventional therapy. Ultimately, it is left to the clinician to determine if systemic antibiotics are warranted on a case-by-case basis.

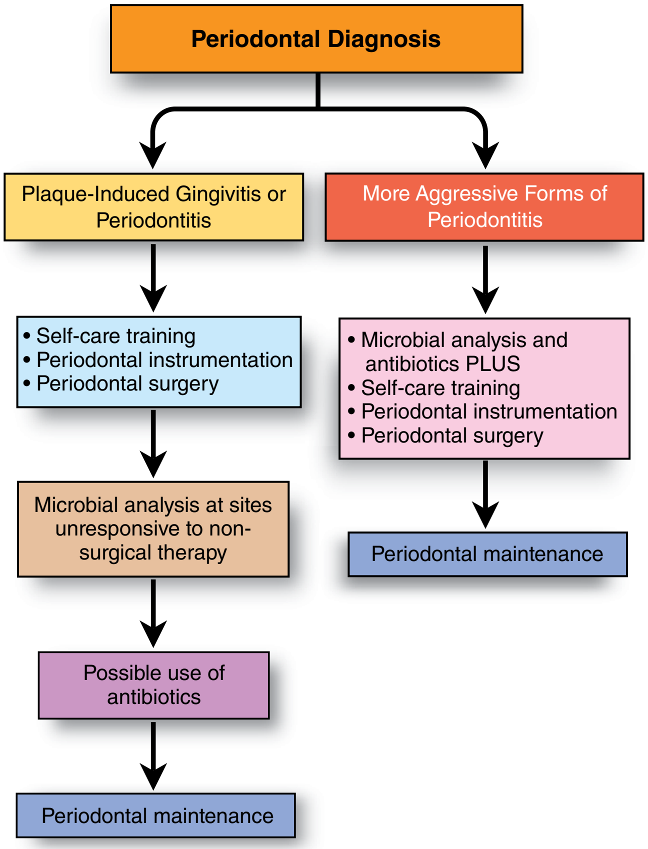
Periodontal Diagnosis
Plaque-Induced Gingivitis or Periodontitis
More Aggressive Forms of Periodontitis
• Self-care training
• Periodontal instrumentation
• Periodontal surgery
• Microbial analysis and antibiotics PLUS
• Self-care training
• Periodontal instrumentation
• Periodontal surgery
Microbial analysis at sites unresponsive to non-surgical therapy
Periodontal maintenance
Possible use of antibiotics
Periodontal maintenance

Figure 28-1 The Relationship Between Periodontal Diagnosis and the Use of Systemic Antibiotics.

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2. **Use of Tetracyclines in Periodontal Patients.** Tetracyclines are a group of antibiotics that have received special attention by researchers because they have certain properties that make them attractive for use in selected periodontal patients (**Figure 28-2**).

Figure 28-2 Systemic Delivery of a Chemotherapeutic. A patient prepares to take an oral antibiotic as an adjunct to conventional periodontal therapy.

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   a. Tetracyclines are broad-spectrum bacteriostatic antibiotics. The term *broad spectrum* means that an antibiotic acts against both Gram-positive and Gram-negative bacteria, as opposed to *narrow spectrum*, which refers to an antibiotic that specifically targets a select group of microorganisms.
   b. Tetracyclines tend to be concentrated in the gingival crevicular fluids. When tetracyclines are administered orally, the drugs permeate body tissues and reach a certain concentration in the blood serum. The level of tetracycline drugs, however, is more concentrated within the gingival crevicular fluids flowing into periodontal pockets than the level found in the blood serum. This results in a higher concentration of the drug in exactly the sites it might be needed in a periodontal patient.
   c. The tetracyclines are effective against most strains of *Aggregatibacter actinomycetemcomitans*. *A. actinomycetemcomitans* is one of the periodontal pathogens associated with the more aggressive forms of periodontitis (formerly known as localized aggressive periodontitis or localized juvenile periodontitis).
   d. The tetracyclines have other effects in addition to their antimicrobial properties.
      1. Tetracyclines inhibit the action of collagenase, which is one of the enzymes responsible, in part, for the breakdown of the periodontium.
      2. Any drug that inhibits the action of collagenase can be expected to slow the progress of the tissue breakdown caused by periodontitis.
   e. Certain tetracyclines are effective in subantimicrobial doses.
      1. Subantimicrobial dose doxycycline (20 mg twice a day) significantly improved clinical parameters associated with periodontal health in patients with periodontitis when used as an adjunct to a maintenance schedule of periodontal instrumentation.[11–13] Subantimicrobial dose doxycycline will be further discussed in Chapter 29.
      2. Several studies assessed whether a long-term subantimicrobial dose doxycycline produces doxycycline-resistant oral microflora in adults with periodontitis. These studies found no evidence of antibiotic resistance in the subantimicrobial dose doxycycline treatment groups.[12,13]
   f. **Overuse.** Sadly, the over-reliance of tetracyclines in medicine and dentistry has led to an emergence of tetracycline-resistant microorganisms, thereby limiting the effectiveness of this antibiotic.

3. **Use of a Combination of Amoxicillin + Metronidazole**
   a. Both amoxicillin and metronidazole are bactericidal agents. But whereas amoxicillin is classified as a broad-spectrum antibiotic, metronidazole is a narrow-spectrum antibiotic that is specifically active against anaerobic bacteria.
   b. The combined benefits of using amoxicillin and metronidazole makes it the most frequent antibiotic regimen prescribed to patients and one of the more effective antibiotic regimens to prescribe to patients with periodontitis with respect to greater reduction in periodontal pathogens levels compared to nonsurgical scaling and root planing alone.[14] This is because of the wide array of aerobic and anaerobic microorganisms residing within the dental biofilm. This same conclusion was validated in a recent systematic review that evaluated the effectiveness of three different antibiotic regimens used in contemporary periodontics (amoxicillin + metronidazole group vs. metronidazole alone group vs. azithromycin alone group); the best clinical outcomes were observed in patients receiving a combination of amoxicillin + metronidazole.[15]
   c. However, at the present time, the lack of a standard prescription protocol for this combined approach makes its effectiveness questionable. To highlight: within the literature, the prescription protocol greatly varies among studies with respect to different dosages, frequency, and time (number of days the patient should be taking the antibiotics).
   d. The oral health care provider must therefore weigh the potential risks and benefits of using this combined antibiotic regimen, especially with the concern of provoking a hypersensitivity reaction or risking the development of antibiotic resistance.

4. **Use of Azithromycin**
   a. Azithromycin (brand name Zithromax®, also known as a “Z-pak”) is a broad-spectrum bacteriostatic agent that emerged in the medical field in the 1990s to combat a wide range of infections, including pneumonia, genitourinary infections (e.g., chlamydia), enteric (intestinal) infections (e.g., typhoid), and malaria.

b. Upon administration of a single 500-mg oral dose, azithromycin offers several benefits that make it an attractive systemic antibiotic: rapid absorption, high tissue concentration, long half-life (68 hours), and a more patient-friendly dosing schedule (once-a-day dosing regimen, typically one 500-mg tab for 7 days).[16] This more patient-friendly dosing schedule makes azithromycin favorable to patients because it adds to patient convenience, which translates to better patient compliance compared to the prescription protocols for other antibiotics that may need to be taken several times a day.

c. The periodontal benefits to using azithromycin in combination with mechanical instrumentation, however, remains controversial because there are conflicting studies that show clinical benefits of azithromycin versus others that fail to validate these benefits.
   1. In a direct comparison of scaling and root planing alone (the control group) versus scaling and root planing + azithromycin (the azithromycin group) versus scaling and root planing + metronidazole + amoxicillin (the metronidazole + amoxicillin group), Sampaio and colleagues found that azithromycin alone did not yield the same benefits as metronidazole + amoxicillin in the treatment of advanced periodontitis with respect to the clinical results (specifically, reduction in probing depths and gain in clinical attachment levels) and microbiological results (specifically a profound beneficial change in the subgingival microbial profile) after 1 year.[17] Moreover, the adjunctive use of azithromycin did not offer statistically significant advantages in comparison with scaling and root planing alone.
   2. A 2017 systematic review, however, demonstrated that in the majority of studies adjunctive use of azithromycin with nonsurgical periodontal therapy resulted in clinical improvements, with the greatest benefit being seen in deeper initial pockets (greater than equal to 6 mm).[18]
   3. Overall, adjunctive azithromycin holds a lot of potential. However, additional studies need to be performed to validate and further justify its use as adjunctive periodontal therapy.

Section 3

Use of Locally Delivered Chemotherapeutic Agents

Local delivery of a chemotherapeutic agent has many potential advantages and disadvantages.

1. **Advantages**
   a. Local delivery makes it possible to directly deposit high and sustained levels of the chemotherapeutic agent at the site of infection. Systemic antibiotics, in contrast, are distributed throughout the body before arriving at the target site, thereby limiting the actual dose of the antibiotic reaching the intended target.
   b. Local delivery has limited potential for systemic absorption and untoward/unintended systemic side effects associated with systemic antibiotics (e.g., disruption in the normal gastrointestinal microflora).
   c. Local delivery reduces the volume of antibiotic needed to achieve the intended result.
   d. Local delivery decreases the risk of antibiotic resistance because the antibiotic is delivered directly to the targeted site.
   e. Local delivery has a minimal effect on the bacterial flora that normally inhabits other areas of the body.
2. **Disadvantages.** Local chemotherapeutic agents can be delivered in varying forms, such as injections, gels, or impregnated beads of synthetic fibers placed directly in the sulcus. Further, many different types of antibiotics—ranging from chlorhexidine to tetracyclines to minocycline—have been reported to be effective in countering biofilm growth and maturation. However, it has not been established which type of antibiotic to use, as well as how much, for how long, and in what form. Comparisons of studies of local antibiotics in certain surgical settings are difficult to perform given the variation in agent, dose, and formulation. For this and other reasons, local antibiotics, although promising, still require more scientific validation.
3. **Types of Locally Delivered Chemotherapeutic Agents.** Locally delivered chemotherapeutic agents can be categorized into three broad categories: (1) controlled-release chemotherapeutic agents (e.g., gels and chips); (2) non-controlled-release chemotherapeutic agents (e.g., sodium hypochlorite and povidone-iodine); and (3) topically administered full-mouth rinses and toothpastes. This last category will be covered in the next section because it is such a broad topic.

a. **Overview of Controlled-Release Chemotherapeutic Agents**
   1. A **controlled-release chemotherapeutic agent** consists of an antibacterial mediator that is embedded within a carrier material. They are designed to be placed directly into the periodontal pocket where the carrier material attaches to the tooth surface and dissolves slowly, producing a steady, sustained release of the antimicrobial agent over a period of several days within the periodontal pocket.[19]
   2. The earliest versions of these products involved coating carrier fibers with chemical agents that would be released following placement of the fibers within periodontal pockets. Use of these nondissolvable fibers required the patient to return to the dental office for removal of the fibers.
   3. The latest versions of these products involve embedding biodegradable antimicrobial agents into carrier materials that dissolve slowly over approximately 1 week. The carrier material can be placed into a periodontal pocket, and as it dissolves, it slowly releases the antimicrobial agent. Unlike early examples of controlled-release devices, the newer types are dissolvable and do not require the patient to return for removal.
   4. Antimicrobial agents currently used in controlled-release chemotherapy include chlorhexidine and some of the antibiotic drugs such as the tetracyclines. In the future, other drugs may be used for this purpose.
   5. **Rationale for Use.** The goal is to subject subgingival bacteria to therapeutic levels of an antibacterial drug over a sustained period. Most of these controlled-release devices continue to deliver chemicals into the pockets for approximately 1 week.
   6. **Benefits of Controlled-Release Chemotherapeutic Agents**
      a. Use of controlled-release chemotherapeutic agents has been shown to result in a small increase in attachment level in a periodontal pocket (about a 0.5- to 2-mm reduction found in probing depths compared to conventional periodontal instrumentation).
      b. Controlled studies are available to guide dental health care providers in the appropriate use of these new devices.
      c. Controlled-release agents may be indicated for use in localized periodontal pockets that are nonresponsive after thorough nonsurgical and surgical periodontal therapy or sites with disease recurrences. In addition, controlled-release delivery may be beneficial for patients who are not good candidates for surgical therapies, such as patients who are medically compromised, smokers, and those with inadequate plaque control.
      d. When used along with periodontal instrumentation, controlled-release chemotherapeutic agents can result in both an improvement in probing depth reduction and a clinical attachment gain. The clinical significance of the small amount of improvement is unclear at this point. Routine use of these agents, as an adjunct to periodontal instrumentation and periodontal maintenance, may show improved results in future phase III clinical studies.
      e. Current guidelines support the use of controlled-release devices in combination with periodontal instrumentation.[3–5,20–23]
   7. **Controlled-Release Agents.** Several controlled-release chemotherapeutic agents have been introduced in the United States over the last few years, and it is likely that more will be available within the next few years. Some of the chemical agents that have been incorporated into these devices and marketed over the past few years are outlined next.
      a. **Minocycline Hydrochloride Microspheres**
         1. Marketed under the brand name Arestin.
         2. Arestin has a controlled-release mechanism that delivers the antibiotic minocycline hydrochloride in a powdered biodegradable microsphere form. Minocycline hydrochloride is a broad-spectrum, semisynthetic tetracycline derivative that is bacteriostatic.
         3. **Application**
            a. A cannula tip is used to expel the microspheres into the pocket (**Figure 28-3**) where it binds to the tooth surface because of the sticky nature of the carrier material.

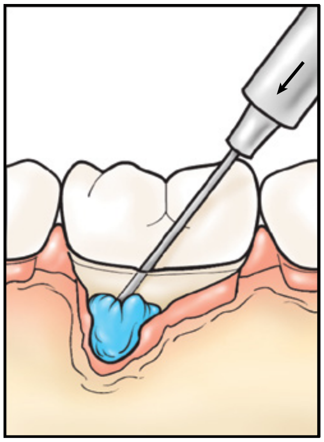

Figure 28-3 Local Chemotherapeutics Delivered Directly into a Periodontal Pocket. Some local chemotherapeutics are delivered directly into the periodontal pocket with a cannula tip. Microspheres containing minocycline hydrochloride (Arestin) and gels containing doxycycline (Atridox) are examples of such products. Upon contact with the tooth, the chemotherapeutics readily adhere to the tooth surfaces and dissolve slowly over time, thereby releasing the antimicrobial agents incorporated within the carrier.

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    b. Over 5 to 7 days, the powdered microspheres dissolve releasing the embedded minocycline so there is nothing to remove from the pocket.
4. Studies demonstrate that repeated subgingival administration of minocycline microspheres in the treatment of adult periodontitis is safe and is more effective than periodontal instrumentation alone in reducing probing depths in periodontitis patients.[24,25]
5. **Adverse Reactions.** Possible adverse reactions include oral candidiasis or an allergic response. In addition, the use of antibiotic preparations may result in the development of resistant bacteria.
6. **Contraindications for Use.** This product is a tetracycline derivative. Thus, it should be avoided in patients who are hypersensitive to tetracycline or in women who are pregnant or nursing.

b. **Doxycycline Hyclate Gel**
    1. Marketed under the brand name Atridox.
    2. This product is a gel system that delivers the antibiotic doxycycline (also a tetracycline derivative) to the periodontal pocket.
    3. **Application**
        a. The gel is expressed into the pocket with a cannula (Figure 28-3). After placement, the gel solidifies into a wax-like substance.
        b. The cannula tip is placed near the pocket base and gel is expressed using a steady pressure until the gel reaches the top of the gingival margin.
        c. A limitation to this delivery system is that the gel tends to cling to the cannula when withdrawn from the pocket, which can be reduced by using a moistened dental hand instrument to hold the gel in place while slowly withdrawing the cannula tip from the pocket.
        d. The gel is biodegradable (it dissolves) so there is nothing to remove from the pocket.
    4. In a 6-month multicenter trial, results indicate that periodontal instrumentation combined with local application of doxycycline in deep periodontal sites can be considered as a justified approach for nonsurgical treatment of periodontitis.[26]

5. **Adverse Reactions**
    a. Possible adverse reactions include oral candidiasis or an allergic response.
    b. The use of antibiotic preparations may result in the development of resistant bacteria.
6. **Contraindications for Use.** This product is a tetracycline derivative; thus, it should not be used in patients who are hypersensitive to any tetracycline or in women who are pregnant or nursing.

c. **Chlorhexidine Gluconate Chip**
1. Marketed under the brand name PerioChip.
2. Another example of a controlled-release device is a tiny biodegradable gelatin chip containing the antiseptic chlorhexidine that is inserted into a periodontal pocket that is 5 mm or greater in depth (**Figure 28-4**).

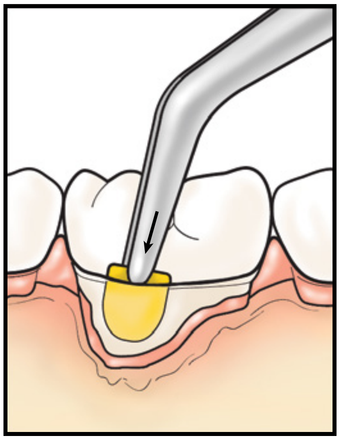

Figure 28-4 Local Chemotherapeutic Gelatin Chip Inserted into a Periodontal Pocket. The gelatin chip is gently inserted into periodontal pockets 5 mm or greater in depth. The gelatin chip adheres to the tooth surface and dissolves slowly, releasing the chlorhexidine antimicrobial agent embedded within the gelatin carrier. An example of this type of local chemotherapeutic is PerioChip.

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3. Investigations indicate that the chlorhexidine gluconate chip, when used as an adjunct to periodontal instrumentation, significantly reduces loss of alveolar bone.[27]
4. **Application**
    a. The gelatin chip is inserted into the periodontal pocket.
    b. The gelatin chip can be difficult to insert into some pockets due to the size and shape of the chip.
    c. The gelatin chip is bioabsorbed so there is no need to have it removed after placement.
5. Because chlorhexidine is not an antibiotic, there is no risk of antibiotic resistance with use of the chlorhexidine gluconate gelatin chip.
6. A 1-year clinical trial by Henke and colleagues suggests that a chlorhexidine chip may reduce periodontal surgical needs at little additional cost.[28]

b. **Overview of Non-Controlled-Release Chemotherapeutic Agents**
1. Like a controlled-release chemotherapeutic agent, a **non-controlled-release chemotherapeutic agent** is locally delivered directly into the periodontal pocket and used as an adjunct to mechanical periodontal therapy.
2. Unlike a controlled-release chemotherapeutics which slowly releases its therapeutic effect over a sustained period of time, a non-controlled-release agent yields its effects all at once.
3. Two non-controlled-release chemotherapeutic agents have garnered recent scrutiny due to the potential benefits of using these agents as antimicrobial irrigants to disinfect periodontal pockets.
    a. **Povidone-Iodine (PVP-I)**
        1. **Chemistry.** PVP-I is a stable complex chemical compound composed of povidone, hydrogen iodine, and elemental iodine. PVP-I is classified as an iodophor—this means that the povidone serves as the carrier of iodine. The active ingredient found in Betadine® solution is PVP-I.

2. **Mechanism of Action**. PVP-I is a water-soluble, broad-spectrum bactericidal chemotherapeutic agent. Its bactericidal activity is based on the properties of iodine. When the iodine is released from the povidone carrier, its small physical size allows it to cross through the cell wall of both Gram-positive and Gram-negative bacteria. Once it enters the intracellular region of the microorganism, it oxidizes key proteins and nucleic acid structures, thereby causing the death of the pathogen.
    a. PVP-I has been shown to have virucidal properties against SARS-CoV-2 (a strain of coronavirus known to cause COVID-19).
    b. Hence, a preprocedural rinse of 0.5% PVP-I for 30 seconds has been advocated as a safe, simple, and inexpensive way of reducing the risk of disease transmission. The prophylactic use of PVP-I rinses may add an additional layer of protection for both the patient and the oral health care provider.[29,30]
3. In an *in vitro* study, dilute PVP-I (10% aqueous PVP-I) was shown to have the potential to neutralize major periodontal pathogens such as *Porphryomonas gingivalis*, *Fusobacterium nucleatum*, *Aggregatibacter actinomycetemcomitans*, *Eikenella corrodens*, and *Capnocytophaga* spp. in as little as 15 seconds of contact time.[31]
4. PVP-I has broad applications in medicine. It is indicated for disinfection of the skin and hands, mucosa antisepsis, wound treatment, and rinsing of body cavities, joints, and even the eye!
5. PVP-I has a high safety profile. Unlike antibiotics and antimicrobials, PVP-I has not been shown to be associated with bacterial resistance. Moreover, allergy to PVP-I is rare. However, individuals who are allergic to iodine or suffer from thyroid dysfunction should avoid physical contact with PVP-I. Further, individuals who inadvertently or deliberately ingest large amounts of topical PVP-I may be at risk of gastrointestinal disturbances and, in severe cases, cardiac and kidney failure. As such, PVP-I should never be consumed.
6. PVP-I is naturally golden-brown in color. If it gets on clothes, it will not permanently stain, and it can be washed off with water, soap, and stain remover. Likewise, if it gets on skin, it may lead to temporary yellow discoloration which can be washed off with soap and water.
7. In periodontal therapy, adjunctive PVP-I has been advocated as a potentially effective subgingival irrigant to disinfect the pocket. However, the therapeutic effectiveness of PVP-I is still debatable. In a human split-mouth randomized clinical trial evaluating the clinical outcomes of multiple irrigations of 10% PVP-I (the test group) versus 0.9% saline (the control group) solution as an adjunct to scaling and root planing in the treatment of periodontitis patients, no significant differences were found between either groups with respect to plaque index score, bleeding on probing score, probing pocket depth, and clinical attachment level.[32] However, in another human split-mouth study design, Hoang et al. found that the adjunctive use of PVP-I as a subgingival irrigant with conventional scaling and root planing resulted in significant improvements in clinical and microbiological parameters compared to quadrants treated with scaling and root planing alone, PVP-I irrigation alone, and water irrigation alone.[33] In contrast, in a systematic review, Sahrmann et al. found that although the adjunctive use of PVP-I resulted in statistically significant reduction in pocket reductions, the differences were too small to be clinically significant.[34]

b. **Sodium Hypochlorite**
    1. **Chemistry.** Sodium hypochlorite, also known as “bleach” in dilute solutions, is a chemical compound that is composed of sodium (Na), oxygen (O), and chlorine (Cl). Hence, its chemical structure is $NaOCl$. When sodium hypochlorite is mixed with water or placed in a watery environment (like the mouth), it appears as a colorless or slightly yellow watery liquid with an odor of household bleach.
    2. **Mechanism of Action.** When sodium hypochlorite mixes with water, it generates highly reactive hypochlorous acid ($HOCl$), which is highly potent against bacteria and fungi. An *in vitro* study demonstrated that it took sodium hypochlorite 15 seconds of contact time to neutralize *Prevotella intermedia* and *Porphyromonas gingivalis*.[35] Hence, sodium hypochlorite has many of the characteristics that make it the ideal antiseptic: broad-spectrum activity, rapid onset, high safety profile when used in dilute solutions, and ease of application.
    3. Sodium hypochlorite has broad medicinal applications. It is one of the most widely used disinfectants of wounds, ulcers, and burns, especially for combat-related injuries.

4. When used topically and at low, dilute dosage, sodium hypochlorite does not induce systemic toxicity or life-threatening complications. However, in high concentrations, sodium hypochlorite can lead to immediate hemorrhage and edema if administered directly into the bloodstream.[36]
    a. In the field of endodontics, sodium hypochlorite is commonly used as a root canal irrigant because of its effective antimicrobial properties and its tissue-dissolving capabilities. Sodium hypochlorite also has a high safety profile when it stays confined within the root canal system.
    b. However, if sodium hypochlorite is injected beyond the root canal system and escapes out the apical foramen of the treated tooth into the apical periodontal tissues, it can lead to pain, swelling, hemorrhage, and potentially secondary infection, which can be infected by pathogenic flora.
5. Currently, there is no universally accepted concentration of sodium hypochlorite for use as a periodontal irrigant. As such, a wide range—0.1% to 5.25%—has been advocated depending on the different treatment protocols of individual practitioners. Although a higher concentration of sodium hypochlorite yields greater antimicrobial action, this must be weighed against the accompanying increased risk of toxicity.
6. Sodium hypochlorite holds a lot of promise as a therapeutic mouth rinse, but its effectiveness as a *subgingival irrigant* remains in question. In a randomized controlled trial that investigated the effectiveness of 0.5% sodium hypochlorite adjunctive therapy, Bizarro and colleagues found that 0.5% sodium hypochlorite + mechanical therapy failed to show significant additional gain in clinical attachment levels or better microbiological parameters compared to saline irrigation + mechanical therapy.[37]

Section 4

Topical Mouth Rinses and Toothpastes

1. **Introduction to Mouth Rinses**
    a. Many mouth rinses (also known as mouth wash) available today are therapeutic mouth rinses. **Therapeutic mouth rinses** are mouth rinses that have actual medicinal benefit (provide some therapeutic action) to the patient in addition to the simple goal of breath freshening and halitosis reduction. In contrast, cosmetic mouth rinses temporarily control bad breath but do not have any antimicrobial properties.
        1. In the context of this chapter, therapeutic mouth rinses would be rinses that decrease dental biofilm enough to resolve the gingival inflammation when used in combination with a daily self-care regimen.
        2. Clinical studies support the effectiveness of therapeutic mouth rinses used in conjunction with a proper daily self-care regimen for the reduction of dental biofilm and the control of inflammation. Insufficient evidence, however, is available to support the claim that therapeutic mouth rinses can reduce the risk of developing periodontitis or the rate of progression of periodontitis.[38,39] Over the last few decades, researchers are still searching for chemicals that can be added to mouth rinses that might actually blunt the effects of periodontitis.
    b. Note that in addition to therapeutic mouth rinses that aid in biofilm control and inflammation, other therapeutic mouth rinses are available that can benefit the patient in a variety of ways, such as decreasing the risk of developing dental caries and treatment of dentinal hypersensitivity.
2. **Characteristics That an Ideal Mouth Rinse Should Possess.** Investigations into chemical biofilm control have not yet produced a biofilm-control mouth rinse that can be used as a total substitute for mechanical biofilm control. However, these investigations have indeed produced mouth rinses that can be effective components of a comprehensive program of patient self-care. An ideal mouth rinse would possess four characteristics that are described below.
    a. **Efficacy.** The active ingredient in the rinse should be effective in inhibiting (bacteriostatic) or killing periodontal pathogens (bactericidal).
    b. **Stability.** The ingredients in the mouth rinse should be stable at room temperature and have a reasonable shelf life.
    c. **Substantivity.** The active ingredient in the rinse should display the property of **substantivity**. This means that the active ingredient would be retained in the oral cavity for a while following rinsing and would be released slowly over time (usually several hours), resulting in a continuing antimicrobial effect against periodontal pathogens.
        1. Substantivity is an important characteristic because dental biofilm grows and matures continuously.

        2. An active ingredient that has the property of substantivity would continue to kill periodontal pathogens over an extended number of hours following rinsing.
    d. **Safety.** The ingredients in the mouth rinse should not produce any harmful effects to the tissues in the oral cavity or systemically to the patient.

3. **Ingredients of Mouth Rinses**
    a. Mouth rinses contain both active ingredients and inactive ingredients.
        1. An active ingredient is a component that produces the intended therapeutic benefit (e.g., a reduction in the severity of gingival inflammation associated with gingivitis).
        2. All mouth rinses also contain inactive ingredients.
            a. Some of the inactive ingredients found in mouth rinses add color, enhance flavor (such as spearmint, cinnamon, and peppermint), and act as preservatives (such as propylene glycol and parabens which add to the shelf-life of the rinse and limit the growth of microbes in the product).
            b. Although inactive ingredients may seem harmless, some inactive ingredients have the potential to provoke unintended side-effects in certain individuals. For instance, certain flavoring enhancers may provoke contact dermatitis (which is a dermatologic condition that occurs when the skin or mucosal tissues come into contact with the allergen) in certain individuals. In such an occurrence, the patient should immediately discontinue using the mouth rinse, and the clinician should refer the patient to a physician or nurse practitioner for a prompt medical evaluation. The clinician should also counsel the patient to use alternative mouth rinses that do not contain the inactive ingredient which provoked the initial allergic reaction.
            c. Many mouth rinses have been investigated for their effect against both biofilm and gingivitis.
        3. Chemicals that reduce biofilm formation to only a minor degree usually have little or no clinically significant effect against gingivitis, and therefore may not be very useful in controlling a disease, such as gingivitis. Many mouth rinses marketed today fall into this category.
        4. Among the many ingredients tested for efficacy against gingivitis, four mouth rinse ingredients that have some effect against gingivitis have been studied extensively:
            a. Chlorhexidine gluconate
            b. Essential oils
            c. Cetylpyridinium chloride
            d. Delmopinol

4. **Mouth Rinses Containing Chlorhexidine Gluconate**
    a. One group of mouth rinses currently available contains chlorhexidine gluconate as the active ingredient (**Figure 28-5**). These rinses are only available by prescription in the United States, but they can be purchased over the counter in some other countries.
        1. The prophylactic use of 0.12% chlorhexidine as a preprocedural rinse against SARS-CoV-2 has been advocated early on in the COViD-19 pandemic.

CHLORHEXIDINE GLUCONATE
ANTISEPTIC MOUTHWASH
PEPPERMINT FLAVOUR
300ml

Figure 28-5 Therapeutic Mouth Rinse. The mouth rinse pictured contains chlorhexidine gluconate as its active ingredient and requires a prescription for purchase in the U.S.

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   2. However, it still remains unknown if chlorhexidine is actually effective in neutralizing SARS-CoV-2. One study postulated that chlorhexidine may inactivate the virus, albeit weakly.
   3. Nevertheless, in spite of this, the prophylactic use of chlorhexidine may be a useful means of reducing the transmission of SARS-CoV-2.[40,41]

b. Mouth rinses containing chlorhexidine gluconate as the active ingredient have been demonstrated to reduce the severity of gingivitis in numerous clinical studies.
c. In the United States, the concentration of chlorhexidine gluconate used in prescription mouth rinses is 0.12%, but note that a higher concentration is used in mouth rinses in some other countries.
d. Currently, chlorhexidine is the most effective antimicrobial agent for long-term reduction of biofilm and gingivitis. For this reason, it is often regarded as the standard against which all other topical chemical biofilm control agents are judged.[42,43] The effectiveness of chlorhexidine gluconate mouth rinses is due to the following characteristics.
   1. Chlorhexidine is bactericidal agent that is effective against both Gram-positive and Gram-negative bacteria.
   2. Chlorhexidine binds with oral tissues in the mouth and is slowly released over time (several hours) in a concentration that will continue killing bacteria. Thus, these rinses display the property of substantivity.[43]
   3. Chlorhexidine has a very low level of toxicity and shows no permanent retention in the body.
e. The primary mechanism of action for chlorhexidine gluconate is disruption of the integrity of the cell walls of bacteria.
f. Chlorhexidine-containing mouth rinses are useful adjuncts to biofilm control in many patients. Current recommendations for use of this mouth rinse (0.12% chlorhexidine gluconate) are to rinse with one-half ounce for 30 seconds twice daily, 30 minutes after toothbrushing.
g. Several groups of patients should be considered for use of chlorhexidine gluconate mouth rinse.
   1. **Special Needs Patients.** The use of a chlorhexidine mouth rinse is suggested for specific groups of patients who have special needs. Two examples of such patients are those with immunodeficiencies who might be more susceptible to infections in general and those who are unable to perform biofilm control because of physical and/or cognitive impairment.
   2. **Postsurgical Care Patients.** Following periodontal surgery, it is frequently difficult for patients to perform adequate mechanical biofilm control during the healing period without damaging the surgical site. In these patients, chlorhexidine mouth rinses can be used for postsurgical rinsing as a temporary adjunct to mechanical biofilm control. Use of a chlorhexidine mouth rinse following periodontal surgery for 4 to 6 weeks can be effective in many patients to promote healing.
   3. **Patients with *Candida* Infections**. Oral candidiasis is a common opportunistic infection of the oral cavity caused by an overgrowth of a yeast-like fungus, the commonest being *Candida albicans*. Note that a variety of medications are used to control *Candida* infections, but chlorhexidine mouth rinses can be used as a disinfectant for dental appliances such as complete dentures or partial dentures in patients who are at risk of developing oral candidiasis.
   4. **Patients with High Caries Risk.** Chlorhexidine is also effective against the bacteria responsible for dental caries. Rinsing with chlorhexidine mouth rinses has been used to reduce the counts of caries-causing bacteria in certain patients.
   5. **Patients with Oral Piercings or Dental Implants.** Chlorhexidine mouth rinses have also been recommended for use by patients for aftercare of oral piercings and dental implants.
   6. **Patients with Dry Mouth.** Chlorhexidine rinsing is recommended for patients who suffer from dry mouth to prevent caries and periodontal diseases. Many studies have shown that some medications reduce salivary flow and cause dry mouth conditions. One example of a patient that may experience severe dry mouth is a person who has been treated with head and neck radiation therapy.
   7. **Preprocedural Rinses for Periodontally Diseased Patients Prior to the Use of Ultrasonic Instrumentation**. Chlorhexidine mouth rinses have been evaluated for their effectiveness as a preprocedural rinse for dental office procedures producing aerosols. Studies suggest that a preprocedural chlorhexidine rinse eliminates the majority of bacterial aerosols generated by the use of an ultrasonic unit.[46,47]
h. **Limitations.** Chlorhexidine mouth rinses do have their limitations.[43–45]
   1. Chlorhexidine is an antiplaque agent that can prevent plaque formation but its mode of action does not allow it to remove plaque already present on tooth surfaces efficiently. In a randomized split-mouth study, Zanatta and colleagues found that a 0.12% chlorhexidine gluconate mouth rinse had little

        antiplaque and antigingivitis effect on already-formed plaque-covered surfaces. These results confirm the diminished effect of chlorhexidine on structured biofilm and reinforce the necessity of mechanical biofilm disruption before using chlorhexidine mouth rinse.[41]
        2. Also, the chlorhexidine molecule reacts with anionic surfactants (like sodium lauryl sulfate) present in certain toothpaste formulations, thus reducing the effectiveness of the chlorhexidine.[38]
        3. The most common side effects of long-term usage of chlorhexidine mouth washes are taste alterations, calculus formation, and extrinsic staining of tooth surfaces and restorations.

5. **Mouth Rinses Containing Essential Oils**
    a. Chemicals referred to as essential oils have been used as active ingredients in some mouth rinses for many years. Chemical agents included in the group of chemicals called essential oils include thymol, menthol, eucalyptol, and methyl salicylate.
    b. Mouth rinses containing essential oils are available over the counter (i.e., available without a prescription). Listerine mouth rinse is one example of a rinse containing essential oils, but there are other products on the market with similar ingredients.
    c. Numerous investigations related to the efficacy of essential oils in controlling gingivitis have been published in the literature.
        1. When used in combination with a daily self-care regimen, essential oils may control biofilm growth. As such, essential oils have received the Seal of Acceptance from the American Dental Association (ADA) for their effect against gingivitis.
        2. A 6-month controlled clinical study demonstrated that the essential oil mouth rinse and the chlorhexidine mouth rinse had comparable anti-plaque and anti-gingivitis activity.[48]
        3. Several investigations found no significant differences with respect to reduction of gingival inflammation between an essential oil mouth rinse and a chlorhexidine mouth rinse. In long-term use, the essential oil mouth rinse appears to be a reliable alternative to chlorhexidine mouthwash with respect to parameters of gingival inflammation.[49,51]
        4. The mechanism of action of essential oils appears to be disruption of the integrity of the cell wall and inhibition of certain bacterial enzymes.
        5. Essential oil mouth rinses are much less expensive than chlorhexidine mouth rinses and can be purchased without a prescription. Insofar as the side effects associated with chlorhexidine mouth rinse (like staining and taste alteration) which may limit patient compliance, essential oil mouth rinses can have a distinct role in the management of patients with periodontal diseases.
6. **Mouth Rinses Containing Quaternary Ammonium Compounds**
    a. Some mouth rinses currently marketed contain the quaternary ammonium compound cetylpyridinium chloride as the active ingredient.
    b. This surface active agent also kills bacteria by disrupting bacterial cell walls.
    c. This chemical agent binds to oral tissues, but is released so rapidly that it has very limited substantivity, thereby minimizing its effectiveness in controlling dental biofilm.
    d. Investigations have shown that cetylpyridinium chloride can reduce the severity of gingivitis and supragingival biofilm, but the level of reduction is less than either chlorhexidine gluconate or essential oils.[51,52]
7. **Mouth Rinses Containing Delmopinol**
    a. Some mouth rinses currently marketed contain delmopinol as the active ingredient, which is a third generation morpholinoethanol derivative and tertiary amine surfactant.
    b. This chemical agent forms a barrier preventing biofilm from adhering to the tooth surface and gingiva. Delmopinol interferes with the enzymes responsible for the formation of biofilm.
    c. Tooth and tongue staining side effects have been reported with delmopinol, but were not found to be comparable with the staining associated with chlorhexidine gluconate.
    d. Investigations have shown that delmopinol effectively reduces the severity of gingivitis and biofilm when used adjunctively with mechanical biofilm removal and is a good alternative to chlorhexidine gluconate for some patients that cannot tolerate the associated side effects and allergies.[53,54]
8. **Problems with Mouth Rinse Ingredients**
    a. No chemicals are completely safe for all patients, and most mouth rinses have produced unwanted side effects in some patients. Reported side-effects discussed above are outlined in **Table 28-4**.

Table 28-4 Possible Side Effects of Mouth Rinses

Essential Oils Rinses	Chlorhexidine Gluconate Rinses
Burning sensation in the mouth Bitter taste Drying out of mucous membranes	Allergic reaction Extrinsic staining of teeth Discoloration of tongue Alterations of taste Increase in calculus formation Transient anesthesia

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b. In addition to the active and inactive ingredients discussed above, some mouth rinses may contain additional ingredients which may provoke unintended consequences.
   1. **Alcohol.** Some mouth rinses have rather high levels of alcohol content, and these should be avoided in patients addicted to alcohol or pregnant mothers.
   2. **Salt.** Some mouth rinses have rather high levels of sodium, making them questionable for use in certain patients with uncontrolled hypertension (high blood pressure).

9. **Toothpastes as Delivery Mechanisms for Biofilm Control Agents.** Dentifrices, such as toothpastes and gels, would appear to be nearly ideal delivery mechanisms for chemical agents that might benefit patients because most patients use these products daily.
   a. **Categories of Toothpastes.** The ADA loosely classifies toothpastes into one of the following categories:
      1. Antitartar activity
      2. Caries prevention
      3. Cosmetic effects
      4. Gingivitis reduction
      5. Biofilm formation reduction
      6. Reduction of tooth sensitivity
   b. **Active Chemical Ingredients.** This ADA classification of toothpastes underscores the broad range of benefits that can be derived from active ingredient chemical agents added to some toothpastes.
      1. Some of these chemical agents are added to impart special benefits to periodontal patients. See **Table 28-5** for some examples of the chemical agents that can be used as active ingredients in toothpastes for their periodontal benefits.

Table 28-5 Examples of Active Ingredients in Toothpaste That Have Periodontal Benefits

Ingredients	Actions
Pyrophosphates	Reduces supragingival calculus
Stannous fluoride	Reduces supragingival biofilm Reduces gingival inflammation
Triclosan	Reduces supragingival calculus Reduces gingival inflammation
Zinc citrate	Reduces supragingival calculus

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      2. Stannous fluoride has been used successfully as an anticaries agent for many years. Studies indicate that stannous fluoride also affects dental biofilm and can also reduce the severity of gingivitis when used as an active ingredient in toothpastes.
      3. Triclosan is a topical antimicrobial agent used in many products and is now available as the active ingredient in a toothpaste.

    a. Triclosan can be combined with copolymers to enhance its substantivity (binding and subsequent slow release), which has been found to be retained in the oral cavity for 12 hours.
    b. Studies indicate that when combined with copolymers, triclosan (when delivered in toothpaste form) can decrease the severity of gingivitis more than dentifrice with stannous fluoride.[55,56] Triclosan can also be combined with zinc citrate to reduce dental calculus formation.
  c. **Inactive Ingredients**. Many toothpaste formulations contain the same inactive ingredients commonly found in mouth rinses to add color, enhance flavor, and act as preservatives. As such, like mouth rinses, certain inactive ingredients found in toothpaste formulations may have the potential to provoke an allergic response in certain individuals. While the incidence of tooth paste allergy is rare (about 0.1-0.3% of the population), the clinician should be prepared to act promptly to any suspicion of an allergic response, then refer the patient to a physician or nurse practitioner for prompt medical evaluation.[57]
  d. **The Future.** Toothpastes appear to be ideal delivery mechanisms for chemical agents that might be expected to control certain periodontal conditions, such as gingival inflammation. It is reasonable to expect that additional research in this area will result in additional toothpaste formulations that target periodontal conditions, such as gingivitis.

Section 5

Educating Patients on the Use of Complementary and Alternative Medicine (CAM) Dental Products/Remedies/Practices

Today's consumers are being inundated with a wide variety of unconventional medical and dental products/remedies/practices that are backed by unproven claims, questionable effectiveness, and dubious safety. Alarmed by this trend, the National Academy of Medicine labelled these types of products/remedies/practices under the broad umbrella known as **complementary and alternative medicine (CAM)**. CAM can be thought of as atypical medicines and health care practices that are not part of standard medical care. Some examples classified as CAM include acupuncture, tai chi or yoga for mind-body therapies, and vitamins and other nutritional therapies. Although some CAM practices have been used with much success, many CAM products/remedies/practices still need more rigorous scientific research to validate their therapeutic benefits.

Despite its growing fanfare, dentistry is not immune to the emergence of CAM therapies. As a result, the ADA cautions: "*Health care interventions, whether or not considered CAM, should be subject to testing using similar research standards and scientific rigor to provide a strong, evidence-based foundation for their safety and appropriate use. Within this context, the notion of CAM as a specific subset of interventions that belong to a specific discipline can be considered questionable.*"[58]

As a result, all members of the dental team must be able to help the patient filter out all the hyperbole and exaggeration associated with some of these unconventional, commercially available products. Equally important, dental clinicians must be prepared to educate the patient that not all over-the-counter dental products have earned the ADA Seal of Acceptance, which is the gold standard for all dental products that demonstrate safety and efficacy according to the requirements developed by the ADA Council on Scientific Affairs.

Two emerging non-ADA-approved CAM therapies that need to be reviewed are (1) charcoal/charcoal-based products and (2) oil-pulling.

1. **Charcoal/Charcoal-Based Dental Products**
  a. **Historical Overview of Charcoal/Charcoal-Based Dental Products**
    1. The first-recorded use of charcoal for oral hygiene dates back to ancient Greece. Since then, the use of charcoal for oral hygiene—in the form of charcoal paste or charcoal powder—has spread worldwide.
    2. In the 1930s and 1940s, the use of charcoal dental products was twice evaluated by the Council of Dental Therapeutics.[59,60] At the time, no evidence was provided to the Council that would support charcoal's safety and effectiveness. Furthermore, there was a lack of evidence to counter the possibility that charcoal products could be a potentially harmful substance. Thus, at each time, the Council judged charcoal-based dental products to be "not acceptable" for patient use.

b. **Current Evidence Related to Charcoal/Charcoal-Based Dental Products**
   1. Currently, much of the claims supporting the use of charcoal is based on anecdotal evidence. To date, there remains a lack of controlled clinical trials and laboratory investigations of charcoal-based dentifrices to substantiate its purported benefits (**Figure 28-6**).

Figure 28-6 **Charcoal Toothpaste.** Charcoal toothpaste is currently one of the biggest trends in oral health care and is commonly found in the toothpaste aisle of grocery stores. Charcoal is a mild abrasive—but still too abrasive for daily use and could cause damage to the enamel of the teeth. Very few charcoal toothpastes contain fluoride; the absence of fluoride may increase the risk of dental caries.

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   2. In the most comprehensive review of the safety and effectiveness of charcoal-based dental products, Brooks et al. found several health risks that may be associated with the use of charcoal-based dental products.[61] First, they found that only 8.0% of charcoal-based products contain fluoride, which may put the patient more at risk of developing caries. Second, they raised the concern that because charcoal is an abrasive material, it may cause damage to the teeth (tooth abrasion) and/or gingiva (gingival recession). Thus, the authors concluded that much of the health benefits, cosmetic, and safety claims made by manufacturers of charcoal-based dental products cannot be verified at this time.

c. **Purported Mechanism of Action of Charcoal/Charcoal-Based Dental Products.** Charcoal is highly porous, which conceivably gives charcoal the ability to "trap" chemicals. In industrial applications, some gas masks have charcoal filters that help trap and filter out noxious substances. In medicine, charcoal is administered in drug overdose cases and acute poisoning cases because it binds to these toxic substances due to its porosity and decreases the body's absorption of these harmful agents. Thinking along the same lines, manufacturers of charcoal-based dental products use this rationale to make the claim that charcoal can be used in the mouth to "trap" and remove toxins and "lift" stains off teeth (tooth-whitening claim).

2. **Oil Pulling**

a. **Historical Overview of Oil Pulling.** Oil pulling is an ancient Indian folk remedy also known as "Kavala Graha" or "Gandusha." Original practitioners of oil pulling used sunflower and sesame oils to prevent gingival bleeding, caries, and oral malodor. Other oils that have been used as part of this remedy include coconut oil, palm oil, and olive oil (**Figure 28-7**).

Figure 28-7 **Oil Pulling.** Oil pulling involves swishing approximately 1 tablespoon of oil—typically coconut, sesame, or sunflower oil—for about 20 minutes, then spitting it out.

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b. **Current Evidence Related to Oil Pulling**
   1. In a 2022 metanalysis investigating the effectiveness of oil pulling, no significant differences in plaque index scores and gingival index scores were noted between the oil pulling group vs the control group (either water or chlorhexidine).[62] Nevertheless, more clinical trials need to be performed to ascertain the actual effect of oil pulling on oral health.
   2. To date, limited robust scientific trials have been performed to either support or disprove oil pulling as part of the regular oral hygiene regimen.
   3. Consequently, both the ADA and Canadian Dental Association (CDA) can neither recommend nor reject oil pulling due to the lack of scientific evidence at this time.

c. **Purported Mechanism of Action of Oil Pulling.** Oil is placed in the mouth and either swished or held in the mouth for 10 to 20 minutes, then spit out. Proponents of this remedy claim the prolonged time that the oil stays in the mouth draws (or "pulls") out all the bacteria and toxins that build up in the mouth.

Chapter Summary Statement

When it comes to the treatment and management of periodontal disease, adjunctive chemotherapy is a topic of great debate. In the short term, adjunctive chemotherapy may assist in controlling periodontal disease. In the long term, however, the possibilities of antibiotic resistance and adverse side effects to the chemotherapeutic agent may result in more harm than good. So, whether you are the oral health care provider or the patient, it is important to weigh the risks and benefits of adjunctive chemotherapeutics because not all periodontal diseases are managed in the same way.

Systemic antibiotics are not routinely used to control periodontal infections because of the high risk of developing antibiotic-resistant strains. Mouth rinses can be useful adjuncts in the treatment of patients with periodontal diseases. Thus far, the most effective ingredients to control biofilm that can be incorporated into mouth rinses include chlorhexidine and the essential oils. Controlled-release chemotherapeutic agents provide a sustained local release of the therapeutic agent lasting over several days to several weeks. Toothpastes are widely used by patients and appear to be an ideal mechanism for delivery of chemical agents to aid in biofilm control. In selecting the appropriate delivery system, the clinician must weigh the efficacy of the products, ease of use, availability, and cost. Although local delivery systems do not replace existing periodontal therapies, they do have a place in the treatment of periodontitis and offer the dental team additional methods to aid in the control of periodontal diseases.

When drugs or remedies fail to perform as intended or patients do not receive sufficient education about chemotherapeutic agents, the consequences can lead to unintended or, even worse, potentially harmful outcomes. Therefore, it is essential that the dental clinician clearly appreciates the rationale for using chemical agents and keeps abreast of the emerging scientific evidence that either support or disprove present day and future chemotherapeutic agents.

Section 6

Focus on Patients

Clinical Patient Care

CASE 1

A patient shows you a bottle of mouth rinse and asks you if it would be all right to use this mouth rinse instead of brushing and flossing so frequently. You study the label on the bottle of mouth rinse and find that the active ingredients are the essential oils. How should you respond to this patient about substituting this rinse for other self-care efforts such as brushing and flossing?

CASE 2

A patient being treated by the members of your dental team has generalized periodontitis. Following your thorough explanation of the nature of periodontitis and your emphasis that this disease is indeed a bacterial infection, the patient asks this question, "If periodontitis is an infection, can you ask the dentist to give me a prescription for an antibiotic?" How should you respond to this patient's question?

CASE 3

A new patient being seen by your dental team has recently moved into your city. She has previously been treated for periodontitis and has been on periodontal maintenance for several years. She is now having trouble with mechanical biofilm control because of increasing dexterity problems. What chemical agents can you recommend that might help reduce the patient's gingival inflammation?

CASE 4

A patient returns to the office for a periodic oral examination. She recalls that she recently watched an online documentary about charcoal-based dental products. She wonders if there is any benefit to using charcoal toothpaste instead of conventional toothpaste. She also asks if the scientific evidence supports the routine use of charcoal toothpaste. How would you respond to her inquiry?

Ethical Dilemma

Your patient, Sandy, is a 24-year-old woman who has come to your office for a second opinion. Her chief complaint is that she feels that her periodontal health is not improving, and if anything, getting worse.

You review her health history, and she states that she has been under the care of her uncle, who is a 70-year-old periodontist. She has been taking tetracycline for the last 5 years and is concerned with her periodontal and overall health. She did not bring any radiographs, as she doesn't want her uncle to know about this appointment.

Your examination reveals that Sandy has relatively good oral hygiene, with slight supragingival visible calculus between her mandibular anterior teeth. However, she presents with generalized severe gingival recession, and her attachment level readings range from 3 to 6 mm, with localized 7-mm readings on her posterior teeth.

It is hard to get a full picture due to the lack of radiographs. Sandy's periodontal status is troubling, and you are not sure if the antibiotics that she is taking are actually helping her. You ask if she ever had a microbiologic analysis to sample her oral bacteria susceptibility. She is not sure of any procedures, as she "just left all of that stuff to her uncle, the periodontal expert."

1. What do you think is the possible cause of the patient's generalized recession?
2. What factors may have contributed to the patient's disease progression?
3. What ethical principles are in conflict in this dilemma?

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CHAPTER 29

Host Modulation Therapy

- Section 1 Introduction to Host Modulation Therapy
- Section 2 Potential Host Modulating Therapies in Periodontal Patients
- Section 3 Host Modulation Therapy as a Part of Comprehensive Periodontal Patient Management
- Section 4 Focus on Patients
  - Evidence in Action
  - Ethical Dilemma

CLINICAL APPLICATION

Host modulation therapy is an exciting strategy that has revolutionized our approach to treating and managing periodontal disease. Rather than targeting the microbial biofilm itself, host modulation therapy shifts the focus *to blunting the host immunoinflammatory response*. Notably, although only one host modulatory agent is presently approved for use to treat periodontal disease in the United States and Canada, the pool of potential host modulating agents is anticipated to expand as additional long-term clinical trials come down the research pipeline proving their effectiveness. Hence, the oral health care provider needs to be well-versed in how host modulation therapy works and be prepared to accept this modality as a potential treatment option in future patient care.

LEARNING OBJECTIVES

- Define the term *host modulation therapy*.
- Discuss the potential importance of host modulation therapy.
- Name some anti-inflammatory mediators.
- Name some proinflammatory mediators.
- List three types of drugs that have been studied for use as possible host modulating agents.
- Explain why subantimicrobial dose doxycyclines are useful as host modulating agents.
- Define the term *subantimicrobial dose*.
- Make a list of treatment strategies for a periodontitis patient that includes host modulation.

KEY TERMS

Host modulation therapy
Osteoporosis
Biochemical mediators
Anti-inflammatory mediators
Proinflammatory mediators
Doxycycline
Subantimicrobial dose doxycycline (SDD)
Nonsteroidal anti-inflammatory drugs (NSAIDs)
Bisphosphonates
Anticytokine therapy
Statins
Probiotics
Specialized proresolution mediators (SPMs)

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Section 1

Introduction to Host Modulation Therapy

Conventional periodontal therapy focuses on the mechanical removal of the dysbiotic biofilm, and, in some severe cases, adjunctive systemic and/or local antimicrobial therapy may be warranted. However, with the alarming rise of antibiotic resistance, a new type of therapy that counters periodontal disease from a different angle has been proposed. This type of therapy is known as host modulation therapy.[1–4] Unlike adjunctive antimicrobial agents that target the biofilm-induced *infection*, host modulation therapy targets the *inflammation*. Further, because host modulation therapy solely targets the inflammation, it neither leads to nor contributes to antibiotic resistance. To shed more light on this novel therapeutic approach, this section will report on the current state of the development of host modulation therapy that are undergoing clinical trials, including several relatively novel agents with potential host modulatory effects.

1. **Host Modulation Therapy Defined.** The intent of **host modulation therapy** is to manipulate the status and/or function of the host inflammatory response to the bacterial challenge, rather than directly targeting the pathogens, as with a systemic/local chemotherapeutic agent. As such, the therapeutic benefits of host modulating agents are via mechanisms unrelated to the antimicrobial properties of many antibiotics/antimicrobial agents. To put it in another way, host modulation therapy works by "controlling the inflammation to control the infection."[5]
2. **Examples of Host Modulation Therapy in Medicine**
    a. Host modulation therapy is not a new concept in medicine, and it has been a part of the medical care for patients with a variety of systemic diseases for many years. One example of a common systemic disease for which physicians frequently use host modulation therapy is osteoporosis.
    b. **Osteoporosis** is a progressive bone disease that is characterized by a decrease in both bone mass and bone density that can lead to an increased risk of bone fractures. Osteoporosis affects millions of patients in the United States.
    c. Treatments for osteoporosis can be broadly divided into two categories based on how the treatment medications affect bone. One group of medications reduces bone resorption, whereas the second group of medications stimulates bone formation.
        1. Antiresorptive agents, which include medications such as estradiol, selective estrogen receptor modulators (Evista®), and bisphosphonates (Fosamax®), reduce bone resorption and help to preserve bone mineral density.
        2. Bone-forming agents, such as teriparatide (Forteo®), stimulate bone formation, thereby increasing overall bone mineral density.
    d. Patients who receive the types of medications discussed above are said to be receiving host modulation therapy; that is, the host response to the disease (loss of bone mass and density) is modulated to minimize the effects of the disease to prevent bone fractures.
3. **Importance of Host Modulation Therapy in Dentistry.** The potential importance of host modulation as one strategy in managing periodontitis patients is huge.
    a. Many adults show signs of periodontal disease, with periodontitis affecting approximately 65 million adults in the United States, according to the most recent national estimates.[6] Because so many patients have inflammatory periodontal diseases, there is a continuing need for cost-effective strategies that can be combined with conventional periodontal treatment for managing periodontitis patients.
    b. As the population in the United States ages, it is reasonable to expect the prevalence of periodontitis to increase, making the need for the most cost-effective therapy even greater.
    c. In addition, there is mounting evidence that periodontal health and several systemic conditions (such as diabetes and cardiovascular disease) are linked, again making it likely that the demand for periodontal therapy will increase over the upcoming decades.
4. **Review of Host Responses That Can Be Modulated**
    a. The fundamental pathologic processes for periodontitis are as follows:
        1. Bacteria (and bacterial products) that are a part of the plaque biofilm initiate an inflammatory response in the periodontium. The bacteria provoke the immune cells to release **biochemical mediators** (i.e., biologically active compounds) that activate the inflammatory response.

2. The inflammatory response functions as a protective mechanism that keeps the bacterial infection from doing serious harm to the periodontium in many patients, partly through the production of chemicals called anti-inflammatory mediators.
3. **Anti-inflammatory mediators** are biochemical mediators that are protective to the host by limiting the bacterial-induced inflammatory response from doing serious harm to the periodontium. These anti-inflammatory mediators include the cytokines interleukin-4 (IL-4) and interleukin-10 (IL-10). **Box 29-1** lists some important anti-inflammatory biochemical mediators.

Box 29-1 Examples of Anti-Inflammatory Biochemical Mediators

The following biochemical mediators play a key role in limiting the tissue-damaging effects of the proinflammatory response:

- Interleukin-4 (IL-4)
- Interleukin-1 receptor antagonist (IL-1ra)
- Interleukin-10 (IL-10)
- Tissue inhibitors of matrix metalloproteinases (TIMPs)

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4. If the bacterial challenge is excessive and disproportionate, however, the nature of the protective responses changes, resulting in the overproduction of biochemical mediators that can lead to actual damage within the periodontium.
   a. Biochemical mediators called **proinflammatory mediators** can damage the periodontium. These proinflammatory biochemical mediators include chemicals such as matrix metalloproteinases (MMPs), certain cytokines, prostanoids, and other less well-understood mediators.[7–11]
   b. Specific examples of these proinflammatory mediators include prostaglandin E_2 (PGE_2), interleukin-1 alpha (IL-1α), interleukin-1 beta (IL-1β), interleukin-6 (IL-6), interleukin-8 (IL-8), and tumor necrosis factor-alpha (TNF-α).
   c. For example, one of the MMPs is an enzyme called collagenase that can actually break down collagen. Collagen is one of the major building blocks in the periodontium, and its breakdown is part of the fundamental pathology in periodontitis.
   d. **Box 29-2** lists some of the proinflammatory biochemical mediators.

Box 29-2 Examples of Proinflammatory Biochemical Mediators

These biochemical mediators are produced by the host immune cells in response to microbial invasion. Elevated levels of these mediators can lead to tissue destruction:

- Interleukin-1 (IL-1)
- Tumor necrosis factor-alpha (TNF-α)
- Interleukin-6 (IL-6)
- Interleukin-8 (IL-8)
- Matrix metalloproteinases (MMPs)
- Prostaglandin E_2 (PGE_2)

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5. In the periodontium, these altered host defense responses can result in both breakdown of connective tissue fibers and resorption of alveolar bone (the precise types of tissue destruction seen in periodontitis).
6. Much of the destruction of the periodontium that accompanies periodontitis is thought to be a result of these altered processes that occur as part of the host defenses (the host inflammatory and immune responses).
   a. Fundamentally, the concept of host modulation therapy is to blunt the damaging effect of the altered host responses by modifying (or modulating) the effect of these destructive biochemical mediators.
   b. Modulation of host defenses is currently an important focus for periodontal research, and host modulation therapy will undoubtedly play a far larger role in periodontal therapy in the future.

Section 2

Potential Host Modulating Therapies in Periodontal Patients

To date, only one host modulatory agent has been approved in the United States and Canada to treat and manage periodontal disease: subantimicrobial dose doxycycline (also known as low-dose doxycycline; brand name = Periostat®). Fortunately, with the expansion of our understanding of the host immunoinflammatory mechanisms that drive the pathogenesis of periodontal disease, newer therapeutic agents with possible host modulatory effects will undoubtedly emerge. Hence, the pool of medications approved for host modulation therapy will most likely expand, increasing the hope for more novel and effective host modulating agents to combat periodontal inflammation. **Figure 29-1** diagrams where in the inflammatory pathway some potential host modulating therapeutic agents can blunt the inflammatory process.

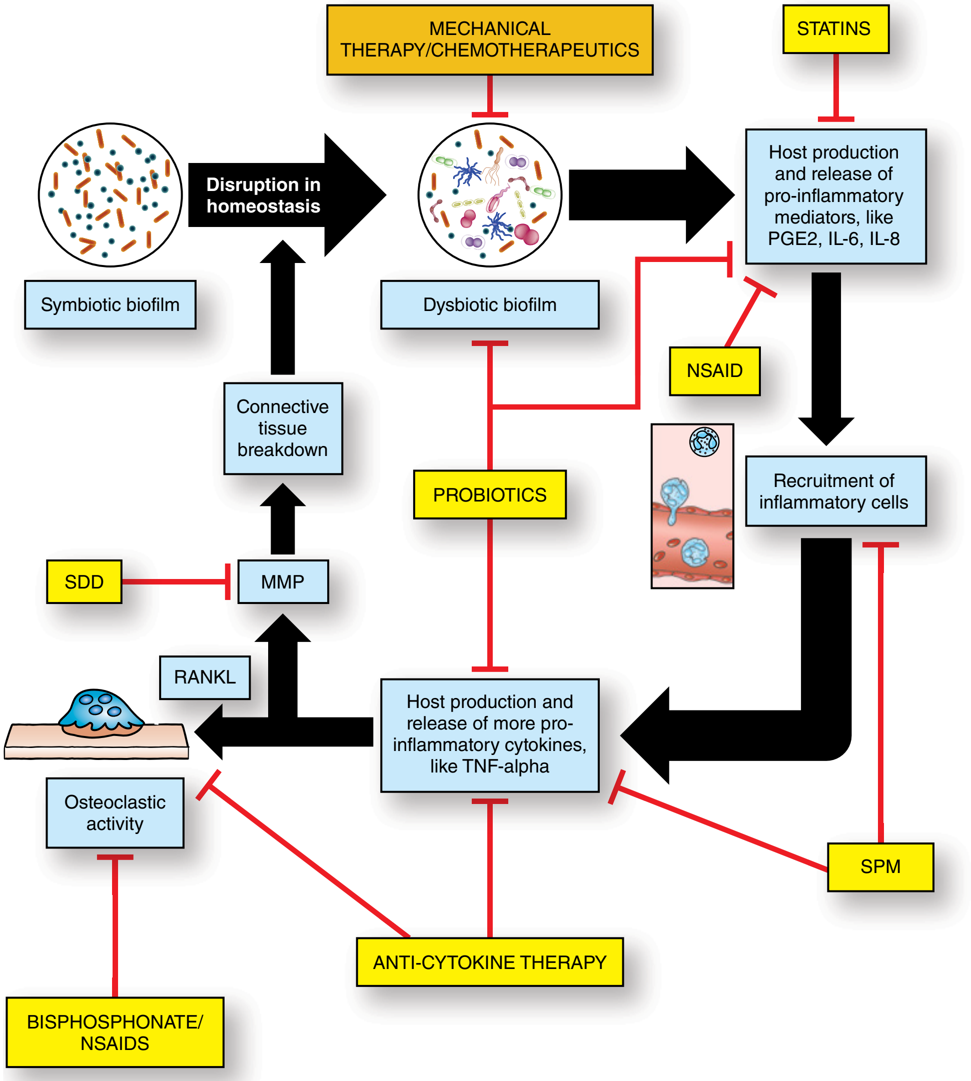
MECHANICAL THERAPY/CHEMOTHERAPEUTICS
STATINS
Disruption in homeostasis
Host production and release of pro-inflammatory mediators, like PGE2, IL-6, IL-8
Symbiotic biofilm
Dysbiotic biofilm
NSAID
Connective tissue breakdown
PROBIOTICS
Recruitment of inflammatory cells
SDD
MMP
RANKL
Host production and release of more pro-inflammatory cytokines, like TNF-alpha
Osteoclastic activity
SPM
ANTI-CYTOKINE THERAPY
BISPHOSPHONATE/ NSAIDS

Figure 29-1 Targets of Various Host Modulating Agents on the Inflammatory Pathway. By blocking the complex host inflammatory response at various phases of the inflammatory process, a host modulating agent can potentially impede the tissue damage caused by inflammation. Nevertheless, it should be emphasized that as of the publication of this textbook, SDD is the only host modulating agent approved to treat periodontitis in the US. Key: SDD = subantimicrobial dose doxycycline; SPM = specialized proresolution mediator; RANKL = receptor activator of NF-kappa ligand; MMP = matrix metalloproteinase. Note: Red lines refer to blocking targets.

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1. **Use of Doxycycline**
    a. **Doxycycline** is a tetracycline-class antibiotic drug that has been used to treat a wide variety of bacterial infections.
        1. As with other antibiotic medications, doxycycline must be given in doses high enough to affect the targeted bacteria to help the body fight an infection.
        2. Antibiotic doses high enough to inhibit or kill bacteria are sometimes called antibacterial doses. A typical antibacterial dose for doxycycline would be 50 to 100 mg every 12 hours.
        3. However, at a dose of 20 mg, **subantimicrobial dose doxycycline (SDD)** functions as a host modulating agent, rather than as an antibiotic. When combined with nonsurgical scaling and root planing, SDD has been shown to be a safe and effective therapeutic strategy to treating periodontal disease.[12–24]
            a. At a low dose (below that needed for any antibacterial effect), SDD inhibits the effects of the enzyme collagenase (one of the MMPs).
            b. As already discussed, MMPs (such as collagenase) are released by host cells as part of the inflammatory response. Elevated levels of MMPs cause the tissue breakdown that typically occurs in periodontitis. Suppressing the action of collagenase can inhibit the progress of periodontal tissue breakdown.
            c. Because SDD functions by altering the body's defenses by inhibiting part of the destruction that can occur in periodontitis instead of directly targeting pathogenic microorganisms, SDD is the classic example of a host modulating agent.[12–24]
                1. SDD must be taken twice daily orally to be effective in periodontitis patients.
                2. No antibacterial effect on the oral bacteria or bacteria in other parts of the body have been found with the use of SDD.
                3. Studies of this drug have also shown a clinical benefit when used as an adjunct to periodontal instrumentation.
                4. Although tetracyclines used at antibacterial doses can have side effects (including nausea, vomiting, photosensitivity, and hypersensitivity reactions), SDD appears to be accompanied by a very low incidence of adverse effects.
            d. Studies have shown reductions in probing depths and gains in clinical attachment levels, as well as the prevention of periodontal disease progression with the use of SDD in periodontitis patients.
2. **Use of Chemically Modified Tetracyclines**
    a. Chemically modified tetracyclines are derivatives of tetracycline group of drugs that lack antimicrobial action but have potent host modulating affects.
    b. Although these drugs lack antimicrobial action, they can inhibit the effects of elevated MMPs, proinflammatory cytokines, and other destructive chemical mediators.[25]
    c. Bone resorption also can be suppressed by reducing the effects of inflammation with these medications.
    d. Development of resistant bacteria and gastrointestinal toxicity seen with antibacterial doses of the tetracyclines is not associated with chemically modified tetracyclines.[25]
    e. Chemically modified tetracyclines are currently being investigated as potential host modulation therapeutic agents in the management of chronic diseases such as periodontitis, but further research is needed to demonstrate their efficacy and safety in periodontal patients.
3. **Use of Nonsteroidal Anti-Inflammatory Drugs**
    a. **Nonsteroidal anti-inflammatory drugs (NSAIDs)** have been used for many years to treat pain, acute inflammation, and chronic inflammatory conditions.
        1. **Box 29-3** lists various NSAIDs.

Box 29-3 Examples of NSAID Medications

- Salicylates (aspirin)
- Indomethacin
- Ibuprofen
- Flurbiprofen
- Naproxen

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        2. NSAIDs can reduce tissue inflammation by inhibiting the action of prostaglandins, such as PGE_2.
    b. In periodontal studies, systemically administered NSAIDs have been evaluated for their effect on periodontitis (a disease intimately associated with inflammation).
        1. In the periodontium, NSAIDs can both reduce inflammation and inhibit osteoclast activity.
        2. Some NSAIDs, when administered daily over 3 years, have been shown to slow the rate of alveolar bone loss associated with periodontitis.
        3. More research is needed in this area to clarify the efficacy and safety of using NSAIDs as host modulating agents in periodontal patients.
    c. Long-term use of NSAIDs in periodontitis patients is not recommended because of significant systemic side effects that can develop with the use of these drugs.
        1. Side effects from NSAIDs can include gastrointestinal problems, hemorrhage (bleeding), and kidney or liver impairment.
        2. In addition, when a patient with periodontitis stops taking daily doses of NSAIDs, there can be an acceleration of the bone loss seen prior to taking the drugs.
        3. At present, no NSAIDs have been approved for the treatment of periodontal disease.
    d. Even though NSAIDs are not currently approved for use in periodontal therapy as host modulatory agents, they are discussed here because of the extensive, ongoing research investigating the potential application of NSAIDs in periodontal therapy.
    e. In addition to systemically administered NSAIDs, topically applied NSAIDs have been studied for their possible benefits to periodontitis patients.
        1. Topical NSAIDs have also been shown to reduce PGE_2 in gingival crevicular fluid in periodontitis patients.[26–29]
        2. Topically administered NSAIDs have not been approved for the management of periodontitis, but more study of the possible use of topical NSAIDs is warranted.

4. **Use of Bisphosphonates**
    a. **Bisphosphonates** are drugs that inhibit the resorption of bone by altering osteoclastic activity (bone resorption activity), though the mechanism of action for these drugs is not fully understood.
    b. Early research indicates there may be some benefit to periodontitis patients from the ability of bisphosphonates to alter osteoclastic activity.[27,30]
    c. Some of the bisphosphonates have side effects that may limit their use in periodontitis patients, but they are discussed in this section because of the interest that has been shown in these drugs over the last few years.
    d. One of the possible side effects of these drugs is osteonecrosis of the jaws following their extended use.[31]Osteonecrosis is the destruction and death of bone tissue, in this case the bone tissue of the jaw. Studies are underway to clarify the precise risk and etiology of this serious side effect of bisphosphonates.
    e. At present, no bisphosphonate drugs have been approved for the treatment of periodontal disease.

5. **Use of Anticytokine Therapy**
    a. **Anticytokine therapy** was originally introduced to control systemic inflammatory autoimmune disorders, such as rheumatoid arthritis and inflammatory bowel syndrome (IBS). Studies have also demonstrated a potential application of anticytokine therapy in the treatment and management of periodontitis. This is because periodontitis and chronic autoimmune disorders (such as rheumatoid arthritis) share common inflammatory pathways. For example, the overexpression of TNF-α is linked to both the onset and development of debilitating rheumatoid arthritis and periodontal disease. Hence, anticytokine therapies that block TNF-α binding may suppress inflammation, and likewise may hold much promise in controlling periodontal inflammation.[32]
    b. Two novel anticytokine therapies are currently being investigated for their promising effect on suppressing the inflammatory response.[33,34]
        1. The first is the neutralizing monoclonal antibody known as denosumab (Prolia®). As an anti-RANKL agent, denosumab blocks the binding of osteoblast-produced RANKL to osteoclast-receptor RANK, thereby suppressing osteoclastic-mediated bone resorption.
        2. The second type is a receptor antagonist that blocks the action of proinflammatory mediators, such as TNF-α. Some of the more common TNF-α antagonists that have recently gained approval are etanercept (Enbrel®), adalimumab (Humira®), and infliximab (Remicade®).

   c. Several preliminary studies have shown that, when used as an adjunct to nonsurgical instrumentation, anticytokine therapy may confer additional clinical benefits in suppressing the inflammatory activity that occurs in periodontitis.[33–35] However, anticytokine therapy is not without risks. First, by their nature, anticytokine agents suppress the systemic inflammatory response, thereby making individuals more susceptible to other diseases. Second, the suppression of a single cytokine, such as TNF-α alone, may not be sufficient in limiting the destructive inflammation driven by a redundant cytokine network.[36]
   d. At this point, more large-scale clinical trials are needed to determine the safety and efficacy of applying anticytokine therapy to periodontal treatment.
6. **Use of Statins**
   a. **Statins** are medications widely prescribed to prevent cardiac and cerebrovascular events. The primary mechanism of action is mediated by its ability to inhibit the key enzyme in endogenous cholesterol production, known as HmG-CoA reductase inhibitor.
   b. But independent of its role in reducing cholesterol, compelling evidence has shown that statins may have a beneficial effect in modulating the immunoinflammatory response. Specifically, statins exert their anti-inflammatory effects on all vascular cells (endothelial cells), blood cells (e.g., platelets), and inflammatory cells (e.g., circulating leukocytes in the blood vessels) involved in the atherosclerotic process by downregulating the intracellular signaling molecules responsible for the production of proinflammatory mediators and C-reactive protein (CRP).[37] In other words, statins blunt the production of several key proinflammatory chemokines, including interleukin-6 (IL-6) and interleukin-8 (IL-8).[38]
   c. In a recent systematic review, Donos and colleagues concluded that local delivery of statin gels into infrabony pockets led to statistically significant reductions in probing pocket depths by approximately 1.83 mm at a 6-month follow-up. Nevertheless, this optimism should be tempered as more long-term studies need to be done to confirm the benefit of statins as an effective host modulator in periodontal thearpy.[35]
7. **Use of Probiotics**
   a. Dietary supplementation is another avenue that may hold promise in modifying the host response to periodontitis, especially if it is possible to enhance the resolution of inflammation through dietary supplements.
   b. A **probiotic** is a dietary substance or preparation that contains the "good" microorganisms that keep the body healthy and in homeostatic balance when consumed or applied to the body. *Lactobacillus*, *Streptococcus*, *Bifidobacterium*, and *Enterococcus* are the beneficial bacteria found in many probiotic products, such as yogurt, fermented foods, and probiotic supplements.
   c. Probiotics exert their host modulatory effects by regulating the microbial population residing within the biofilm and by suppressing pathogen colonization of the host. Further, probiotics naturally produce special antibiotic-like compounds, known as bacteriocins, which destroy disease-causing microorganisms.[39] In all, probiotics naturally protect host cells without the use of exogenous, synthetic systemic/local antimicrobial chemotherapeutics.[40]
   d. Current evidence is conflicting when it comes to the effectiveness of adjunctive probiotic therapy. One recent systematic review reported significant reduction in probing pocket depths and clinical attachment loss (CAL) gains in patients receiving adjunctive probiotics and nonsurgical periodontal instrumentation at 3-month follow-up and 12-month follow-up.[40] In contrast, another systematic review concluded that adjunctive probiotic therapy did not confer additional clinical benefits compared to individuals who received nonsurgical periodontal instrumentation alone.[35]
   e. All existing studies investigating the effectiveness of probiotics in periodontal therapy are based on small sample sizes and short follow-up times. Hence, additional studies that involve larger populations and longer follow-up periods are needed to confirm the effectiveness of adjunctive probiotic therapy.
8. **Specialized Proresolution Mediators (SPMs)**
   a. The possibility of consuming foods with specific, health-protective biomolecules, such as essential fatty acids, is currently being investigated for its effect upon periodontitis.[41-43]
   b. Consuming foods rich in omega-3 fatty acids (fish, flaxseed oil, soy foods, and nuts) to modify the host response to periodontitis and reduce the tissue-destructive impact of periodontitis would certainly be a straightforward strategy for host modulation therapy if studies demonstrate the efficacy of such supplements.

   c. One such derivative of omega-3 fatty acids are the **specialized proresolution mediators (SPMs)**, which include resolvins and protectins. SPMs promote the resolution phase of inflammation by limiting further neutrophilic infiltration to the target site, thereby allowing the body to return to homeostasis. It is important to note that SPMs do not block the initial phase of inflammation because the inflammatory response is a natural response to infection.
   d. Preliminary studies have shown that topical application of SPMs may be able to limit further bone loss and attachment loss in animal models.[44]
   e. Currently, large clinical human trials are underway to test the effectiveness of SPMs and substantiate the initial promising results.
9. **Use of Other Types of Host Modulation Agents**
   a. Several potential agents have been investigated for use as adjuncts to periodontal surgical procedures.
   b. These drugs do not produce the same types of effects as the previously discussed potential host modulating agents, but they are mentioned here because some authors have referred to them as host modulating agents.
   c. These agents are generally applied topically during periodontal surgical procedures and have been suggested for use for possible enhancement to wound healing or possible enhancement of regeneration of periodontal tissues following the surgery.
   d. Some agents of this type that have been investigated are enamel matrix proteins, bone morphogenetic proteins (BMP-2, BMP-7), and certain growth factors.
   e. Currently, the only local host modulation agent approved for adjunctive use during periodontal surgery is an enamel matrix protein called Emdogain®.
   f. Members of the dental team should anticipate additional host modulation products to be investigated and to appear on the market; careful evaluation of emerging host modulation products will be needed.[2]

Section 3

Host Modulation Therapy as a Part of Comprehensive Periodontal Patient Management

1. Periodontal therapy based upon minimizing the bacterial challenge has been a primary therapeutic modality for many years, and this therapy has proved to be successful in many patients.
   a. Studies indicate, however, that markers of periodontal disease in patients with periodontitis undergo little change following conventional mechanical periodontal therapy, despite successful clinical outcomes.
   b. These observations should not distract from a clinician's enthusiasm for recommending conventional periodontal therapy, but they should make clinicians eager for more therapies to be developed and to be added to those currently available.
2. Employing host modulation therapy in the comprehensive management of some periodontal patients seems to be a promising strategy to employ in the future.
   a. It should be reemphasized that, at present, the only host modulation therapy agent that has been approved by the U.S. Food and Drug Administration (FDA) for the treatment of periodontitis is subantimicrobial dose doxycycline (SDD, brand name: Periostat®). But as discussed, investigations have indicated that, at least theoretically, other possible host modulation therapies may one day be employed in periodontal patients.
   b. Also note that when used in periodontitis patients, sound clinical practice dictates that the use of SDD therapy be accompanied by all of the usual treatment strategies, including risk factor reduction (e.g., smoking cessation counseling) and bacterial challenge reduction (e.g., self-care training and periodontal instrumentation).
3. **Box 29-4** lists the array of therapeutic strategies that can be employed when managing a patient with periodontitis to illustrate that host modulation therapy may be viewed as another therapy among the long list of options available for a patient with periodontal disease.
4. **Figure 29-2** shows some theoretical possibilities for host modulation as a part of overall management of periodontitis patients.

Box 29-4 Therapeutic Options for a Periodontitis Patient

1. Patient education and training in self-care
2. Employment of motivational strategies for patient self-care
3. Reduction of the bacterial challenge by periodontal instrumentation
4. Use of local delivery systems for antimicrobial agents
5. Elimination of local contributing factors
6. Systemic risk factor reduction
7. Host modulation therapy
8. Periodontal surgery
9. Periodontal maintenance

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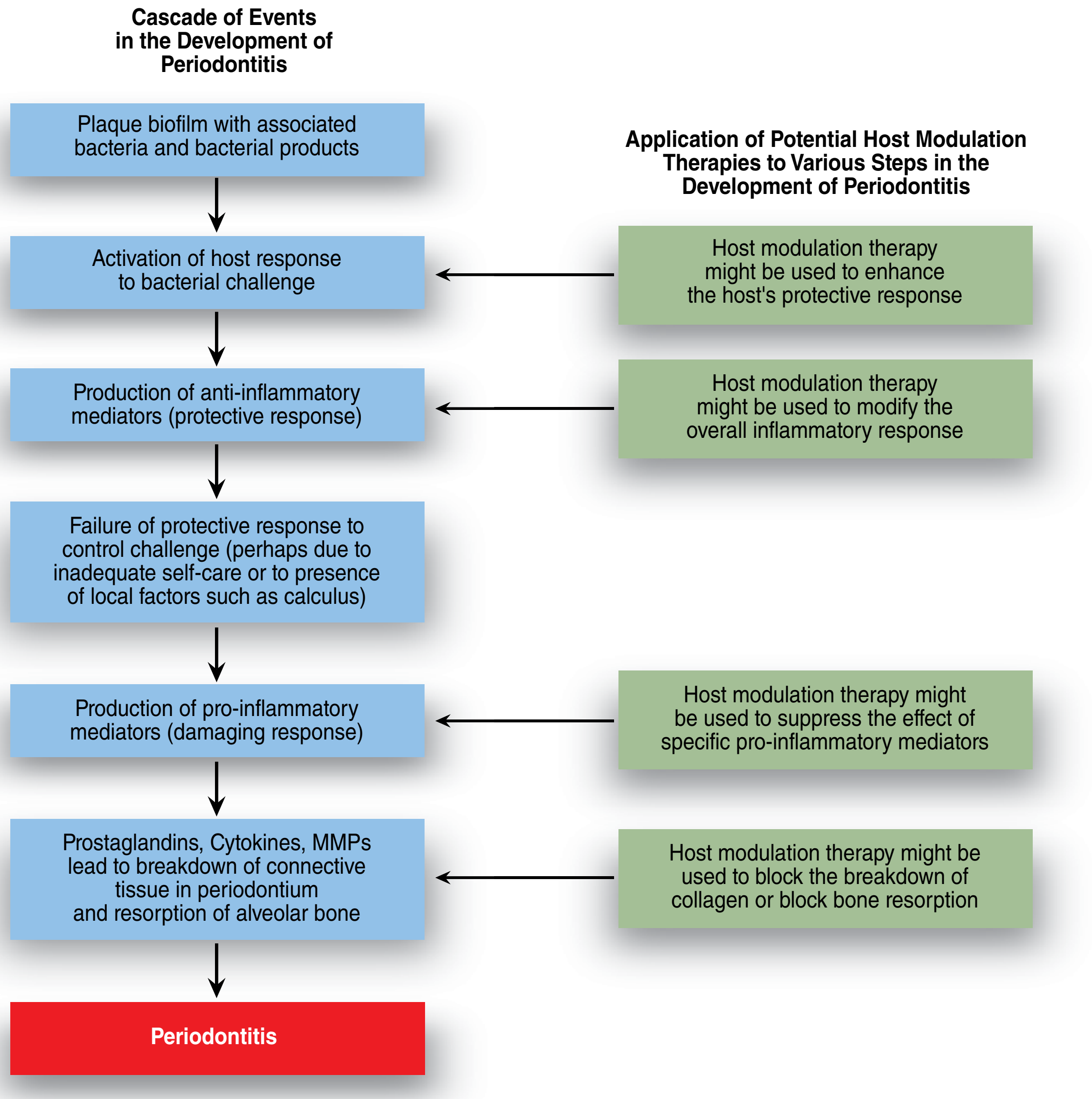
Cascade of Events in the Development of Periodontitis
Plaque biofilm with associated bacteria and bacterial products
Activation of host response to bacterial challenge
Production of anti-inflammatory mediators (protective response)
Failure of protective response to control challenge (perhaps due to inadequate self-care or to presence of local factors such as calculus)
Production of pro-inflammatory mediators (damaging response)
Prostaglandins, Cytokines, MMPs lead to breakdown of connective tissue in periodontium and resorption of alveolar bone
Periodontitis
Application of Potential Host Modulation Therapies to Various Steps in the Development of Periodontitis
Host modulation therapy might be used to enhance the host's protective response
Host modulation therapy might be used to modify the overall inflammatory response
Host modulation therapy might be used to suppress the effect of specific pro-inflammatory mediators
Host modulation therapy might be used to block the breakdown of collagen or block bone resorption

Figure 29-2 Application of Potential Host Modulation Therapies. Potential host modulation therapies can be applied to a cascade of events starting with plaque biofilm and ending with periodontitis. This figure shows the wide range of potential applications that research may make possible in the future.

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Chapter Summary Statement

In periodontal therapy, the focus of attention has shifted to moderating the host inflammatory response with the adjunctive use of host modulation therapy. To date, subantimicrobial dose doxycycline is the only FDA-approved host modulating agent approved for use in humans with periodontitis. When used at such a low dose, SDD inhibits the progress of periodontitis by targeting collagenase, rather than the dysbiotic biofilm itself.

Even though many of its practical applications remain in their infancy and need to be further investigated and developed, many other host modulatory agents with possible applications to periodontal therapy are coming down the research pipeline. As such, host modulation therapy has the potential to dramatically influence the way that oral health care providers treat and manage periodontitis. Excitingly, the future looks bright, and oral health care providers will undoubtedly need to acclimatize themselves to emerging host modulating therapies over the next several decades.

Section 4

Focus on Patients

Evidence in Action

Case 1: A new patient in your dental team's office has a periodontal diagnosis of generalized Stage III, Grade B periodontitis. Explain how host modulation therapy might be included among other treatment strategies used to help control the damage to the periodontium that normally accompanies periodontitis.

Ethical Dilemma

Tammy is a 60-year-old artist with generalized periodontitis who you see every 3 months for periodontal maintenance. As you review her health history today, she states that she is now taking Fosamax, which was prescribed by her primary care physician, Dr. James. She states that she has been diagnosed with osteoporosis and has been taking Fosamax for the last 2 months. Dr. James also told Tammy that the medication would be helpful in stabilizing her periodontal disease, which was a side benefit. He assured her that there is no downside in taking the medication.

Tammy has done some research online and has found some disturbing evidence of the possible side effects of Fosamax. She asks for your thoughts and opinion.

1. Are bisphosphonates approved for the treatment of periodontal disease?
2. What would you tell Tammy about the possible side effects of bisphosphonates?
3. Are there ethical principles in conflict with this dilemma?

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CHAPTER 30

Periodontal Surgical Concepts for the Dental Hygienist

- Section 1 Introduction to Periodontal Surgery
  - Evolution of Concepts Related to Periodontal Surgery
  - Indications and Contraindications for Periodontal Surgery
  - Possible Outcomes for Periodontal Surgery
  - Terminology Used to Describe Healing After Periodontal Surgery
- Section 2 Understanding the Periodontal Flap
- Section 3 Descriptions of Common Types of Periodontal Surgery
- Section 4 Biological Enhancement of Surgical Outcomes
- Section 5 Patient Management Following Periodontal Surgery
  - Use of Sutures in Periodontal Surgical Wounds
  - Use of Periodontal Dressing
  - Postsurgical Instructions and Follow-up Visits
- Section 6 Focus on Patients

CLINICAL APPLICATION

The primary objective for both nonsurgical and surgical periodontal therapy is to debride the root surface effectively to thoroughly remove the dental biofilm and inflamed periodontal tissues, thereby halting the destructive host inflammatory process. Granted, nonsurgical periodontal therapy alone may be sufficient for Stage I or Stage II periodontitis cases. However, in more advanced cases (Stage III and Stage IV periodontitis cases), surgical therapy may be warranted. As members of the dental team, dental hygienists must understand fundamental concepts related to periodontal surgery so that they can discuss this important topic with both patients and with other health care providers. In addition, hygienists often play a primary role in the management and maintenance of patients following periodontal surgery. A basic understanding of periodontal surgical procedures lays the framework for improved patient care during critical stages of healing of periodontal surgical wounds. This chapter provides foundational information about basic periodontal surgical concepts along with the rationale, clinical implications, and risks/benefits associated with surgical periodontal therapy.

LEARNING OBJECTIVES

- List the objectives of periodontal surgery.
- Be able to distinguish the terms *relative contraindications* and *absolute contraindications* for periodontal surgery.
- Define the terms *repair*, *reattachment*, *new attachment*, and *regeneration*.

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- Explain the difference between healing by primary intention and healing by secondary intention.
- Explain the rationale, indications, and advantages of elevating a periodontal flap.
- Explain two methods for classifying periodontal flaps.
- Describe two types of incisions used during periodontal flaps.
- Describe healing following flap for access and open flap debridement.
- Describe the typical outcomes for apically positioned flap with osseous surgery.
- Define the terms *ostectomy* and *osteoplasty*.
- Define the terms *osteogenesis*, *osteoinductive*, and *osteoconductive*.
- Define the terms *autograft*, *allograft*, *xenograft*, and *alloplast*.
- Name two types of materials available for bone replacement grafts.
- Explain why a barrier membrane is used during guided tissue regeneration.
- Define the term *periodontal plastic surgery*.
- List two types of crown lengthening surgeries.
- List some disadvantages of gingivectomy.
- Explain what is meant by *biological enhancement* of periodontal surgical outcomes.
- Name two broad categories of materials used for suturing periodontal wounds.
- Describe the interrupted interdental suture technique.
- List the general steps for suture removal.
- Describe the technique for periodontal dressing placement.
- List general guidelines for periodontal dressing management.
- Explain the important topics that should be covered in postsurgical instructions.
- List steps in a typical postsurgical visit.

KEY TERMS

Resective surgery
Osseous defect
Relative contraindication
Absolute contraindication
Repair
Reattachment
New attachment
Regeneration
Primary intention
Secondary intention
Tertiary intention
Periodontal flap
Flap elevation
Full-thickness flap
Blunt dissection
Partial-thickness flap
Sharp dissection
Nondisplaced flap
Displaced flap
Horizontal incision
Crevicular incision
Internal bevel incision
Vertical incision
Flap for access
Open flap debridement
Periodontal osseous resective surgery
Ostectomy
Osteoplasty
Apically positioned flap with osseous resective surgery
Bone replacement graft
Osteogenesis
Osteoinduction
Osteoconduction
Autograft
Allograft
Xenograft
Alloplast
Guided tissue regeneration (GTR)
Periodontal plastic surgery
Mucogingival surgery
Free soft tissue autograft (FSTA)
Subepithelial connective tissue graft
Laterally positioned flap
Coronally positioned flap
Semilunar coronally repositioned flap
Frenectomy
Crown lengthening surgery
Functional crown lengthening
Esthetic crown lengthening
Gingivectomy
Gingivoplasty
Periodontal microsurgery
Nonabsorbable suture
Absorbable suture
Periodontal dressing

Section 1

Introduction to Periodontal Surgery

The primary objectives for any periodontal procedure—whether it be of a nonsurgical or surgical nature—are (1) to eliminate, or at least minimize, periodontal inflammation and (2) to reestablish and maintain periodontal homeostatis throughout the lifetime of the patient. However, in instances where the severity of periodontitis is more severe, arresting disease progression with nonsurgical therapy alone becomes increasingly daunting. Thus, the need for periodontal surgery as part of comprehensive patient care becomes ever more apparent.

Evolution of Concepts Related to Periodontal Surgery

1. **Historical Perspective for Periodontal Surgery.** For many years, various types of periodontal surgical procedures have been recommended for dental patients with periodontitis and other periodontal conditions to control and eliminate the disease.
   a. Until the middle of the 20th century, the objectives of periodontal surgery were (1) to intentionally excise what was thought to be dead or infected periodontal tissue and (2) to reshape gingival and osseous tissues to attain a harmonious topography. These early periodontal surgical techniques are referred to as resective procedures. The term **resective surgery** refers to those procedures that simply cut away and remove some of the periodontal tissues.
   b. The concept of periodontal resective surgery (cutting away tissues) is still done today in clinical practice; however, it shares little in common with other periodontal surgical procedures that focus on regenerating periodontal tissues.
2. **Move Toward Modern Periodontal Surgical Techniques**
   a. Since the late 20th century, as our understanding of basic sciences and regenerative medicine have advanced, an evolution of both the objectives and techniques for periodontal surgery has taken place.
   b. The emphasis in periodontal surgery has shifted away from the resective types of periodontal surgery to periodontal surgical procedures that rebuild or regenerate periodontal tissues damaged or lost because of disease.

Indications and Contraindications for Surgery

1. **Indications for Periodontal Surgery.** A periodontist can employ a wide array of surgical techniques that are directed toward different outcomes. The most common indications for periodontal surgery are outlined below and in **Box 30-1**.

Box 30-1 Indications for Periodontal Surgery

- To provide enhanced access for more effective periodontal instrumentation of root surfaces by temporarily pulling back the gingival tissues to expose the underlying root surfaces and bone (i.e., flap reflection or flap elevation)
- To reduce pocket depths
- To provide access to periodontal osseous defects
- To resect or remove tissue
- To regenerate the periodontium lost due to disease
- To graft bone or bone-stimulating materials into osseous defects
- To improve the appearance of the periodontium
- To facilitate prosthodontic treatment (e.g., pre-prosthetic surgeries, such as an alveoloplasty)
- To allow for the placement of a dental implant

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   a. **To Provide Access for Better Periodontal Instrumentation of Root Surfaces**
      1. Periodontal surgery provides enhanced access and visualization to the root surfaces for more thorough periodontal instrumentation. This is done by gently lifting (or pulling back) the gingival tissues to expose the underlying root surfaces and surrounding bone for better access and visualization. The gentle lifting of the gingival tissues is known as flap reflection (or flap elevation). Since nonsurgical instrumentation of deep periodontal pockets is challenging, the improved access that is provided by periodontal surgery can be a huge advantage for clinicians.
      2. As probing depths increase, the clinician encounters difficulty in effectively reaching root surfaces with nonsurgical instrumentation alone. **Figure 30-1** illustrates a case where initial nonsurgical periodontal therapy was ineffective in removing subgingival calculus from deep periodontal pockets. Consequently, periodontal surgery was performed to gain better access to the residual deposits.

3. In addition, the presence of anatomical factors, such as radicular grooves, furcation invasions, and root concavities can hinder the best attempts in nonsurgical therapy. **Figure 30-2** shows a surgical case of burnished subgingival calculus that bridges over a root concavity that was inadvertently left behind following nonsurgical therapy. **Figure 30-3** is a surgical case of furcation invasions on molar teeth. By itself, nonsurgical treatment of a furcation invasion is extremely challenging due to the depth of the furcation defect and the inaccessibility to nonsurgically access the invasion itself. All these factors can stymie the best attempts by the clinician to nonsurgically remove the plaque biofilm burrowed inside the furcation defect.

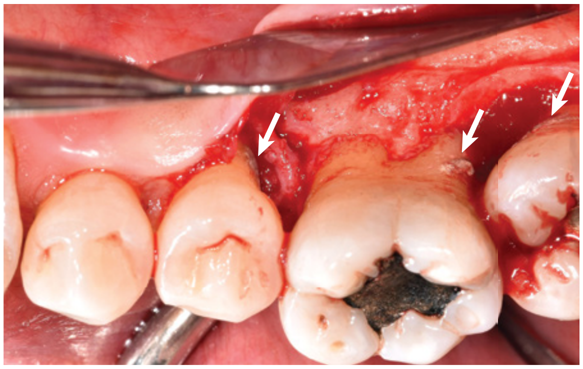


Figure 30-1 Challenges to Nonsurgical Periodontal Instrumentation: Deep Periodontal Pockets. Two months prior to this photo being taken, these teeth presented with initial probing depths exceeding 5 mm. As such, the clinician initiated nonsurgical scaling and root planing. Yet, despite the clinician's valiant efforts, nonsurgical periodontal instrumentation was ineffective in resolving the periodontal inflammation. Consequently, periodontal surgery was initiated to gain better access to the residual subgingival calculus deposits. This photo was taken at the time of flap reflection. With the flap reflected, the clinician now has better visibility and enhanced access to remove the heavy bands of residual calculus still present on the root surfaces (white arrows).

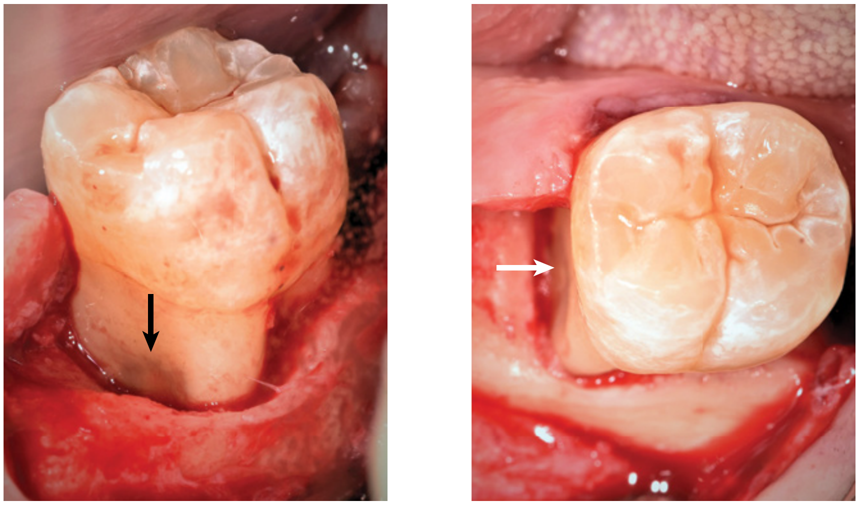


Figure 30-2 Challenges to Nonsurgical Periodontal Instrumentation: Root Convexity. Proximal and occlusal views of a lone-standing molar following surgical flap reflection. Surgical flap reflection gives the oral health care provider enhanced visiblity and access to debride the burnished calculus (black arrow on the proximal view) trapped within the recessed concavity found on the mesial root (white arrow on the occlusal view). Nonsurgical detection and removal of burnished caclulus is a frustrating task for any clinician because of how smooth and flat the residual deposit is following ineffective nonsurgical instrumentation (that was previously performed using a dull scaling instrument). To complicate matters, the burnished calculus resides within a root concavity which is an area that is extermely challenging to nonsurgically instrument. The most predicable and effective means of removing burnished calculus is to surgically reflect the flap so that effective root debridement can be performed.

Courtesy of Dr. Halide Namli, Indianapolis, IN.

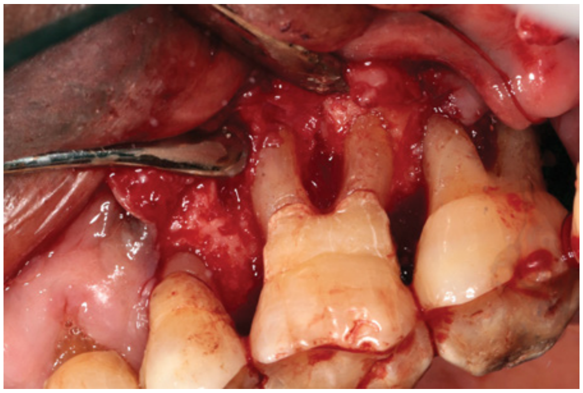


Figure 30-3 Challenges to Nonsurgical Periodontal Instrumentation: Furcation Invasions. Nonsurgical management of a furcation invasion is very challenging due to the difficulty in accessing and visualizing the full extent of the invasion itself. As such, the most predictable means of effectively debriding a furcation invasion is to gain surgical access.

4. Periodontal surgery involving carefully planned incisions through the gingiva can allow for temporary lifting of the soft tissue off the tooth surface for improved access. More details about this type of surgery are presented under flaps for access in the following descriptions of periodontal surgery.

b. **To Reduce Pocket Depths**
   1. As pocket depth increases, it becomes increasingly difficult for patients to perform effective self-care techniques. Further, plaque biofilms that thrive in the protected environment of the deep pocket can make it impossible to blunt the inflammatory process.
   2. Periodontal surgical procedures reduces the pocket depths so that a combination of daily self-care and periodic professional periodontal maintenance improves the chance of maintaining the periodontium in health throughout the life of the patient.

c. **To Provide Access to Periodontal Osseous Defects**
   1. An osseous defect is a deformity in the tooth-supporting alveolar bone usually resulting from periodontitis. **Figure 30-4** shows an osseous defect as viewed following flap elevation.

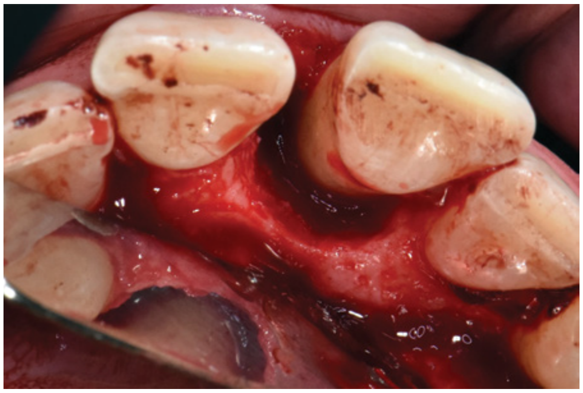


Figure 30-4 Periodontal Osseous Defect Exposed During Surgery. The soft tissues have been incised and temporarily lifted away from the teeth to reveal the bone contour. Note the extensive alveolar bone loss around the left central incisor which appears as a moat-like defect around the tooth. This type of bone defect would be an ideal site for bone replacement graft discussed later in the chapter.

        a. As periodontitis advances, alveolar bone loss results in changes in the normal contour and structure of the supporting alveolar bone.
        b. The pattern of bone loss can vary from one tooth to the next and even affect only a specific site of a single tooth, creating an array of different types of defects in alveolar bone contours. This is referred to as **osseous defects**.
    2. Periodontal surgery to modify the alveolar bone level or contour is called periodontal osseous surgery.
        a. Bone defects can be managed surgically through a variety of techniques discussed later in this chapter.
        b. Information about how osseous defects can be managed using periodontal surgery is presented under the topics osseous resective surgery, apically positioned flap with osseous surgery, bone replacement graft, and guided tissue regeneration in other sections of this chapter.

d. **To Resect or Remove Tissue**
    1. Enlarged gingival tissues can be unsightly and can also interfere with proper self-care; in some patients, enlarged gingiva can even interfere with comfortable mastication.
    2. Even though the focus of most modern periodontal surgery is not resection of tissues, this surgical approach can still be indicated in some instances.
    3. Periodontal surgery can be used to remove and reshape enlarged gingiva; additional information on this type of periodontal surgery is found in the chapter section that discusses the gingivectomy.

e. **To Regenerate the Periodontium Lost Due to Disease**
    1. One of the ultimate goals in periodontics is to be able to regenerate periodontal tissues that have been damaged by disease. The term *regenerate* describes the reconstitution of new tissue. Likewise, the objectives in periodontal regenerative surgery are to stimulate the formation of new cementum, new functionally aligned periodontal ligament fibers, and new alveolar bone that were lost due to periodontitis.
    2. Although it is not possible to regenerate the periodontium in all instances, it is possible to achieve this regeneration in many sites using some sophisticated periodontal surgical techniques; information on regenerative periodontal surgery is presented under guided tissue regeneration in a later section of this chapter.

f. **To Graft Bone or Bone-Stimulating Materials into Osseous Defects**
    1. Some periodontal osseous defects offer the opportunity for the periodontist to graft either bone or bone-stimulating materials into the defects.
    2. Although this surgery may seem quite similar to periodontal regeneration surgery, grafting bone does not necessarily imply regeneration of other parts of the periodontium, such as cementum and periodontal ligament. More information on this interesting topic is located under bone replacement graft in a later section of this chapter.

g. **To Improve the Appearance of the Periodontium**
    1. Some patients have gingival levels or gingival contours that result in an unattractive smile; periodontal surgery includes a variety of techniques for improving the appearance of the gingiva and improving the quality of a patient's smile.
    2. Many restorative techniques are available for improving the appearance of the teeth themselves. But, for many patients, alteration of the appearance of the gingiva involves active collaboration between the dental hygienist, the restorative dentist, the prosthodontist, the periodontist, and even the orthodontist to achieve a truly pleasing appearance. More information on this topic is found under the periodontal plastic surgery and crown lengthening surgery sections.

h. **To Facilitate Prosthodontic Treatment**
    1. Prosthodontic treatment may require a variety of pre-prosthetic periodontal surgical procedures that involve altering alveolar ridge contours, lengthening the crown of a tooth, augmenting the amount of gingiva and/or augmenting the bone in an edentulous site prior to implant placement.
    2. Modern periodontal surgery includes many procedures directed toward preparing the mouth for a fixed (like a bridge or implant) or removable (like a denture) dental prosthesis.

i. **To Allow for the Placement of a Dental Implant**
    1. Replacement of missing teeth with a dental implant is an option that must be considered when natural teeth are lost. The topic of dental implants is discussed in Chapter 8, but is listed here as one of the indications for periodontal surgery for completeness.
    2. Periodontal surgery can also be used to prepare sites for dental implants.
        a. One of the basic tenets of dental implant placement is that the implant must be surrounded by sound alveolar bone.

b. It is not at all unusual for edentulous sites—where implants are to be placed—to be deficient in the amount of alveolar bone needed to surround the implant. Such sites will require some type of bone grafting procedure prior to implant placement.

2. **Contraindications for Periodontal Surgery**
   a. **Relative Versus Absolute Contraindications.** A contraindication is a condition/factor that makes a particular treatment or procedure potentially inadvisable. A contraindication may be absolute or relative. Most contraindications for periodontal surgery are relative contraindications rather than absolute contraindications because the benefits of performing periodontal procedures outweigh the risks.
      1. A **relative contraindication** means that caution should be exercised when performing the procedure. In contrast, an absolute contraindication means that the procedure should be avoided because of the severe and potentially life-threatening risks involved. An example of an **absolute contraindication** for periodontal surgery might be a patient with full-blown AIDS.
      2. An example of a relative contraindication for periodontal surgery might be a patient with well-controlled hypertension (high blood pressure).
         a. A patient with uncontrolled hypertension would not be a candidate for periodontal surgery as long as the blood pressure remained severely elevated.
         b. On the other hand, a patient with well-controlled hypertension may be a suitable candidate for periodontal surgery.

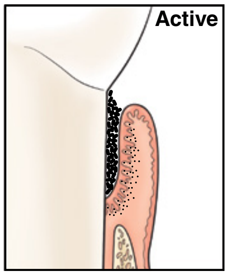
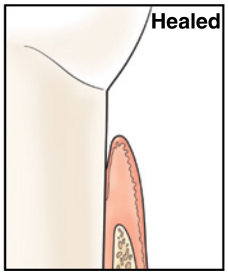
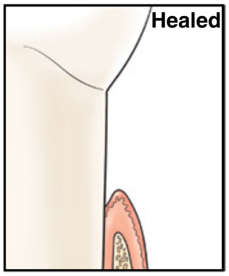
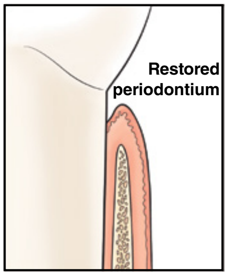
   b. **Common Relative Contraindications for Periodontal Surgery.** Common relative contraindications for periodontal surgery are outlined below.
      1. **Patients Who Have Certain Systemic Diseases or Conditions**
         a. The following systemic diseases or conditions can be contraindications for periodontal surgery:
            1. Hypertension
            2. Recent history of myocardial infarction (heart attack)
            3. Diabetes
            4. Certain bleeding disorders
            5. Kidney dialysis
            6. History of radiation to the jaws
            7. HIV infection
         b. If there is any doubt about the patient's health status or if there is any suspicion that the health status might affect the outcome of treatment, consultation with the patient's physician is always prudent.
      2. **Patients Who Are Totally Noncompliant with Self-Care**
         a. The success or failure of periodontal surgery may be dictated by how well the patient adheres to a plaque control regimen before and after surgical therapy.
         b. A lack of patient compliance will most likely compromise the surgical outcome and should be a foreboding sign that surgery may be a relative contraindication.
      3. **Patients Who Have a High-Risk for Dental Caries**
         a. Some types of periodontal surgery result in exposure of portions of tooth roots. In a patient with uncontrolled dental caries where the risk for dental caries is quite high, it may not be wise to perform the types of periodontal surgery that increase root exposure due to the potentially devastating effect of root caries.
         b. Most often a high risk for dental caries can be altered. But, when bringing the caries risk to an acceptable level is impossible, this risk can be a relative contraindication for some types of periodontal surgery.
      4. **Patients Who Have Totally Unrealistic Expectations**
         a. Periodontitis damages the tissues that support the teeth, and surgical correction of that damage does not always result in a perfectly restored periodontium even when performed by the most skilled periodontist.
         b. If a patient cannot understand the nature of periodontal surgery and cannot develop realistic expectations for the outcomes of any planned periodontal surgery, it would not be wise to proceed to periodontal surgery. Thus, patient expectations can also be a relative contraindication for periodontal surgery.

5. **Patients Who Smoke.** Smoking can jeopardize an individual's overall health (see Chapter 17). This includes, but is not limited to, delayed wound healing and impaired immune response. The extent of smoking's negative impact extends to oral health. In periodontal therapy, for instance, a smoker is more likely to not respond favorably to any type of periodontal therapy—whether it be of nonsurgical or surgical means—compared to nonsmokers. Hence, it is important to counsel the patient to undergo tobacco cessation so that improvements following any type of periodontal therapy can be predictably achieved.

Possible Outcomes for Periodontal Surgery

Table 30-1 and **Figures 30-5A-D** illustrate possible outcomes that may result from successful periodontal surgery.

Table 30-1 Possible Outcomes from Successful Periodontal Surgery

 **Figure 30-5A** © Wolters Kluwer	**Periodontal Pocket Prior to Therapy.** The periodontal pocket with plaque biofilm and inflammation within the tissues prior to therapy.
 **Figure 30-5B** © Wolters Kluwer	**Healing by Long Junctional Epithelium.** Healing in the area of the former pocket at the site by formation of a long junctional epithelium. When healing by long junctional epithelium occurs following periodontal surgery, the junctional epithelium closely attaches to the cementum (root surface).
 **Figure 30-5C** © Wolters Kluwer	**Healing with Tissue Shrinkage.** Healing at the site by resolution of inflammation may result in tissue shrinkage with exposure of some parts of the root surface.
 **Figure 30-5D** © Wolters Kluwer	**Healing by Regeneration.** Healing at the site by regeneration of the periodontal tissues.

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1. Formation of a long junctional epithelium (as can be seen in response to nonsurgical therapy).
2. Resolution of inflammation and the associated periodontal pocket (as can be seen in response to nonsurgical therapy).
3. Regeneration (which is an expectation for periodontal regenerative surgery but not expected as a result of nonsurgical therapy alone).

Terminology Used to Describe Healing After Surgery

Two sets of terminology are frequently used to describe healing of periodontal surgical wounds. One set of terms attempts to describe the various types of wound healing that can result from the surgery, and the second set of terms describes the degree of wound closure achieved at the time of surgery. The dental hygienist should be familiar with both sets of terms, because all members of the dental team are likely to use both sets when communicating with other dental health care practitioners.

1. **Terminology Describing Types of Wound Healing.** Four terms are used to describe the types of healing of the periodontium following periodontal surgery: repair, reattachment, new attachment, and regeneration. These terms are used to convey very specific wound healing concepts when describing the results of periodontal surgery.
    a. **Healing by Repair**
        1. **Repair** is healing of a wound by formation of tissues that do not fully restore the original architecture or original function of the body part.
            a. An example of healing by repair would be the formation of a scar during the healing of an accidental cut involving a finger.
            b. Certainly, the healing of the finger wound is complete following formation of the scar, but the scar tissue is not precisely the same type of tissue in appearance, form, or function that existed on that part of the finger before the cut.
        2. Repair is a perfectly natural type of healing for many types of wounds, including some wounds created during periodontal surgery.
            a. An example of repair in the periodontium is the healing that occurs following periodontal instrumentation (scaling and root planing).
                1. The usual healing of the wound created by periodontal instrumentation results in a close adaptation of epithelium to the tooth root.
                2. This adaptation of epithelium to the tooth root has been referred to as formation of a long junctional epithelium (see Chapter 25).
                3. Healing by long junctional epithelium occurs because the rate of mitosis and migration of epithelial cells across a debrided root surface is faster than other cell types. Thus, if cells of the epithelium make contact with the root surface before other cell types, then a long junctional epithelium will form.
            b. A long junctional epithelium is an acceptable outcome of healing, but it does not fully reconstitute the original periodontal tissues that were lost due to disease.
            c. To summarize, periodontal repair is characterized by the formation of a long junctional epithelium; however, there is no reconstitution or regeneration of new periodontal tissues.
    b. **Healing by Reattachment**
        1. **Reattachment** means to “attach again.” It is simply the healing of a periodontal wound by the reunion of the connective tissue and roots where these two tissues have been separated by surgical incision or physical injury, but *not* by disease.[1]
        2. Frequently in periodontal surgery, it is necessary to elevate (gently separate) healthy tissue away from the underlying tooth root or bone temporarily during some types of periodontal surgery. For example, elevating the tissue may be necessary to allow for access to remove healthy bone for the purpose of increasing the clinical crown height (surgical crown lengthening) of a tooth prior to restorative therapy. The expected type of healing is referred to as healing by reattachment.
    c. **Healing by New Attachment**
        1. **New attachment** is a term used to describe the union of a pathologically exposed root with connective tissue or epithelium. This should not be confused with healing by reattachment.

2. Healing by new attachment occurs when the epithelium and connective tissues are newly attached to a tooth root where periodontitis had previously destroyed this attachment (i.e., where attachment loss has occurred).[2,3]
3. New attachment differs from reattachment because new attachment only occurs in an area formerly damaged by disease, whereas reattachment occurs when tissues are separated in the absence of disease (frequently because of the surgical procedure).
4. **Figure 30-6** illustrates the specific area on a tooth that must have newly attached epithelium and connective tissue for the healing to be called new attachment.

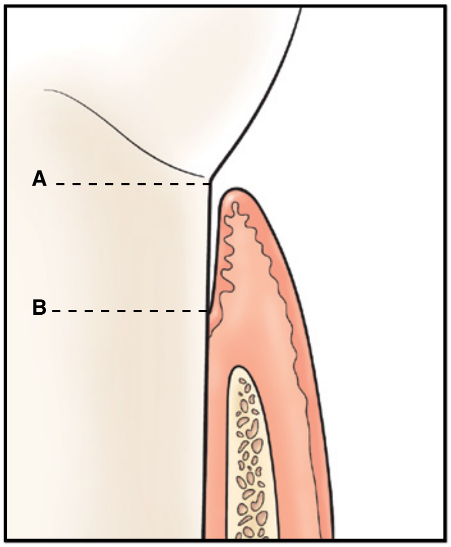

Figure 30-6 Area of the Tooth Root Involved in New Attachment. This drawing shows a site on a root where attachment loss has occurred because of disease. The region on the root from label A to label B represents the attachment loss that has occurred because of periodontitis. If the epithelium or connective tissue is attached to the root surface starting at label B, then healing by new attachment has occurred.

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d. **Healing by Regeneration**
   1. **Regeneration** is the biologic process by which the architecture and function of lost tissue is completely restored.
   2. Unlike healing by new attachment, which is characterized by the union of epithelium or connective tissue with a root surface that has been deprived of its original attachment apparatus, healing by regeneration results in the reestablishment of the original tissues that were present before the disease or damage occurred.
   3. For healing of the periodontium to be described as regeneration, the healing would have to result in the reformation of new cementum, new functionally oriented periodontal ligament fibers, and new alveolar bone.
   4. Regeneration of the periodontium is indeed possible with modern periodontal surgical procedures, but can still be challenging. Research is ongoing to improve the predictability of periodontal regenerative outcomes.

2. **Terminology Describing the Degree of Wound Closure.** A second set of terms has also been used to describe events following periodontal surgery. These terms describe the degree of wound closure (i.e., how the margins or edges of the surgical wound relate to each other following the surgery but prior to the healing).
   a. **Healing by Primary Intention**
      1. Healing by **primary intention** occurs when the wound margins are closely adapted to each other. The term *wound approximation* is used in medicine and dentistry to describe the gentle pulling together of wound edges from opposite sides so that they appear touching.

2. An example of this type of healing is a finger wound that has been stitched to bring the wound edges closely together.
3. Healing by primary intention is usually faster than the other types of healing, but it is not always possible for wound margins to be closely adapted following periodontal surgery.
4. Intraoral healing by primary intention in the periodontium may pose challenges that differ from the healing by primary intention that takes place in other sites in the body—such as healing of a finger wound. One edge of a surgical wound in the periodontium may be a tooth root that is avascular and cannot contribute any living cells to facilitate the wound healing process. This differs from healing by primary intention for a finger wound because both edges of the wound contribute living cells and blood vessels to the healing process.

b. **Healing by Secondary Intention**
   1. Healing by **secondary intention** takes place when the margins or edges of the wound are not closely adapted (i.e., the two wound edges are not in close contact with each other).
   2. When healing by secondary intention takes place, granulation tissue must form to close the gap between the wound margins prior to growth of epithelial cells over the surface of the wound.
   3. Healing by secondary intention is generally slower than healing by primary intention because more vascular and cellular events are required in this type of healing.
   4. Ideally, all wounds created during periodontal surgery are wounds that would be expected to heal by primary intention. But, in reality, many of the wounds created during surgery involve healing by secondary intention. The healing of a tooth extraction socket (where the wound edges cannot be approximated together) is a classic example of healing by secondary intention (**Figure 30-7**).

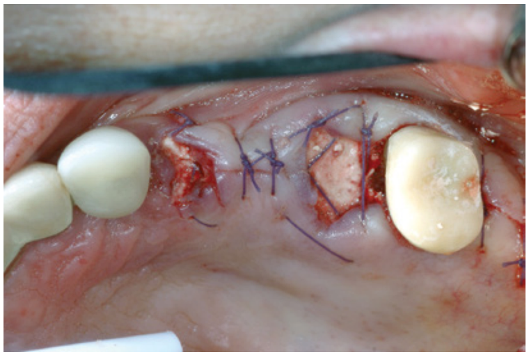

Figure 30-7 Healing by Primary and Secondary Intention. This photograph shows two types of healing occurring in the same surgical region: (1) healing by primary intention and (2) healing by secondary intention. Healing by primary intention will occur across the incision line because the wound edges are closely adapted together. In contrast, healing by secondary intention will occur over the extraction sockets because the wound edges are not able to be brought together at the time of suturing.

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c. **Healing by Tertiary Intention**
   1. With healing by **tertiary intention**, the wound is first cleaned by the surgeon and deliberately left open to allow for the purulence to drain before it is closed at a later date. This type of healing can be thought of as *delayed* primary wound healing.
   2. An example of tertiary intention is healing of a wound from a dog bite.
   3. Healing by tertiary intention is not normally a type of healing that applies to healing of periodontal surgical procedures and is mentioned here only for completeness.

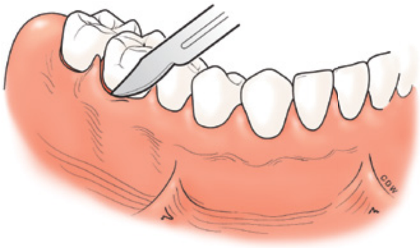
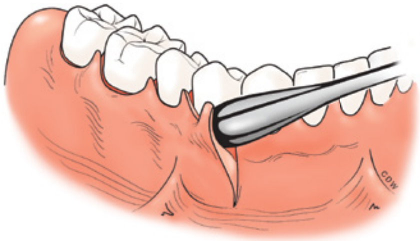
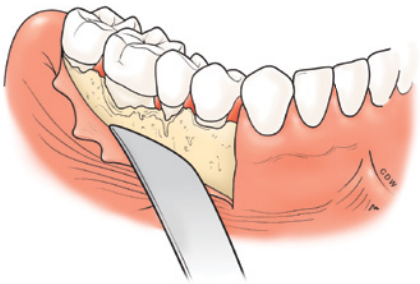
Section 2

Understanding the Periodontal Flap

Many modern periodontal surgical techniques begin by performing a periodontal flap, and the periodontal flap is an important step in most periodontal surgical procedures. As periodontitis progresses, it damages the attachment of the connective tissue to the tooth roots and destroys the surrounding alveolar bone. Treating patients with periodontitis and repairing damage done to the underlying periodontium requires gaining access both to tooth roots and to alveolar bone. As attachment loss associated with periodontitis progresses, access to tooth roots with conventional nonsurgical periodontal therapy becomes difficult, if not impossible. Elevating a periodontal flap provides the means to gaining access to the underlying periodontal structures and to the tooth roots affected by the disease. Any overview of periodontal surgery requires some understanding of the techniques involved in performing a periodontal flap. This section discusses the techniques and the relevant terminology related to periodontal flap surgery.

1. **Introduction to Periodontal Flaps**
    a. **Procedure Description**
        1. A **periodontal flap** is a surgical procedure in which incisions are made in the gingiva or mucosa to separate the soft tissues (gingiva and connective tissue) from the underlying tooth roots and alveolar bone.
        2. Separation of the soft tissues from the underlying tooth root and alveolar bone is known as **flap elevation** or reflection. The term *elevation* involves the gentle retracting of the soft tissues away from the root and the alveolar bone.
        3. Once the gingiva or mucosa is elevated off the underlying roots and bone, it can be returned and sutured to its original position (known as a nondisplaced flap), or it can be displaced to different locations (known as a displaced flap). These two types of flaps will be discussed in more detail in upcoming sections.
        4. **Table 30-2** and **Figures 30-8A-C** illustrate the step-by-step technique to performing a typical periodontal flap surgical procedure used to gain access to the underlying tooth roots and alveolar bone.
    b. **Indications for a Periodontal Flap**
        1. Most modern periodontal surgical procedures require performing periodontal flaps as a part of the procedure.
        2. Basically, the flap elevation is done to provide access to either the tooth roots and/or alveolar bone.
            a. Periodontal flaps are elevated to enhance access to tooth root surfaces and to facilitate meticulous periodontal instrumentation (scaling and root planing) that was begun as a part of nonsurgical periodontal therapy (**Figure 30-9**).
            b. The use of a periodontal flap for improved access to tooth roots is discussed in more detail in Section 3 of this chapter under the heading *flap for access*.
2. **Classification of Periodontal Flaps.** Several classification schemes are used to describe periodontal flaps. Two of the most common include (1) the degree of bone exposure after flap elevation and (2) the location of the margin (or edge) of the flap when it is sutured back into place. **Box 30-2** provides an overview of common terminology used to classify periodontal flaps.
    a. **Classification by Degree of Bone Exposure.** One method of classifying periodontal flaps is to describe the flap based upon the degree of exposure of alveolar bone following flap elevation. Using this method of classification, flaps would be categorized as either full-thickness or partial-thickness (**Figure 30-10**).
        1. The **full-thickness flap**, or mucoperiosteal flap, provides complete access to underlying bone when bone replacement grafting or periodontal regeneration procedures are anticipated.
        2. The full-thickness flap is elevated with what is generally referred to as blunt dissection.
            a. **Blunt dissection** means that the instrument used to elevate (or raise) the flap has a dull edge (i.e., blunted or slightly rounded on the edge); blunt dissection minimizes the chance of accidental damage to the flap (e.g. flap perforation).

Table 30-2 **Steps to Performing a Typical Periodontal Flap Procedure to Access the Underlying Tooth Roots and Alveolar Bone**

 **Figure 30-8A** © Wolters Kluwer	Step 1: Use a scalpel blade to make an incision to allow for separation of the soft tissue from the roots and alveolar bone.
 **Figure 30-8B** © Wolters Kluwer	Step 2: Use a periosteal elevator to elevate (or raise) the soft tissue flap away from the roots of the teeth and alveolar bone.
 **Figure 30-8C** © Wolters Kluwer	Step 3: With the periosteal elevator, fully retract the soft tissue to gain better access to the underlying root surface and alveolar bone.

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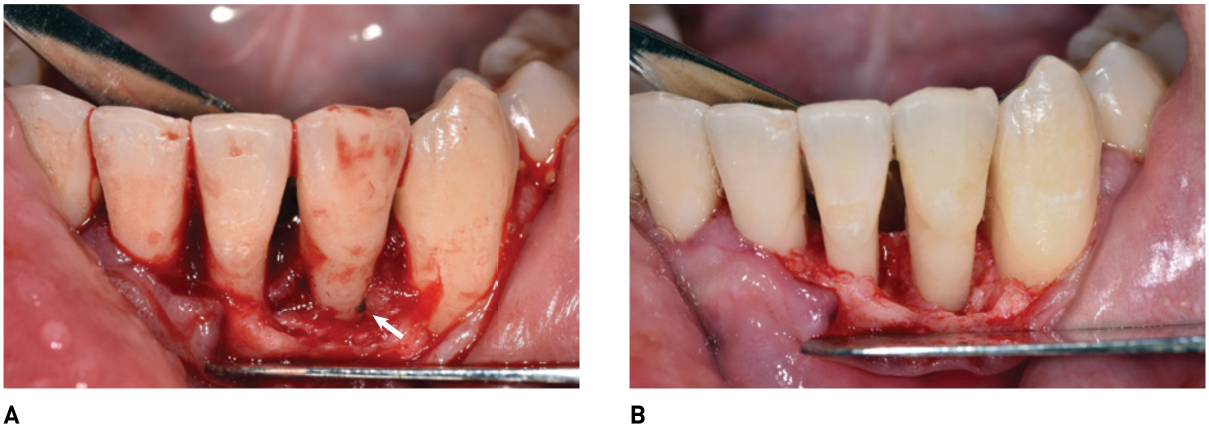

Figure 30-9 **Periodontal Flap for Access. A.** Surgical photo shows calculus (white arrow) at the distofacial line angle of the left lateral incisor and a large amount of loose granulation tissue. **B.** Surgical photo following root debridement and mechanical removal of loose granulation tissue. Note the absence of the calculus deposit and loose granulation tissue. Also, note the irregular bony contours that can now be properly recontoured with a combination of rotary instruments and hand instruments.

Box 30-2 Common Terminology Used to Classify Periodontal Flaps

- Based on alveolar bone exposure
- Based on location of flap margin
- Full-thickness flap
- Partial-thickness flap
- Nondisplaced flap
- Displaced flap

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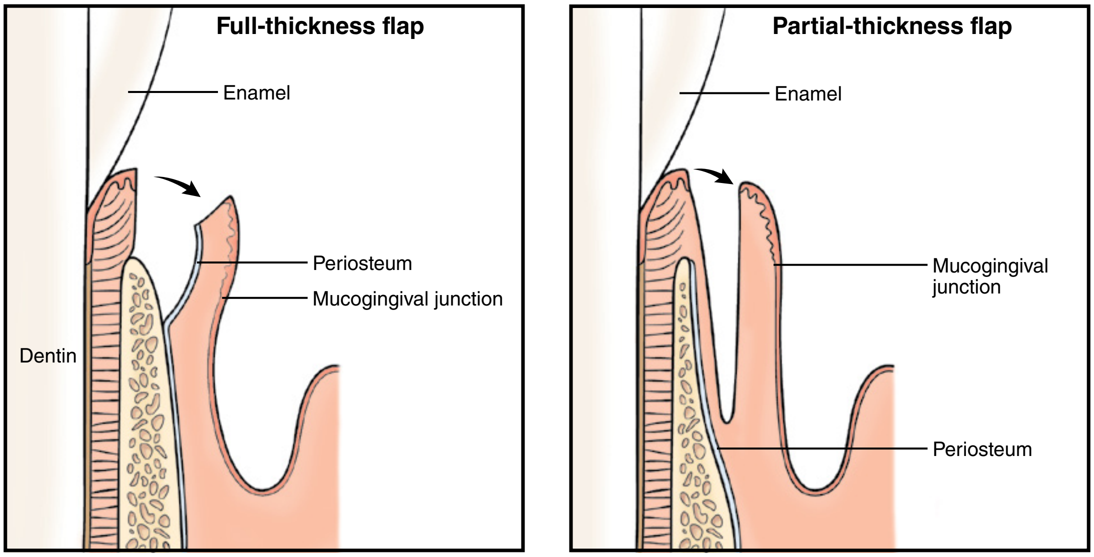
Full-thickness flap
Enamel
Periosteum
Mucogingival junction
Dentin
Partial-thickness flap
Enamel
Mucogingival junction
Periosteum

Figure 30-10 Flap Classification by Degree of Bone Exposure. The illustration on the left depicts a full-thickness flap to obtain access to the underlying alveolar bone. The illustration on the right depicts a partial-thickness flap in which the alveolar bone is not exposed.

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b. In this type of flap elevation, the flap is gently lifted or teased off by using surgical instruments called periosteal elevators (it is elevated in a manner quite similar to lifting the peeling off an orange). **Figure 30-11** illustrates a full-thickness flap elevated during a periodontal surgical procedure.

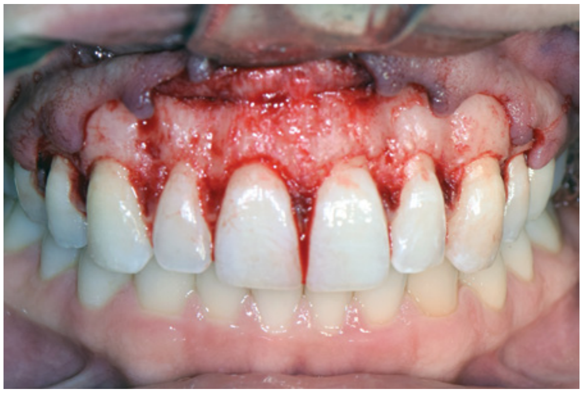

Figure 30-11 Full-Thickness Flap. This surgical photo shows a full-thickness flap (i.e., a flap of soft tissue that includes epithelium, underlying connective tissue, and the periosteum elevated off the teeth and alveolar bone). Note the complete exposure of the underlying avascular alveolar bone.

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3. A **partial-thickness flap**, or split-thickness flap, describes elevation of only the epithelium and a thin layer of the underlying connective tissue rather than the entire thickness of the underlying soft tissues (**Figure 30-12**).

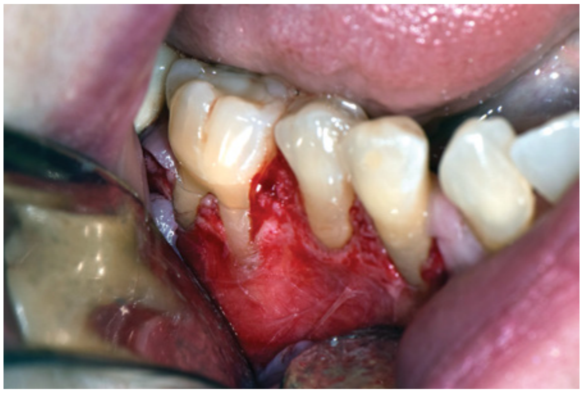

Figure 30-12 **Partial-Thickness Flap.** In a partial-thickness flap, the bone is not exposed and remains covered by the vascularized periosteum.

Courtesy of Dr. Hawra AlQallaf, Indianapolis, IN.

   a. The partial-thickness flap is elevated with sharp dissection; **sharp dissection** requires incising the underlying connective tissue in such a manner as to separate the epithelial surface plus a small portion of the connective tissue from the periosteum. Use of this technique would leave the periosteum covering the bone.
   b. To perform the sharp dissection needed in a partial-thickness flap, a surgeon uses a sharp scalpel blade to separate the flap from the underlying periosteum. The surgeon must also limit this approach to areas of gingiva that are relatively thick. If sharp dissection is attempted in areas of thin gingiva, then there is a risk of perforating (tearing) through the flap, which can lead to flap necrosis.
   c. Research indicates that when alveolar bone is exposed during a flap procedure there is a potential loss of post-surgical bone resorption following the procedure. While this does not affect final healing, the possibly of post-surgical bone resorption can make use of a full-thickness flap inadvisable in certain instances.

b. **Classification by Location of the Soft Tissue Margin.** Another method of classifying periodontal flaps is to categorize flaps based upon the location of the soft tissue margin when it is sutured back in place. Using this method of classification, flaps are classified as either nondisplaced or displaced.
   1. A **nondisplaced flap** is a flap that is sutured with the margin of the flap at its original position in relationship to its prior location before flap elevation.
   2. In contrast, a **displaced flap** is a flap that is sutured with the margin of the flap placed at a position other than its original position in relationship to its prior location before flap elevation. Note that a displaced flap can be relocated either apically, coronally, or laterally in relationship to its original position.
      a. For a displaced flap to be moved to a new position (such as apically, coronally, or laterally), the surgeon must perform the flap elevation in such a manner that the base of the flap extends into the moveable mucosal tissues. To achieve this, vertical incisions are made (discussed below).
      b. Displaced flaps are generally not possible to perform on the palatal gingiva because of the absence of movable mucosa in the posterior palatal region.
      c. Both a full-thickness flap and a partial-thickness flap can either be displaced or nondisplaced.

3. **Types of Incisions Used During Periodontal Flap Surgeries.** Most of the incisions made prior to elevation of a periodontal flap are made with surgical scalpel blades. A wide variety of different types of incisions can be made with these surgical scalpel blades. Some understanding of the terminology related to flap incisions could be useful to the dental hygienist. These incisions can be broadly classified as either horizontal or vertical incisions. **Box 30-3** provides an overview of the types of incisions utilized during periodontal flaps.

Box 30-3 Types of Incisions Utilized During Periodontal Flaps

Horizontal incisions
- Crevicular incision
- Internal bevel incision

Vertical incisions
- Vertical releasing incision

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a. **Horizontal Incisions. Horizontal incisions** are directed along the gingival margins in a mesiodistal direction.

1. One type of horizontal incision commonly employed during flap surgery is the crevicular incision. In the **crevicular incision**, the surgical scalpel blade is carefully placed into the gingival crevice (also known as the sulcus) and gently advanced until the tip of the blade contacts bone.
2. A second type of horizontal incision is the internal bevel incision.
   a. In an **internal bevel incision**, the surgical scalpel blade enters the marginal gingiva, but is not placed directly into the crevice. Instead, the scalpel blade enters the gingiva approximately 0.5 to 1.0 mm away from the margin and follows the general contour of the scalloped marginal gingiva.
   b. An internal bevel incision leaves a thin soft tissue collar around the tooth root (including the lining of the preexisting periodontal pocket); this collar of tissue is subsequently removed with hand instruments following flap reflection.
3. Terminology related to these incisions can be quite confusing.
   a. The internal bevel incision has also been referred to as a reverse bevel incision or the initial incision because it is usually made as a first step during a routine periodontal flap procedure. It is also known as extrasulcular incision or extracrevicular incision because the scalpel blade enters the gingiva outside of the sulcus.
   b. The crevicular incision has also been referred to as the second incision because it is usually made as the second step during a routine flap procedure. Another term for this type of incision is sulcular incision because the scalpel blade enters the gingiva inside the sulcus. **Figure 30-13** shows both an internal bevel incision and crevicular incision.

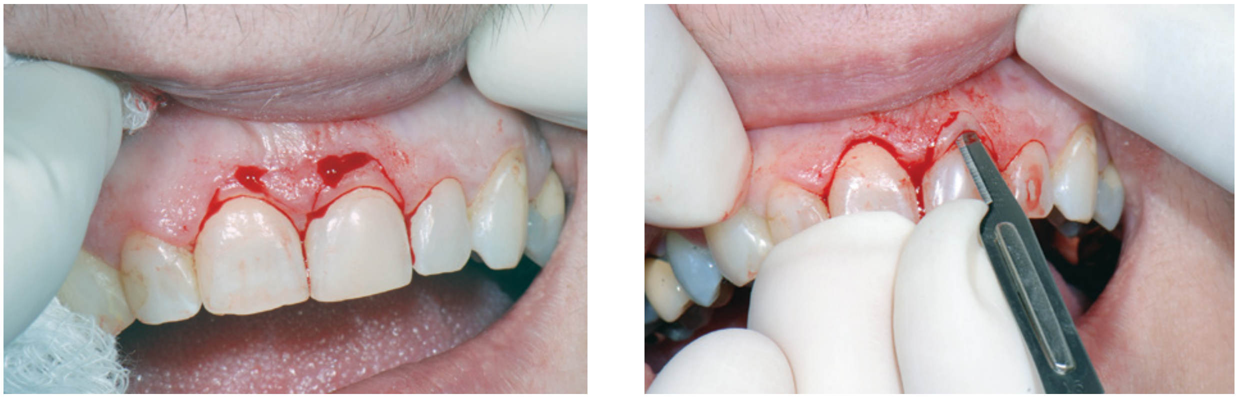

Figure 30-13 Horizontal Incisions. The photo on the left shows that the initial incision is an inverse bevel incision. Note how the inverse bevel incision is made from a distance from the marginal gingiva. The right-hand photo shows that the second incision is the crevicular incision. Note the location and direction of the scalpel blade in relation to the marginal gingiva when the crevicular incision is made.

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b. **Vertical Incisions. Vertical incisions** run perpendicular or oblique to the gingival margin in an apico-occlusal direction (**Figure 30-14**).

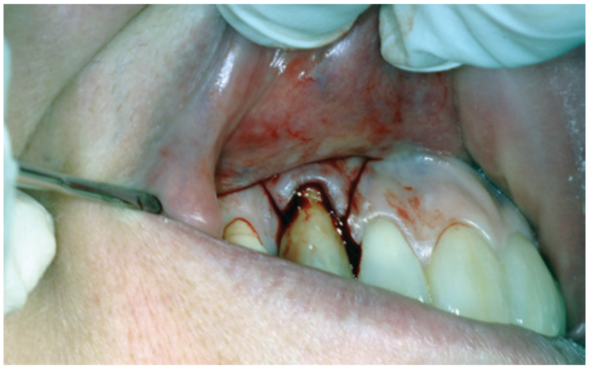

Figure 30-14 Vertical Incision. A vertical incision starts at the line angles of the tooth and extends apically into the alveolar mucosal tissues. Each vertical incision connects to one end of the horizontal incision.

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1. Vertical incisions are primarily used to allow for the elevation of the flap without stretching or damaging the soft tissues during the surgical procedure.
2. The vertical incisions also are referred to as vertical releasing incisions because once this incision extends apical to the mucogingival junction, the flap is "released" and can be displaced (moved) apically, laterally, or coronally (**Figure 30-15**).

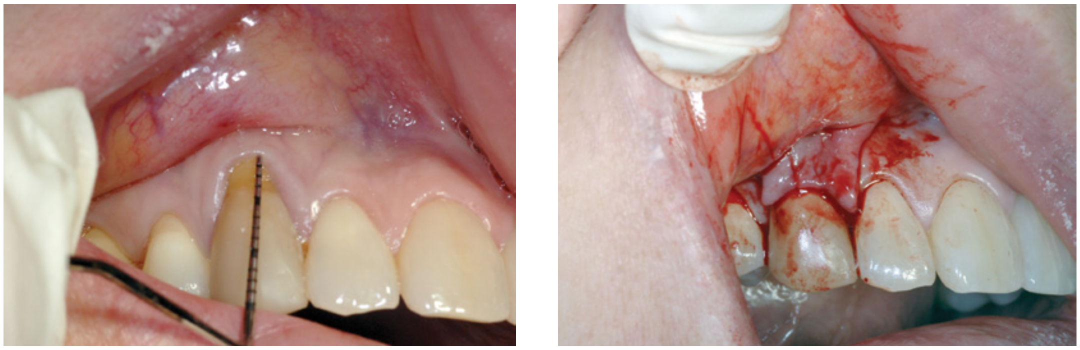

Figure 30-15 Coronal Displacement of Flap. The photo on the left is a presurgical photograph. Note the presurgical location of the gingival margin on the maxillary right canine. The photo on the right is a surgical photo following coronal displacement of the flap. Note the "new" location of the gingival margin as a result of the two vertical incisions. The vertical incisions "released" the flap, thereby allowing the clinician to coronally displace the flap and cover the recession defect.

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Section 3

Descriptions of Common Types of Periodontal Surgery

1. **Flap for Access**
    a. **Procedure Description**
        1. **Flap for access** (or modified Widman flap surgery) is used to provide access to the tooth roots for improved root preparation.[1,4–6] In this surgical procedure, both an internal bevel incision and a crevicular incision are made in the gingival tissues. The soft tissue is then gently elevated from the tooth roots. **Figure 30-16** shows a flap for access procedure showing the root anatomy and the alveolar bony architecture.

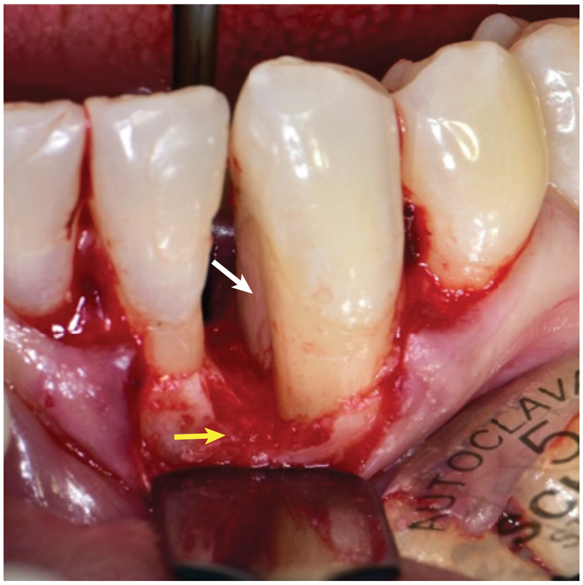

Figure 30-16 Flap for Access in Progress. This photo was taken following completion of surgical root debridement. Take special attention to the root anatomy and the osseous architecture at this stage of the surgery. Note the radicular concavity on the mesial root (white arrow) and the irregular bony architecture surrounding the canine (yellow arrow). After this photograph was taken, odontoplasty (reshaping of the tooth to remove plaque retentive areas like the root convexity) and osseous surgery were performed to correct the deficiencies.

2. **Advantages of Flap for Access**
    a. Flap for access surgery offers excellent access to the tooth roots for thorough instrumentation in sites where deep pocket depths may have hindered periodontal instrumentation during nonsurgical therapy.
    b. Flap for access surgery also leads to intimate adaptation of healthy connective tissues to the debrided tooth roots following suturing of the wound. This also facilitates healing by primary intention.
3. The tissues are elevated only enough to allow good access for periodontal instrumentation of the tooth roots. Following root treatment, the gingival tissue is replaced at its original position (i.e., a nondisplaced flap) and stabilized with sutures.

b. **Steps in a Typical Flap for Access.** The usual steps followed during flap for access surgery are outlined below.

1. An internal bevel incision is begun through the surface of the gingiva surrounding the teeth; the incision is made approximately 0.5 to 1.0 mm away from the gingival margin and follows the scallop of the marginal gingiva.
2. The internal bevel incision that was begun as the first incision through the surface gingiva is retraced and extended apically all the way to the alveolar bone. (Vertical incisions are rarely incorporated in this type of procedure because the flap will ultimately return to its original location.)
3. The flap is elevated far enough to provide good access to the tooth roots.
4. A crevicular incision is then made from the base of the pocket to the bone to facilitate removal of the small collar of tissue remaining around the necks of the teeth.
5. If needed, an incision may be made at the base of remaining tissue collar to completely free this tissue, and the tissue collar is removed with a hand curette.
6. With the flap elevated, tooth roots are instrumented to remove remaining plaque biofilm, calculus deposits, and root irregularities. While debriding, residual periodontal ligament fibers adhering to the tooth root near the base of the pocket are left undisturbed.

7. The flaps may be thinned if needed to allow for intimate adaptation of the gingiva to the necks of the teeth; alveolar bone contour is not altered unless slight recontouring is needed to allow for proper flap adaptation.
8. The flap is repositioned by suturing the flap margin to its original position (nondisplaced); special effort is made to ensure that the tips of the facial and lingual papillae are in actual contact to promote healing by primary intention.

c. **Healing After Flap for Access**

1. The type of healing expected from flap for access surgery is healing by repair and is characterized by the formation of a long junctional epithelium.
2. Research shows that flap for access surgery can result in a stable dentogingival unit that can be maintained in health with periodic periodontal maintenance by the dental team and proper self-care by the patient.

d. **Special Considerations for the Dental Hygienist**

1. During routine nonsurgical periodontal instrumentation, it may not be possible to perform thorough subgingival biofilm and calculus removal if the pocket depths are deeper than 5 mm.[7] Flap for access surgery provides greatly improved access to root surfaces in areas of deeper pockets where the effectiveness of conventional nonsurgical periodontal instrumentation alone would be limited.
2. Even in patients where flap for access surgery is part of the treatment plan, every effort should be made to minimize the inflammation associated with periodontitis by performing complete nonsurgical therapy prior to the surgical intervention.
    a. The dental hygienist plays a critical role in promoting patient understanding of how nonsurgical and surgical treatment are related—first, nonsurgical therapy followed by surgical therapy, if needed.
    b. Thorough and meticulous nonsurgical periodontal instrumentation even in areas of deep pockets can reduce the extent of any planned periodontal surgical treatment and is always an important part of patient care.

2. **Open Flap Debridement**

a. **Procedure Description**

1. **Open flap debridement** is a term that describes a surgical procedure quite similar in concept and execution to flap for access surgery.
2. Historically, the term *open flap debridement* was used to describe some of the original flap procedures that were first developed by periodontists many years ago.
3. Today, open flap debridement is usually performed with steps quite similar to flap for access with the following exceptions:
    a. **Exception 1.** Open flap debridement usually includes more extensive flap elevation than flap for access—providing access not only to the tooth roots but also to the alveolar bone defects. Remember that during flap for access surgery, the flap is elevated only far enough to provide sufficient access to the tooth roots.
    b. **Exception 2.** Whereas flap for access is a nondisplaced flap, open flap debridement may involve displacing the flap margin to a new location (i.e., during an open flap debridement, the flap margin may be sutured in a position more apical to its original position).

b. **Steps in Typical Open Flap Debridement**

1. **Figure 30-17** illustrates critical steps in a typical open flap debridement. The procedure begins with horizontal incisions that can be either crevicular or internal bevel incisions. The decision to use either crevicular incision or internal bevel incision is based on the amount of keratinized tissue. If there is a sufficient band of keratinized tissue available, both a crevicular incision and an internal bevel incision will be employed. However, if there is insufficient keratinized tissue, then only a crevicular incision will be used so as to preserve the tissue. Vertical releasing incisions can be included as needed to allow for atraumatic flap elevation.

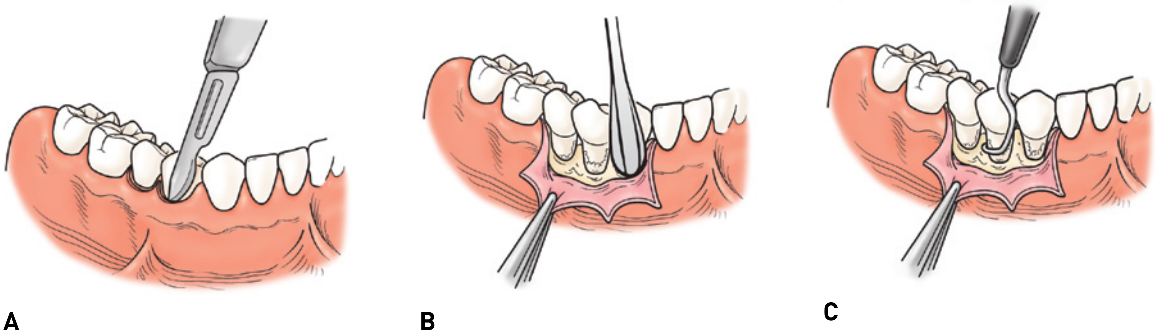

Figure 30-17 **Critical Steps During a Typical Open Flap Debridement. A.** Incisions being made to bone from within the crevice or pocket base. **B.** Flap elevation to expose tooth roots and alveolar bone. **C.** Periodontal instrumentation of the roots of the teeth.

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2. Full-thickness (mucoperiosteal) flaps are elevated to provide access both to tooth roots and to the underlying alveolar bone defects (**Figure 30-18**).

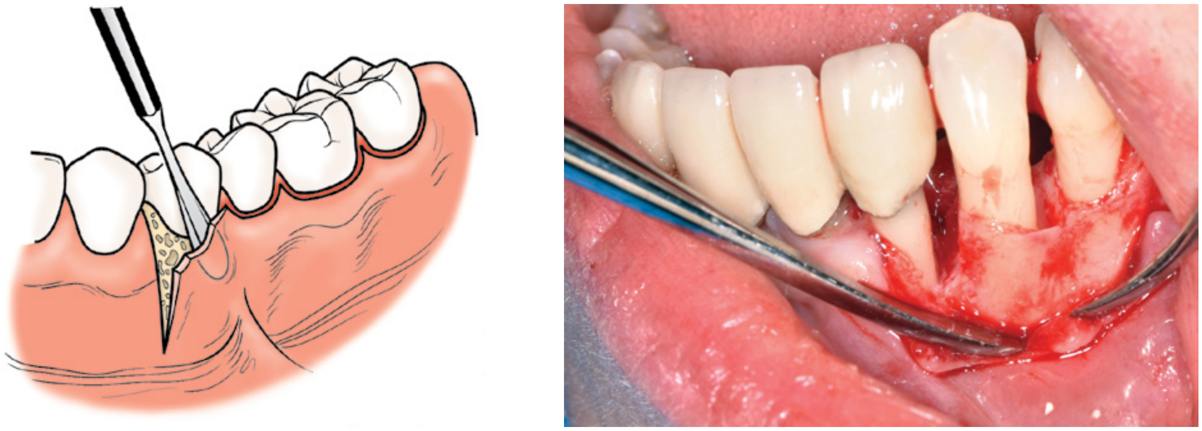

Figure 30-18 **Open Flap Debridement.** The illustration on the left shows a blunt instrument being used to elevate the overlying soft tissue away from the underlying bone. This type of flap elevation is classified as a full-thickness flap. The accompanying clinical photograph shows a blunt-end instrument (known as a periosteal elevator) being used to elevate a full-thickness flap to reveal a deep, wide osseous defect between the lateral incisor and canine. Surgical flap procedures allows for better access to both the underlying root surfaces and accompanying bony defects. Compared to Figure 30-16 (which shows a more conservative flap elevation for a Flap for Access procedure), open flap debridement involves elevating the flap past the mucogingival junction to give the clinician greater access to both the underlying tooth root and alveolar bone.

Illustration: © Wolters Kluwer

3. Granulation tissue is removed from existing osseous defects and from interdental areas (refer back to Figure 30-9A and B). Tooth roots are instrumented to remove remaining plaque biofilm, calculus deposits, and root irregularities. Osteoplasty, which is not normally included in open flap debridement, is performed only if it is needed to allow for readaptation the tissues to the tooth roots.
4. Flaps are sutured either at their original level (nondisplaced) or at a level more apical to their original position (displaced).

c. **Healing Expected After Open Flap Debridement**
   1. Healing following open flap debridement resolves much of the existing inflammation within the periodontal tissues.
   2. The formation of a long junctional epithelium can occur along with slight remodeling of some of the osseous bone defects caused by periodontitis.
   3. Periodontal regeneration is not an outcome that occurs following open flap debridement. Instead, healing following open flap debridement is characterized by periodontal repair.
   4. Although not ideal, residual periodontal pockets may still remain following this procedure—thus complicating both patient self-care and professional periodontal maintenance.

3. **Periodontal Osseous Resective Surgery.** The word *osseous* is defined as "having to do with bone." Thus, periodontal osseous resective surgery is surgery involving the alveolar bone.
   a. **Procedure Description**
      1. **Periodontal osseous resective surgery** (or periodontal osseous surgery) is a term used to describe periodontal surgery employed to correct many of the irregular deformities of the alveolar bone that often result from periodontitis. This is done by using a combination of rotary and hand instrumentation to physically reshape or resculpt the abnormal bony contours in order to recreate physiologic bony contours.[8,9]
      2. The fundamental goal for this type of periodontal surgery is to eliminate periodontal pockets, and this goal can be achieved when osseous surgery is combined with an apically displaced flap, as discussed in the next section.
   b. **Rationale for Periodontal Osseous Resective Surgery**
      1. Gingiva tends to follow its natural architecture with or without the support of underlying alveolar bone.
      2. As periodontitis progresses, the contours of alveolar bone are altered by the formation of osseous defects. Osseous defects can be classified by names such as *osseous craters* and *one-walled*, *two-walled*, *three-walled*, and *circumferential osseous defects* (**Figure 30-19**). To review the different patterns of bone loss in periodontitis, the reader can refer to Section 2 of Chapter 3. In areas where these are osseous defects, attachment loss accompanies the changes to alveolar bone contour and periodontal pockets form.

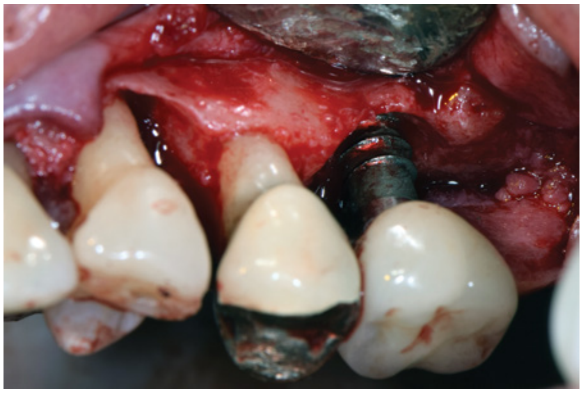

Figure 30-19 **Irregular Bone Morphology Caused by the Progression of Periodontitis/Peri-Implantitis.** After elevation of a full-thickness flap, two types of bony defects are apparent. The first bony defect is the one-wall angular defect on the distal of the maxillary first premolar; the second bony defect is the circumferential (moat-like) defect around the implant.

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3. The objectives of osseous resective surgery are twofold: (1) to reestablish physiologic (or close to physiologic) alveolar bone contours that are in harmony with the natural contours of the gingiva and (2) to recontour bone so that it resembles the bony contours undamaged by periodontitis. Thus, periodontal osseous surgery minimizes the discrepancy between the bone contour and the gingival contour to eliminate periodontal pockets following complete healing (**Figure 30-20**).

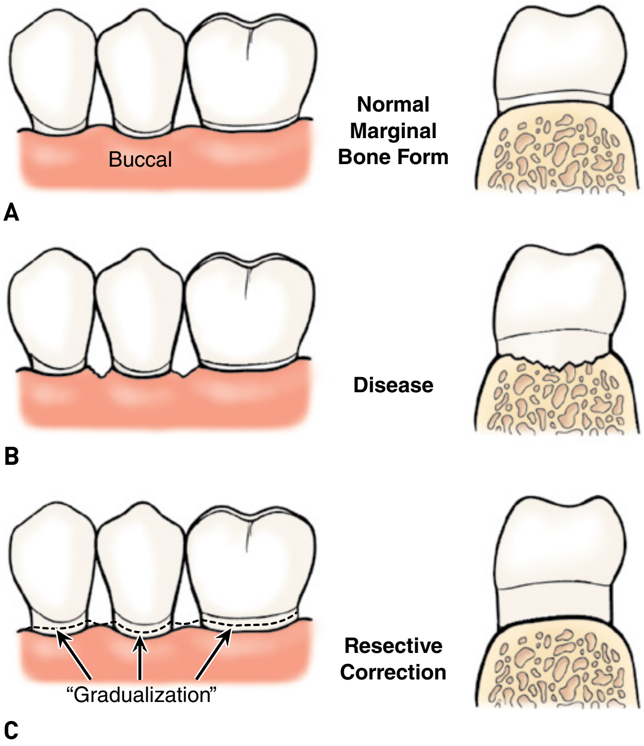

Figure 30-20 Osseous Resection. A. Ideal osseous form. The interproximal bone is consistently more coronal to the marginal bone. Note that the ideal form of the marginal bone has a gradual curved, scalloped configuration in between the interdental peaks. **B.** Diseased osseous form. Note the irregular crestal deformities in the interproximal regions and the loss of the scalloped marginal bone configuration. **C.** Resective correction. Note that osseous surgery has eliminated the irregular crestal deformities and reestablished ideal bony contours that will be in harmony with the overlying gingival tissues.

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4. Periodontal osseous resective surgery is commonly performed in patients with Stage I periodontitis or Stage II periodontitis where the bone defects created by the periodontitis are primarily osseous craters.
5. However, periodontal osseous resective surgery is contraindicated in patients with Stage III or Stage IV periodontitis. Because this is a resective procedure, the intentional removal of bone in a such advanced cases may further jeopardize the support around the tooth.

c. **Special Terminology Associated with Periodontal Osseous Resective Surgery: Ostectomy and Osteoplasty.** Two terms frequently arise during discussions of periodontal osseous resective surgery, and these terms can be a bit confusing. **Box 30-4** summarizes the key difference between *ostectomy* versus *osteoplasty*.

Box 30-4 Special Terminology Associated with Periodontal Osseous Resective Surgery

- Ostectomy—removal of tooth-supporting bone
- Osteoplasty—reshaping of the surface bone contours without removing tooth-supporting bone

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1. **Ostectomy.** **Ostectomy** (also *osteoectomy*) refers to the removal of tooth-supporting bone (also known as alveolar bone proper).
   a. Ostectomy results in the immediate loss of a small amount of attachment at certain sites, and for that reason ostectomy must be performed with caution by the surgeon.
   b. In spite of the slight attachment loss that occurs during ostectomy, osseous resective surgery is the most predictable method of eliminating periodontal pockets. The removal of small amounts of supporting bone is justified by the attainment of the physiologic form and contours of alveolar bone that is compatible with the natural contours of the overlying gingiva.
2. **Osteoplasty.** **Osteoplasty** refers to reshaping the surface of alveolar bone without actually removing any of the tooth-supporting bone.
3. In reality, most periodontal osseous resective surgeries involve a combination of both ostectomy and osteoplasty. When performed with precision, these procedures can result in alveolar bone contours that mimic the contours of the gingiva following complete healing. **Figure 30-21** shows a case where a combination of osteotomy and osteoplasty procedures were implemented simultaneously to treat a patient's periodontitis.

d. **Steps in Periodontal Osseous Resective Surgery**
   1. Incisions are made, and flaps are elevated to provide access to the osseous defects and to the surrounding alveolar bone; these incisions typically are done on both the facial and lingual surfaces of the teeth and typically include both horizontal and vertical releasing incisions.
   2. Granulation tissue associated with the osseous defects is thoroughly debrided to allow full visualization to the extent and shape of the osseous defects.
   3. All remaining soft tissue tags in the surgical site are identified and removed usually using a combination of hand and ultrasonic instrumentation.
   4. Tooth root surfaces are debrided to remove all plaque biofilm, calculus, and root irregularities.
   5. Osteoplasty is performed to remove thick bone ledges where they exist on the facial and lingual surfaces of the alveolar bone. A combination of hand instruments and surgical burs can be used when doing the osteoplasty.
   6. Ostectomy is performed to eliminate interproximal osseous defects. Then, the ostectomy is extended one tooth mesial and one tooth distal to the operating site with the intent of resecting the bone so that it blends naturally with the neighboring teeth. A combination of hand instruments and surgical burs can be used when doing the ostectomy.
   7. The gingiva is sutured into place (usually at a more apical position than the original level as discussed in the next section).

e. **Healing After Periodontal Osseous Resective Surgery**
   1. When periodontal osseous resective surgery is performed in areas of the dentition where osseous craters exist, it is possible for the surgeon to recreate a natural contour to the alveolar bone.
   2. When this osseous resective surgery is combined with the apically positioned flap (as discussed in the next section of this chapter), it is possible to reestablish a normal crevice or sulcus depth without the presence of residual periodontal pockets following the surgery.
   3. For most patients who are treated with this type of surgical procedure, it is possible to maintain a healthy periodontium provided that the patient performs reasonable self-care and complies with their periodontal maintenance regimen.

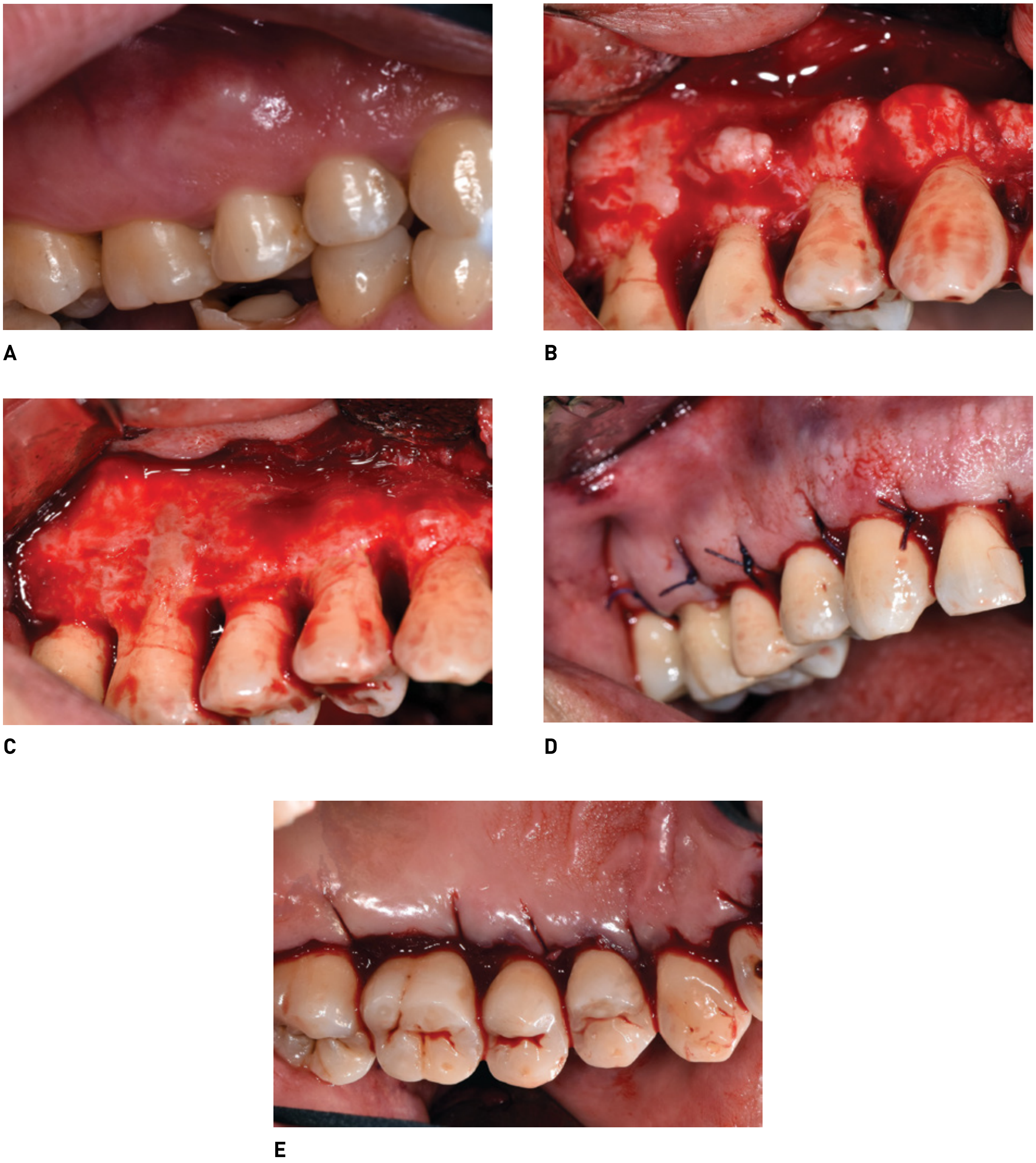
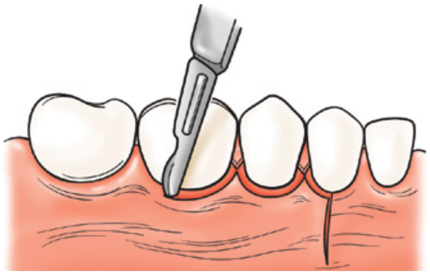
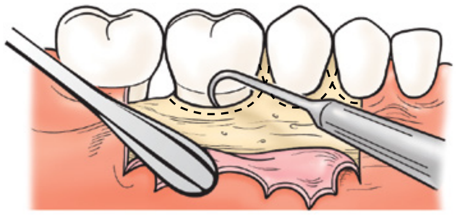
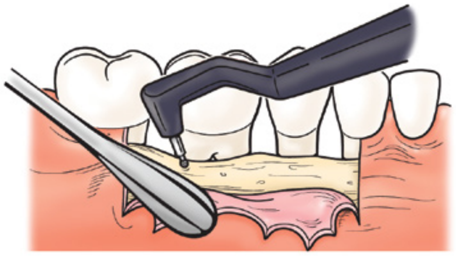
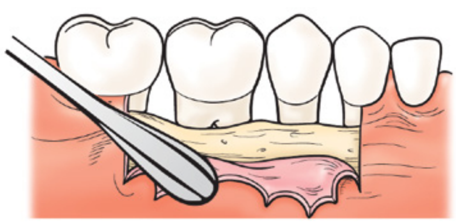
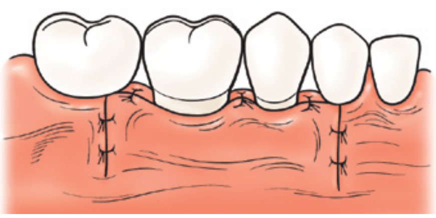
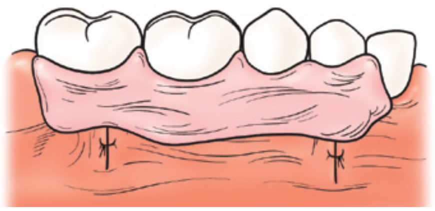


Figure 30-21 **Combined Ostectomy/Osteoplasty Procedure.** 43-year-old patient presented with pathologic pockets (from 5-7 mm) and bleeding upon probing in the maxillary right posterior sextant. Previous nonsurgical periodontal therapy was not successful in arresting the inflammation. Photographs obtained at surgical phase of treatment. **A.** Presurgical photo. Observe the blunted papilla and thickened gingival margin. **B**. Full-thickness flap reflection. Note the bony exostoses and irregular crestal bony architecture caused by periodontitis. **C**. Next, a combination of a surgical burs and hand instruments is used to blend the crestal bone to achieve a scalloped physiologic architecture. Additionally, the same instruments are used to remove the bony exostoses. **D.** Buccal suturing photo. Note the intimate adaptation of the soft tissue to the necks of the teeth. **E.** Palatal suturing photo. Note the intimate adaptation of the soft tissue to the necks of the teeth.

4. **Apically Positioned (or Displaced) Flap with Osseous Resective Surgery**
   a. **Procedure Description**
      1. An **apically positioned flap with osseous resective surgery** is a periodontal surgical procedure involving a combination of a displaced flap (displaced in an apical direction) plus resective osseous surgery.
         a. As already discussed, correction of bony defects to mimic the contours of healthy alveolar bone is usually referred to as periodontal osseous resective surgery.
         b. Following contouring of the alveolar bone, the flap is sutured at a position that is apical to its original position in relation to the CEJs.
      2. This surgical procedure is ideal for minimizing periodontal pocket depths in patients with osseous craters caused by Stage I or Stage II periodontitis.
         a. An apically displaced flap can result in a gingival margin that is apical to the CEJ of the tooth. This new position of the gingival margin means that more of the root is exposed.
         b. The reduced pocket depth is easier to maintain by both the patient and the oral health care provider.
   b. **Steps in an Apically Positioned Flap with Osseous Resective Surgery.** **Table 30-3** and **Figures 30-22A-F** illustrate the critical steps in an apically positioned flap with osseous resective surgery.

Table 30-3 **Critical Steps in an Apically Positioned Flap with Osseous Resective Surgery**

 **Figure 30-22A** © Wolters Kluwer	Step 1: Internal bevel incision and vertical releasing incision being made around the teeth.
 **Figure 30-22B** © Wolters Kluwer	Step 2: Removal of the collar of soft tissue following flap elevation.
 **Figure 30-22C** © Wolters Kluwer	Step 3: Ostectomy being performed after identification of osseous defects.

 **Figure 30-22D** © Wolters Kluwer	Step 4: Inspection of the final bone contours after ostectomy and osteoplasty.
 **Figure 30-22E** © Wolters Kluwer	Step 5: Suturing of both the flap margins and the vertical releasing incisions. Note that the level of the flap margin is displaced in an apical position compared to its original position.
 **Figure 30-22F** © Wolters Kluwer	Step 6: Placement of periodontal dressing to stabilize the flap at its new position during the early phase of healing.

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1. This procedure normally begins with an internal bevel incision. The internal bevel incision can preserve the width of keratinized tissue that is important to the overall procedure because this width of keratinized tissue will be displaced apically as a final step.
2. The internal bevel incision is followed by flap elevation and crevicular incision prior to removal of the collar of tissues around the necks of the teeth.
3. Vertical releasing incisions are made as needed to avoid damage to the flap.
4. Granulation tissues are debrided, and osseous defects are exposed, as discussed in the previous section.
5. Periodontal osseous resective surgery is performed to mimic the contours of healthy alveolar bone; this usually involves both ostectomy and osteoplasty procedures.
6. The flap is sutured at a position apical to its original position (usually near the tooth–bone junction). The surgical site is covered with periodontal dressing to stabilize the flap at its apical location.

c. **Healing of an Apically Positioned Flap with Osseous Resective Surgery**

1. Final healing of this type of surgery results in a normal attachment (both junctional epithelium and connective tissue attachment) at a position more apical on the tooth root.
2. It should be emphasized that apically positioned flap with osseous resection cannot eliminate all periodontal osseous defects, especially where the defects are quite severe.
3. Research has shown that an apically positioned flap combined with periodontal osseous surgery can result in a stable dentogingival junction that can be maintained in health with reasonable self-care by the patient and periodic periodontal maintenance by the dental team.
4. **Figure 30-23** illustrates the results of a healed apically positioned flap used to treat a furcation involvement on a molar tooth.

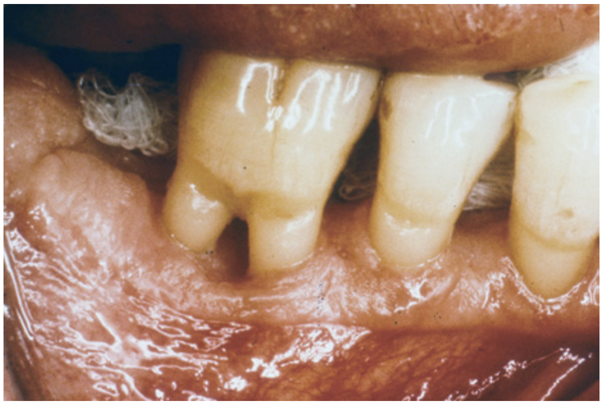

Figure 30-23 Results of an Apically Positioned Flap. This apically positioned flap was performed to treat a large through-and-through furcation invasion on the molar tooth. The intent of this surgical procedure was to relocate the position of the gingival margin apical to the CEJ and the furcation entrance. This is known as furcation tunneling. The objective of furcation tunneling is to create an "open tunnel" through the furcation that is more accessible and cleansable for a patient using an interdental brush.

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d. **Special Considerations for the Dental Hygienist**

1. During surgery to minimize periodontal pockets, it is common for the final location of the gingival margin to be positioned at a more apical level to the CEJ than it originally occupied.
    a. This apical positioning of the gingival margin results in exposure of a portion of the root to the oral cavity.
    b. Visibility of a portion of the root may be an esthetic concern for the patient. Hence, this type of surgery may be contraindicated in the easily visible esthetic zones, such as the maxillary anterior region in a patient with a high smile line or hypermobile lip.
    c. In a patient with a high caries risk, exposure of root surface in the oral cavity may predispose the tooth to developing root caries. Therefore, this type of surgery may be contraindicated in patients with a high risk for dental caries.
2. Temporary dentinal hypersensitivity is a frequent patient postsurgical complaint following this type of surgery. Dentinal hypersensitivity may diminish over time if the patient maintains good biofilm control (see Section 2 of Chapter 25).
3. Before surgery, the members of the dental team should inform the patient about anticipated changes in appearance and about the potential for dentinal hypersensitivity. The dental hygienist should also assure the patient that if sensitivity does occur, measures could be taken to minimize the sensitivity. Measures to address dentinal hypersensitivity is also discussed in Section 2 of Chapter 25.

5. **Bone Replacement Grafts**

a. **Procedure Description**

1. **Bone replacement graft** is a broad class of implantable materials that are used in medicine and dentistry to stimulate the body's own bony forming cells to augment and/or regenerate tissue that has been lost as a result of disease, trauma, or injury.
2. Bone grafting has been commonplace in medicine for many years, but transplanting bone replacement graft materials into the periodontium offers some unique challenges that differ from bone grafting in medicine.
    a. Bone grafts placed into periodontal defects are subject to constant contamination from bacteria and saliva traveling along tooth roots adjacent to the graft site; the possibility for bacteria contamination would not be the case in many bone grafts in medicine (such as a bone graft done during a hip replacement).
    b. In addition, the healing of bone grafts in periodontal defects can be disrupted by the apical downgrowth of the epithelium into the wound that can lead to graft failure; this potential disruption by the growth of epithelium would also not be the case in most medical bone graft surgical procedures.

b. **Terminology Associated with Bone Replacement Grafts**
   1. Bone replacement graft materials support or promote bone healing through an osteogenic, osteoinductive, and/or osteoconductive processes.
      a. **Osteogenesis** is the term used to describe the formation of new bone by viable, living cells that are contained in the graft material.[10] Examples of bone replacement graft materials that have osteogenic potential are bone harvested from the patient's own tuberosity or ramus.
      b. **Osteoinduction** refers to a chemical process whereby complex, biologically active signaling molecules contained in the graft material—known as bone morphogenetic proteins (BMPs)—actively recruit and attract host bone-forming cells (osteoblasts) to the site of implantation to form new bone.
      c. **Osteoconduction** is a physical, passive process whereby the graft material itself acts as a scaffold for host bone-forming cells (existing outside the graft) to migrate, attach, and grow on.
   2. Using this terminology, the ideal bone replacement graft material would be one that has osteogenic, osteoinductive, and osteoconductive potentials.

c. **Broad Categories of Materials Used for Bone Replacement Grafting.** In general, bone grafting materials fall into one of four broad categories: autografts, allografts, xenografts, and alloplasts. These four categories of bone graft material have widely varying degrees of success in regenerating lost periodontal tissues. **Box 30-5** provides an overview of materials used for bone replacement grafts.

Box 30-5 Materials Used for Bone Replacement Grafts

- Autograft—bone harvested from patient's own body
- Allograft—bone harvested from another human
- Xenograft—bone harvested from another species
- Alloplast—synthetic (human-made or manufactured) bone-like material

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   1. **Autografts** are bone replacement grafts taken from the patient who is receiving the graft. Autografts can be taken from intraoral sites, such as the ramus or tuberosity, or occasionally from extraoral areas of the patient's body, such as the hip.
   2. **Allografts** are bone replacement grafts taken from individuals who are genetically dissimilar to the donor (i.e., another human). An allograft may be either obtained from a living donor or from a cadaveric donor. Because of the source, allografts must undergo a rigorous screening and sterilization process to eliminate the potential for rejection and/or disease transmission.
   3. **Xenografts** are bone replacement grafts taken from another species, such as bovine (cow) or porcine (pig) bone, that can be placed in a human. Like allografts, xenografts must also undergo a rigorous screening and sterilization process to eliminate the potential for rejection and/or disease transmission.
   4. **Alloplasts** are bone replacement grafts that are synthetic materials or inert foreign materials. Unlike autografts, allografts, and xenografts, an alloplast is unique because it is a nonbone graft material. Examples of alloplasts include, but are not limited to, sclera, dura, plaster of paris, coral-derived materials, and ceramics.
   5. Autografts have the highest osteogenic potential, and alloplasts have the least osteogenic potential. Allografts and xenografts have an osteogenic potential that is in between those two extremes.
   6. Although autografts have the highest osteogenic potential, it is not always possible to procure enough autogenous bone from a patient to fill the osseous defect that is being grafted. This creates the need to procure bone graft material from a source other than the patient being treated.
   7. In some instances, grafting materials can be used in combinations, such as mixing autogenous bone with an allograft material to obtain the needed volume of grafting material for a particular grafting site.

d. **Examples of Specific Materials Used for Bone Replacement Grafting** Numerous materials have been studied for use for bone replacement grafting over the past several decades. None of the materials is ideal, but many of them have been shown to have some osteogenic potential. The discussion below describes common types of bone replacement graft materials used in periodontal surgery.
   1. **Autogenous Bone Grafts (Autografts) from Intraoral Sites**
      a. As already discussed, autografts are graft materials obtained from the patient's own body.

   b. Autografts from intraoral sites have been used in periodontics and oral surgery for many years, and currently these autogenous grafts are considered the gold standard compared to other grafting materials due to their high osteogenic potential.
   c. It should not be surprising that autogenous bone is the most effective grafting material, because it already contains living bone cells and viable bone growth factors from the patient—in contrast to alloplasts, which have neither living viable cells nor growth factors.
   d. Intraoral sources for the autograft material can be harvested from bone removed during ostectomy or osteoplasty, from exostoses removed during surgery, from bone removed from edentulous ridges, from bone taken from healing extraction sites, from bone taken from the chin, and from bone harvested from the jaws distal to the most terminal tooth in a dental arch.
   e. A variety of techniques for harvesting the graft material and preserving its osteogenic potential have been advocated. These techniques usually involve exposing the alveolar bone by elevating a periodontal flap, removing granulation tissue associated with an osseous defect, treating the tooth root adjacent to the defect, placing the graft material into the defect, and closing the flap by suturing it to its original level on the teeth.
   f. In addition to harvesting particles or larger pieces of bone, autogenous bone grafts include the use of materials such as osseous coagulum. Osseous coagulum is a mixture of bone dust and blood taken from the patient and mixed with blood from the surgical site.
   g. One technique for collecting autogenous graft material from a patient during a periodontal surgical procedure involves the bone blend technique. This technique involves collecting bone in a plastic capsule and pestle and triturating the material into a workable mass of plastic-like bone graft that can be placed in an osseous defect.
   h. Although small particles or pieces of cortical bone are usually selected as autogenous grafting material, cancellous bone marrow may also be used. One common intraoral site to harvest bone marrow is from a maxillary tuberosity.
   i. One disadvantage to using autogenous bone grafts is that they frequently require a second surgical site for harvesting the graft material, increasing the potential for postsurgical problems. Another disadvantage is that it may be difficult to procure adequate amounts of this type of graft material to treat large periodontal defects.
2. **Autogenous Bone from Extraoral Sites**
   a. Iliac (or hip) cancellous marrow has been studied as an autogenous bone replacement graft material.
   b. When used in periodontal defects, this material results in bone formation in osseous defects. Today, marrow from the hip is rarely used for autogenous grafting because of the potential for root resorption adjacent to the grafting site, the potential for postoperative problems associated with the donor site, the difficulty in surgically obtaining this type of donor material, and the difficulty in procuring adequate amounts of this material for large defects.
3. **Freeze-Dried Bone Allografts**
   a. Bone allografts (harvested from another human and processed) are attractive surgical options as bone replacement graft materials. There has been a lot of interest in periodontics in identifying an ideal bone allograft. If the ideal bone allograft could be identified for use in periodontal patients, there would be no need for a second surgical site. In addition, there would be no concerns about the limited availability of the amount of graft material needed to treat large osseous defects.
   b. As already discussed, allografts are graft materials that are either harvested from another living individual of the same species or from another human who is recently deceased.
   c. Bone allografts (both mineralized and demineralized) have been used successfully as bone replacement allografts, and bone allograft products have been available commercially for some time.[11–17]
      1. Bone allograft materials are obtained from the cortical bone of a donor within a few hours of death, defatted, washed in alcohol, frozen, and vacuum sealed into sterile vials until used in a clinical setting. This type of mineralized bone allograft is known as freeze-dried bone allograft (FDBA).
      2. A second type of bone allograft is known as demineralized (or decalcified) freeze-dried bone allograft (DFDBA). This graft material is processed along the same lines as FDBA; however, the outer cortical layer of DFDBA is decalcified so as to expose the underlying bone morphogenetic proteins (BMP) and improve its osteogenic potential.

- d. Because allografts are materials that are foreign to the body of the patient receiving the graft, and because potential donors may have diseases that could be transmitted to the patient being treated, steps must be taken to maximize the safety of this type of grafting material. These steps usually include:
    1. Screening out potential donors who are members of high-risk disease groups.
    2. Examining donated grafts to exclude donors with infection or malignant disease.
    3. Treating the allograft with chemical agents or with other techniques to inactivate viruses.
- e. Although the risk of disease transmission using allografts is not zero, the risk has been reported to be highly remote.

4. **Bovine-Derived Bone**
    - a. Bovine-derived bone is an example of a xenograft material; xenografts are materials taken from another species.
    - b. Xenografts, such as bovine-derived bone, have been used as bone replacement grafts in periodontal defects.[18]
    - c. An anorganic bovine bone has been tested and marketed, and studies have shown successful regrowth of some bone with this material.
    - d. Anorganic bovine bone is cow bone that has been treated to remove all of its organic components to eliminate the risk of rejection.
        1. Removing the organic components from bovine bone leaves a porous structure, similar in structure to human bone.
        2. It has been suggested that the porous structure of anorganic bovine bone can act as scaffolding for new bone. As such, anorganic bovine bone acts as an osteoconductive material.
5. **Plaster of Paris**
    - a. Plaster of paris has been used as an alloplastic bone replacement grafting material.[19] When placed in a periodontal defect, plaster of paris acts as a scaffold, and only has an osteoconductive potential.
    - b. Plaster of paris is actually calcium sulfate, which is porous and biocompatible when placed in periodontal wounds; when calcium sulfate is placed in a periodontal wound, it resorbs within a few weeks.
    - c. Although this material has been studied and used in humans as a bone replacement graft material, its efficacy related to osteogenic potential has yet to be proven.
6. **Bioactive Glass**
    - a. Bioactive glass ceramics have been studied and used as alloplastic bone replacement grafting materials.[20–22]
    - b. This ceramic consists of calcium salts, phosphates, and silicone dioxide. When used as an alloplastic bone replacement graft, bioactive glass is used in particulate form.
    - c. When bioactive glass is placed within the periodontal tissues, the particulate surfaces can bind to host-derived proteins that ultimately attract osteoblasts to the defect.
7. **Calcium Phosphate**
    - a. Calcium phosphate biomaterials have been used as alloplastic grafting materials for several decades. Calcium phosphate is osteoconductive and is well tolerated by body tissues.
    - b. Two types of calcium phosphate materials have been used: hydroxyapatite and tricalcium phosphate.
    - c. Although these materials can result in some clinical repair of periodontal defects, they are either poorly resorbable (tricalcium phosphate) or not resorbable at all (hydroxyapatite), and can remain encapsulated within the periodontal tissues.

e. **Healing After Bone Replacement Grafting**

1. Final healing expected from bone replacement grafting usually includes a partial rebuilding of alveolar bone lost because of periodontitis.
    - a. It is not known, however, if successful bone grafting always results in the complete reformation of cementum and periodontal ligament in addition to the alveolar bone.
    - b. In spite of what appears to be good radiographic and clinical healing, it is difficult to ascertain if the regenerated bone is actually attached to the cementum by functionally oriented periodontal ligament fibers.
    - c. The only way to conclusively prove that periodontal regeneration has occurred is through histological assessment of the defect after the healing phase.

2. Research has shown that a successful bone graft combined with reasonable self-care by the patient and periodic periodontal maintenance by the dental team can result in a long-term successful outcome.

f. **Special Considerations for the Dental Hygienist**

1. The site of a bone replacement graft should be left undisturbed for several months and should not be probed until an appropriate interval has elapsed. The dental hygienist should consult with the dentist to determine when a grafted site may be probed safely.
2. Meticulous plaque control in any grafted site is critical. In the early stages of the healing, the dental team maintains some of the responsibility for biofilm control at the site, because the patient may either be temporarily unable to perform adequate self-care measures or the patient may have been instructed by the dentist to temporarily refrain from performing self-care measures during the initial 1 to 2 weeks after surgery to avoid disturbing the grafted site.

6. **Guided Tissue Regeneration**

a. **Procedure Description**

1. **Guided tissue regeneration (GTR)** is a periodontal surgical procedure intended to stimulate regeneration of lost periodontal structures (i.e., to regrow lost cementum, lost periodontal ligament, and lost alveolar bone) by "guiding" the movement of progenitor cells toward the treated root surface while excluding the migration of the gingival epithelial cells and fibroblasts. The fundamental tenet of GTR is based on classic regeneration studies that demonstrated that only cells from the periodontal ligament have the potential to induce periodontal regeneration.[23,24] All other cells—gingival epithelial cells, gingival connective tissue cells, and osteoblasts—do not have this potential.
   a. When a periodontal surgical wound is created, such as the elevation of a flap, the healing of the wound may involve cells from multiple sources surrounding the wound.
   b. **Figure 30-24** illustrates the potential sources of cells that could contribute to healing tissues within a periodontal surgical wound. They are, namely, gingival epithelial cells, gingival connective tissue cells, bone-forming cells (osteoblasts), and cells from the periodontal ligament space.

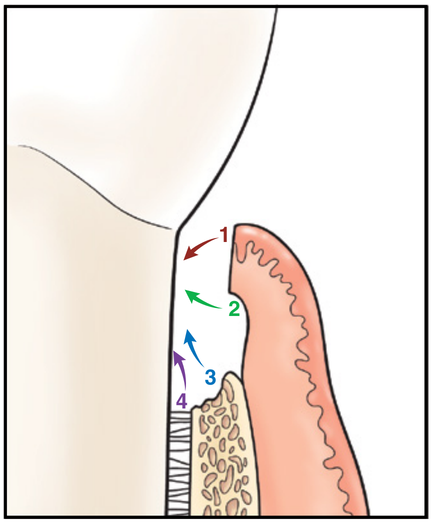

Figure 30-24 Potential Sources of Cells in Healing a Periodontal Surgical Wound. Four potential sources of cells can contribute to the healing of a periodontal surgical wound: (1) gingival epithelial cells, (2) gingival connective tissue cells, (3) osteoblasts, and (4) periodontal ligament cells. Of these four types of cells, gingival epithelial cells proliferate into the healing periodontal wound the fastest. If their downward migration is unimpeded, the gingival epithelial cells will be the first cell type to populate the treated root surface. When this occurs, a long junctional epithelial attachment will adhere to the root and interfere with periodontal regeneration.

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c. GTR techniques involve the use of a physical barrier membrane to impede the normally rapid downgrowth of gingival epithelial cells into a healing periodontal wound; the rapid growth of epithelium into the wound can interfere with the slower growth of other cells critical to the regenerative process.

d. **Figure 30-25** illustrates how a barrier membrane is used to physically impede the downgrowth of unwanted types of cells into the healing periodontal wound and create an environment that favors repopulation of the area by cells from the periodontal ligament space.

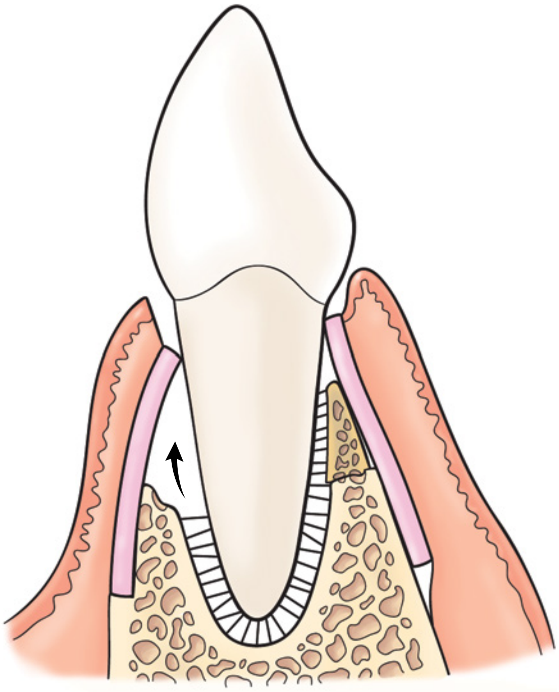

Figure 30-25 Use of Barrier Membrane to Inhibit the Rapid Growth of the Gingival Epithelial Cells. The barrier membrane has been placed under the flap margin to block the downgrowth of the gingival epithelial cells. Consequently, the undifferentiated cells from the periodontal ligament have a chance to proliferate into the healing periodontal wound, attach to the decontaminated root surface, and contribute to the formation of new cementum, new functionally oriented periodontal ligament fibers, and new bone.

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e. Impeding the downgrowth of gingival epithelial cells into a healing periodontal wound will theoretically allow for undifferentiated cells from the periodontal ligament (PDL) to populate the root area and differentiate into the tissues that normally comprise the periodontium (i.e., cementum, PDL, and alveolar bone). At the same time, the barrier membrane will exclude cells from the epithelium and connective tissue from populating the root surface during the regenerating phase.

f. A GTR procedure involves the combined use of a barrier membrane and bone graft materials. (**Figure 30-26**).

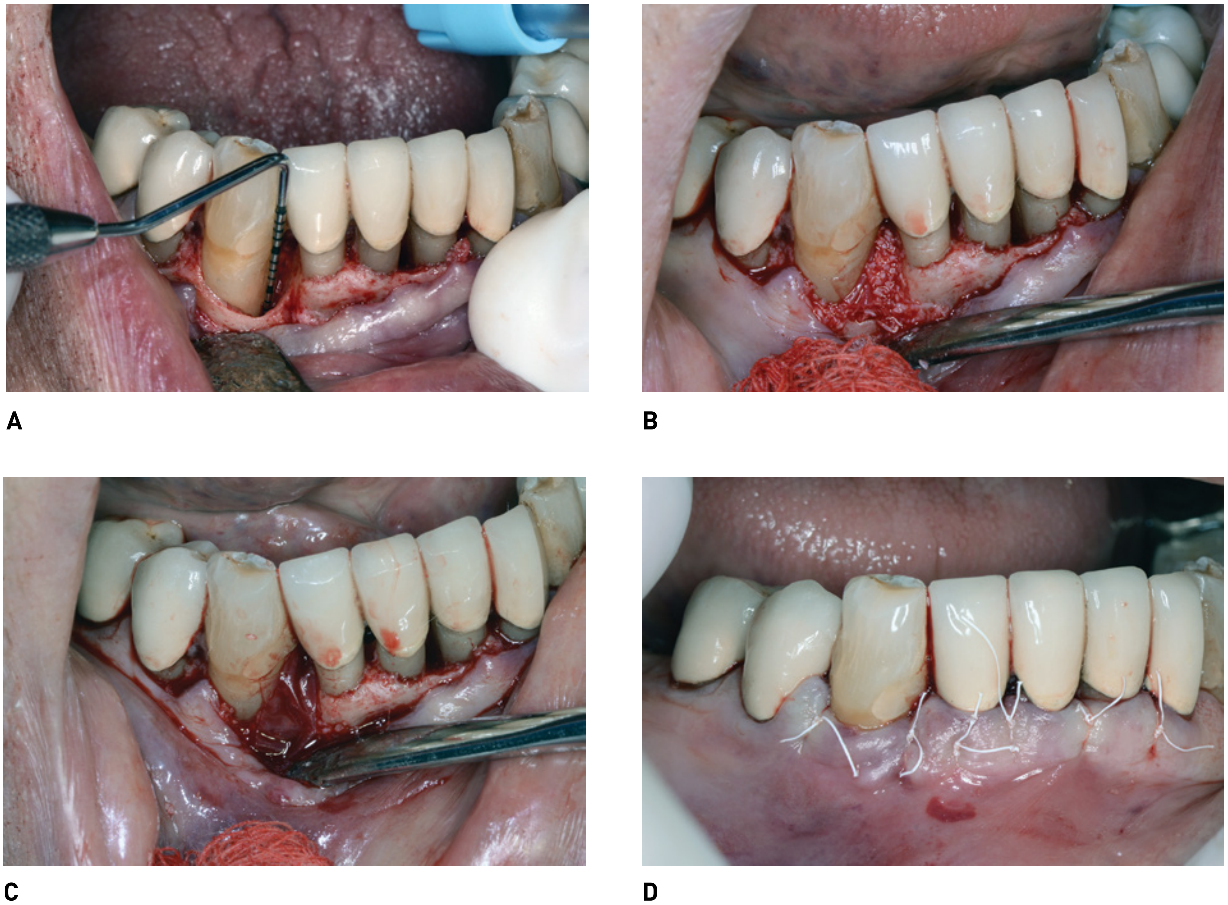

Figure 30-26 Guided Tissue Regeneration on Mandibular Right Canine. A. Note the deep angular defect on the mesial aspect of the right canine. **B.** Following thorough root debridement and degranulation of the osseous defect, an allograft was used to fill the osseous defect. **C.** A resorbable barrier membrane was then placed over the bone graft to exclude epithelial migration into the wound. **D.** Flap was coronally positioned to ensure complete closure of the grafted site.

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    g. As the name implies, GTR facilitates true regeneration of the periodontium. **Figure 30-27** shows the regenerative outcome of GTR therapy.

b. **Goal of GTR**
   1. When the entire array of types of periodontal surgery is viewed, periodontal regeneration is the ultimate goal.
   2. Although regeneration of the cementum, periodontal ligament, and alveolar bone is the ultimate goal of periodontal therapy, regeneration of the periodontium is not completely predictable with techniques in use today.

c. **Steps in a Typical Guided Tissue Regeneration Procedure**
   1. The first step in GTR is to make appropriate incisions and elevate a full-thickness flap. In this procedure, the flap usually is elevated one to two teeth beyond the site of the osseous defects.
   2. The osseous defects are thoroughly debrided and the roots in the site are instrumented with a combination of ultrasonic instruments and hand instruments.
   3. The selected membrane is trimmed to the size needed for the site; during this membrane trimming, the membrane is allowed to extend several millimeters beyond the defect in all directions.
   4. The membrane is sutured into place with a sling suture placed around the tooth.
   5. The flap also is sutured into place (frequently slightly coronally) so that the flap covers the membrane completely.

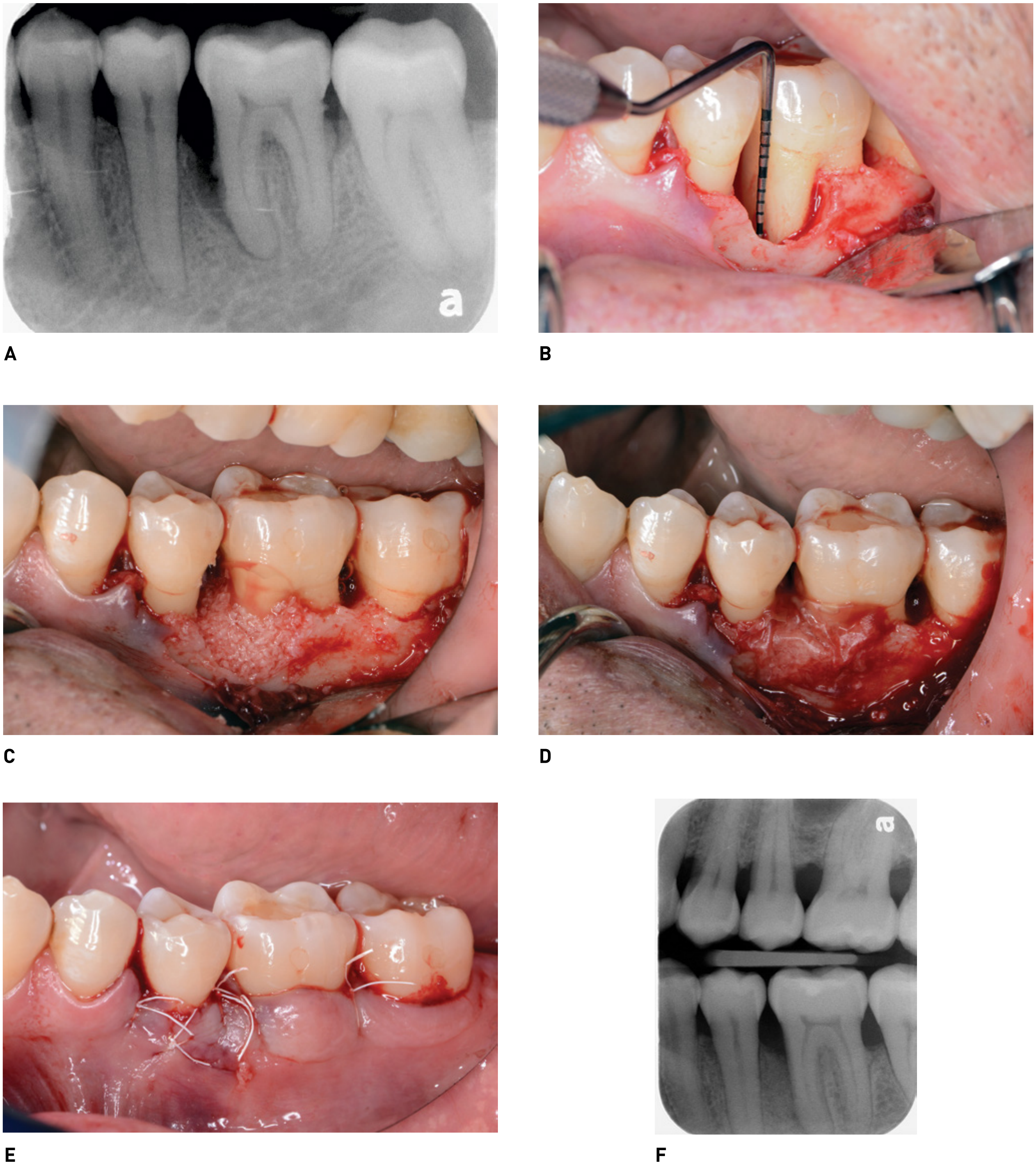
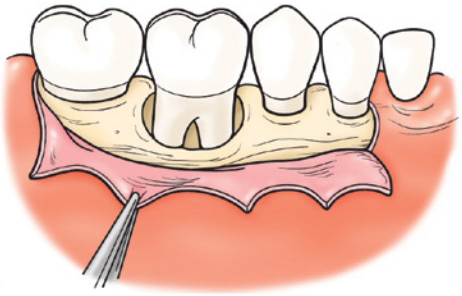
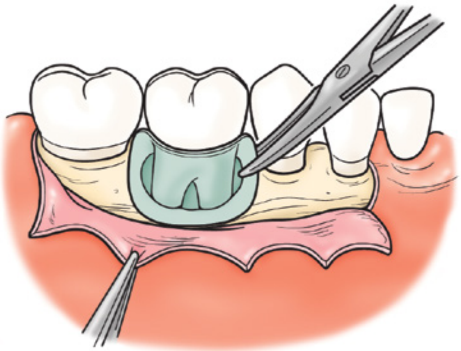
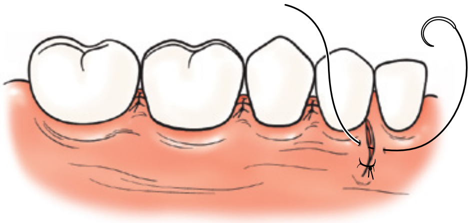


Figure 30-27 **Guided Tissue Regeneration. A.** Presurgical radiograph reveals a deep angular defect along the mesial root of the first molar and a furcation invasion affecting the same tooth (note the loss of radiodensity in the furcal region of the aforementioned tooth). Additionally, heavy clusters of calculus along the mesial root surface and distal root surface are radiographically apparent. **B.** Surgical photo following root debridement reveals the extent of the angular defect and confirms the presence of a furcation invasion. **C.** Both angular and furcal defects filled with particulate allograft. **D.** Barrier membrane placed over the defects. **E.** Flaps sutured. **F.** Six-month postsurgical radiograph. Note the bone fill in both the angular defect and the furcation defect.

6. **Table 30-4** and **Figures 30-28A-C** illustrate the steps to using a barrier membrane during a GTR procedure for treatment of furcation involvement in a molar tooth.

Table 30-4 Steps to Using a Barrier Membrane During Guided Tissue Regeneration in the Treatment of a Molar Tooth with Deep Furcation Involvement

 **Figure 30-28A** © Wolters Kluwer	Step 1: Flap is incised and elevated prior to debridement of the osseous defect and the tooth root.
 **Figure 30-28B** © Wolters Kluwer	Step 2: A barrier is selected, custom trimmed to size, and sutured into place.
 **Figure 30-28C** © Wolters Kluwer	Step 3: Flap is sutured into place completely covering the barrier material.

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d. **Barrier Membranes Used During Guided Tissue Regeneration**

1. Some of the barrier Membranes in current use do not resorb (dissolve) in the human body. This necessitates a second surgical procedure to re-enter the surgical site to remove the barrier.
   a. Dense polytetrafluoroethylene is the most commonly used nonresorbable membrane.
   b. These nonresorbable membranes are embedded with titanium strips to prevent collapse of the membrane into larger osseous defects (**Figure 30-29**).

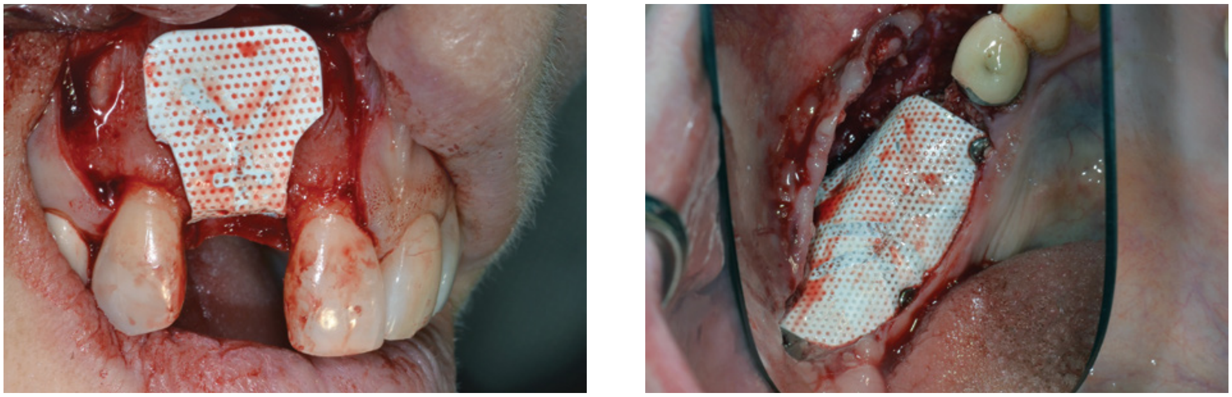

Figure 30-29 Titanium-Reinforced Membranes. Both clinical photos show titanium-reinforced membranes covering a defect. Note the embedded metallic titanium strip in each of the membranes. The embedded titanium strip adds stiffness to the membrane which prevents the membrane from collapsing into the defect and maintains the space during the regenerating phase.

Courtesy of Dr. Yusuke Hamada, Indianapolis, IN.

2. Other barrier membranes in current use are resorbable. Hence, they do not require removal at some later date; bioresorbable membranes are preferred by many clinicians for most surgical applications.
    a. Several types of bioresorbable membranes are available; these types include polyglycoside synthetic polymers, bovine and porcine collagen, and calcium sulfate (**Figure 30-30**).

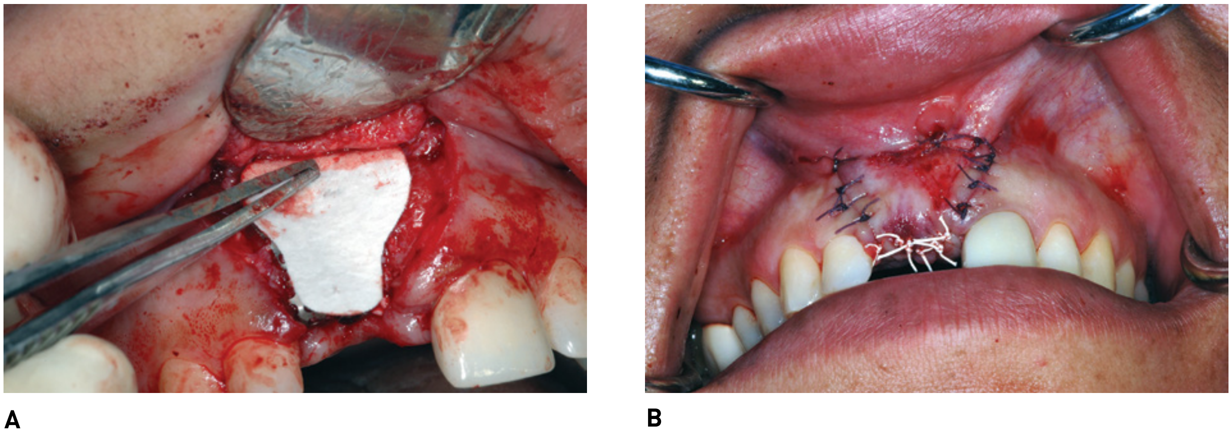

Figure 30-30 Resorbable Membrane. A. A stiff bovine-derived resorbable membrane is trimmed to fit over the grafted site. **B.** Primary closure is obtained to cover the entire surgical site.

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    b. One disadvantage of bioresorbable membranes is that they lack the same degree of rigidity offered by titanium-reinforced membranes.

e. **Use of Guided Tissue Regeneration with Bone Replacement Grafts**
    1. The simultaneous use of barrier membranes to promote regeneration along with bone replacement grafts is one clinical option. Figures 30-26 and 30-27 are cases where a barrier membrane was used in conjunction with bone graft.
    2. Available studies suggest that regeneration outcomes can be enhanced by the combined use of barrier materials with bone replacement grafts.

f. **Healing Following Guided Tissue Regeneration**
    1. The anticipated healing outcome following GTR therapy is regeneration of part or all of the periodontium that was destroyed by periodontitis.

2. As already mentioned, GTR requires the use of a barrier membrane.
    a. During surgery, a barrier is placed under the flap to impede the rapidly proliferating gingival epithelial cells from migrating along the root surface and interfering with the connective tissue regrowth on the root. (It is the connective tissue components from the periodontal ligament space that actually provide the cells needed to regrow cementum, periodontal ligament, and alveolar bone.)
    b. It is important to remember that if a barrier was not used, the gingival epithelial cells would proliferate very rapidly, covering the tooth root and blocking attachment to the root by the slower migrating connective tissue and undifferentiated cells of the periodontal ligament. The epithelial growth covering the root blocks the undifferentiated cells of the periodontal ligament from making contact with the root surface, thereby decreasing the likelihood of periodontal regeneration.

g. **Special Considerations for the Dental Hygienist**
1. During the GTR surgical procedure, every effort is made to close the wound to cover the barrier material completely.
    a. If exposure of part of the barrier is visually noted at any of the postsurgical visits, corrective measures should be instituted to minimize bacterial contamination of the barrier material.
    b. For example, a patient with exposed barrier membrane may need to be instructed to carefully apply a topical antimicrobial agent (such as chlorhexidine) over the surgical site daily to minimize bacterial contamination and postsurgical infection.
2. Sites treated by GTR should not be probed for several months following the procedure. The dental hygienist should consult with the dentist to determine when each individual site can be probed safely.

7. **Periodontal Plastic Surgery**

a. **Procedure Description**
1. **Periodontal plastic surgery** is the term most commonly used in modern dentistry to describe periodontal surgical procedures that are directed toward correcting or eliminating deformities associated with the gingiva or alveolar mucosa. Examples of periodontal plastic surgery include, but are not limited to, root coverage procedures, esthetic crown lengthening procedures, vestibuloplasty (a surgical procedure used to increase the depth of the vestibule), and frenectomy. Note that bone grafting procedures and surgical implant procedures are not covered under the broad category of periodontal plastic surgery.
2. The term *periodontal plastic surgery* includes an array of periodontal surgical procedures that can be used to improve esthetics of the dentition and to enhance prosthetic dentistry as well as to deal with damage resulting from periodontitis.[25–30]
3. Some of these procedures include techniques that have been used in medical plastic surgery for many years. Periodontal plastic surgery can be used to alter the tissues surrounding both natural teeth and dental implants.

b. **Terminology Related to Periodontal Plastic Surgery**
1. Readers of periodontal literature can sometimes be confused by the terminology associated with periodontal plastic surgery because other terms have also been used to describe procedures currently included under this term.
2. The term **mucogingival surgery** has been used in the past to describe periodontal surgical procedures that correct any deformity associated with the gingiva and mucosa. Some of the periodontal plastic surgical procedures utilized in modern dentistry were previously described as *mucogingival surgical procedures*, and this older terminology can still be encountered.
3. Another term that has been used to describe some of these types of procedures is *reconstructive surgery*; the term *reconstructive surgery* underscores that the goal of some of these procedures is to reconstruct (or rebuild) periodontal tissues such as gingiva.

c. **Goals of Periodontal Plastic Surgery**
1. Many periodontal plastic surgical procedures are designed to alter components of the keratinized gingiva, and this type of procedure can dramatically alter the appearance of the tissues. Most patients want a pleasing smile, and because the gingiva is readily visible in many patients, patients frequently seek improvements in the appearance of the gingiva.
2. In addition to altering the appearance of the tissues, some periodontal plastic surgical procedures improve function. Function can be compromised when lack of keratinized gingiva on a tooth limits the restorative options.

3. This section includes an overview of some of the more common types of procedures included under the heading periodontal plastic surgery. **Box 30-6** provides an overview of the types of procedures commonly included in periodontal plastic surgery.

Box 30-6 Overview of Procedures Commonly Included in Periodontal Plastic Surgery

- Free soft tissue autograft (previously called the free gingival graft)
- Subepithelial connective tissue autograft
- Laterally positioned flap
- Coronally positioned flap
- Semilunar coronally repositioned flap
- Frenectomy
- Crown lengthening surgery

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8. **Free Soft Tissue Autograft**
   a. **Procedure Description**
      1. A **free soft tissue autograft (FSTA)** is a type of periodontal plastic surgery that was one of the first procedures used to augment the width of keratinized tissue. This procedure previously was called the free gingival graft.
      2. In the free soft tissue autograft (FSTA) procedure, epithelial and subepithelial soft tissues (connective tissue), from the same patient, are transplanted from one area of the mouth to another. As such, there are two intraoral wounds that are created during this surgery: the donor site and the recipient site.
         a. The donor tissue for a FSTA includes both the surface epithelium and some of the underlying connective tissue.
         b. Harvesting the tissue from the donor site leaves an open wound that can be discomforting and painful for the patient if left exposed during the healing phase (**Figure 30-31**). To minimize adding postoperative pain and discomfort to the donor site, the clinician will typically protect the donor site with a dressing material or with a plastic stent. Over several weeks, the donor site will eventually heal by secondary intention.

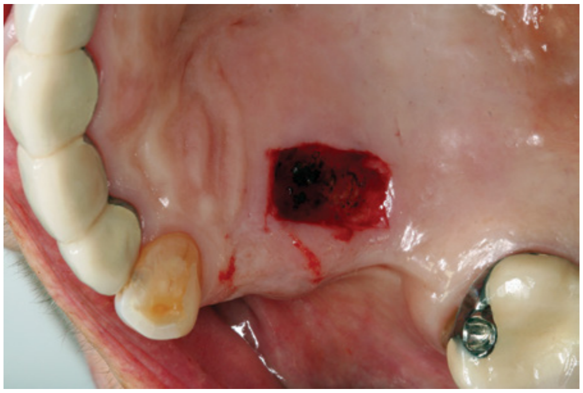

Figure 30-31 Donor Site of a Free Soft Tissue Autograft. This donor site on the palate will heal as an open wound (since the epithelium has been removed as part of the donor tissue). The donor site should be covered with a plastic stent or dressing material to minimize patient discomfort, aid in bleeding control, and protect the wound from the tongue, food, and drink. If the donor site remains covered during the healing phase, the open wound will eventually heal by secondary intention.

Courtesy of Dr. Jennifer Chang, Indianapolis, IN.

3. **Figure 30-32** shows a free soft tissue autograft case from start to finish.

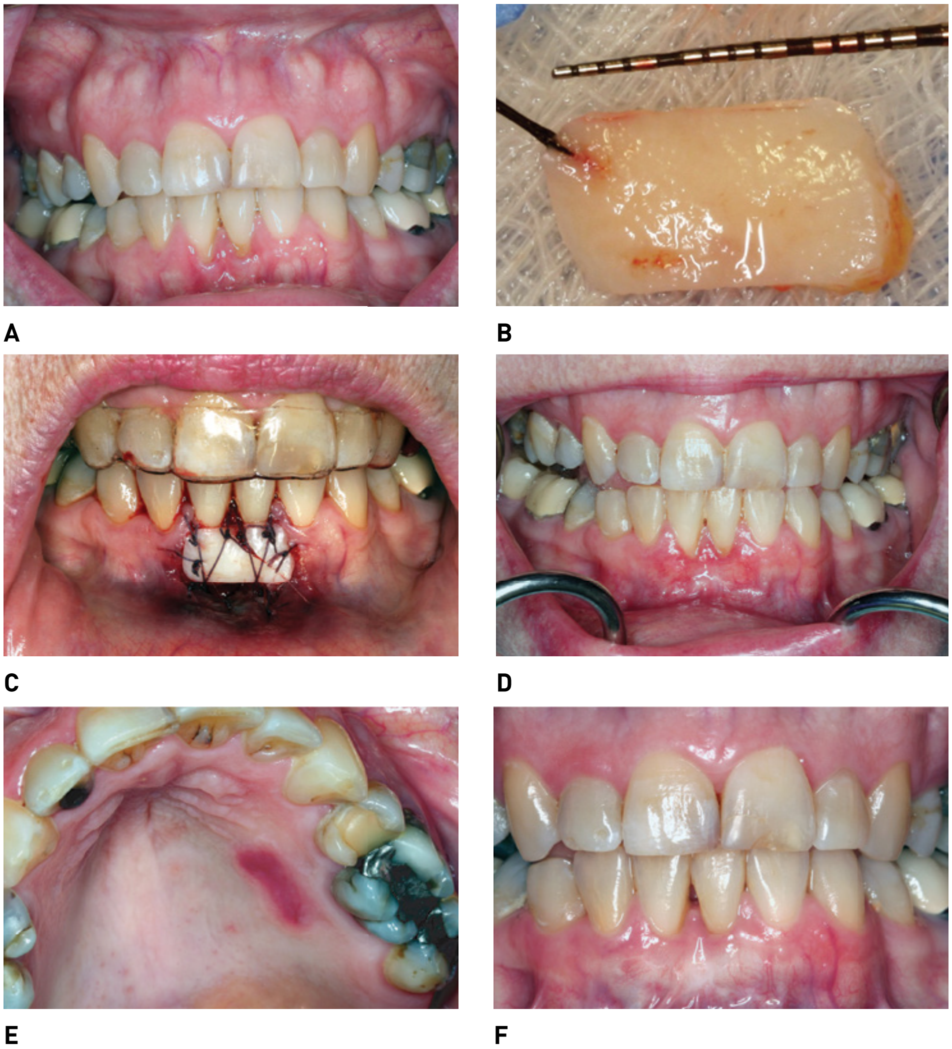

Figure 30-32 **Free Soft Tissue Autograft. A.** Presurgical (pregrafting) photo shows gingival recession on the mandibular central incisors. **B.** Donor tissue obtained from the patient's own palate. **C.** Donor tissue sutured over the recipient site. Although the appearance of the donor tissue may look as if the autograph is failing, this is a typical appearance of the donor tissue 7 to 10 days after placement. **D.** Healing of recipient site at 1 month. **E.** Healing of donor site at 1-month. **F.** Healing of recipient site at 12 months. Note the increase in root coverage obtained and maintained after 1 year.

Courtesy of Dr. Jennifer Chang, Indianapolis, IN.

4. The FSTA may be indicated in cases that require root coverage over exposed root surfaces or in cases that require augmentation of the width of keratinized gingiva, without the need for obtaining root coverage.
5. One complicating factor of the FSTA is that the graft is completely severed from its blood supply and at least a portion of the graft is then placed over an avascular root surface. Thus, special care is required to promote diffusion of nutrients to the graft to maintain its viability during the early stages of healing.

b. **Steps in Performing a Free Soft Tissue Autograft Procedure**

1. The root surfaces in the area of gingival recession are instrumented to remove plaque biofilm, calculus, and root irregularities.
2. Horizontal and vertical incisions are made at the recipient site after determining the precise location of the needed graft. Using sharp dissection, surface epithelium is removed to prepare a firm connective tissue bed to receive the graft material.
3. A template (frequently made from foil) is prepared to provide a pattern for the exact size and shape of the donor graft that will be needed.

4. Using the template as a guide for the size and shape, the graft is obtained from the donor site (usually the palate) by incising through the epithelium and through a thin layer of connective tissue beneath the epithelium; the graft is removed from the site using sharp dissection.
5. The graft is sutured to the recipient site; during suturing, care is taken to prevent a blood clot from forming between the graft and the recipient vascular bed. A clot in between the graft and recipient site may interfere with vascularization of the donor tissue and may lead to graft necrosis.
6. Both the donor site and recipient site are protected with periodontal dressing; in some instances, the donor site on the palate is covered with a previously prepared acrylic retainer to hold the dressing over the donor site.

c. **Healing Expected with a Typical Free Soft Tissue Autograft**

1. Successful healing of a FSTA depends upon the survival of the connective tissue part of the graft. In most instances, the superficial layer of epithelium sloughs off during the healing period, later to be replaced by new epithelium.
2. Survival of the tissues depends initially upon diffusion of fluid from the vascular recipient bed, followed by growth of new blood vessels into the grafted material. Immobilization of the autograft during the healing phase is a critical element in allowing the diffusion of nutrients, reconnection of existing blood vessels, and formation of new blood vessels.
3. Successful augmentation of gingiva as well as successful root coverage has been reported following the use of the free soft tissue autograft.
4. Unfortunately, in some cases, following complete healing of the free soft tissue autograft, the esthetic results may appear less than ideal. For example, there may be a less than ideal color match between the healed graft and the adjacent gingiva. Moreover, the healed free soft tissue autograft may appear "lumpy" and bulkier than the surrounding tissues, giving the grafted tissue what is known as a "tire-patch" appearance (**Figure 30-33**).

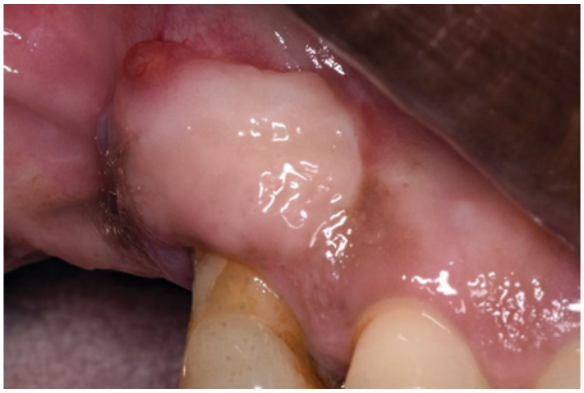

Figure 30-33 "Tire-Patch" Appearance of a Free Soft Tissue Autograft. Postsurgical photo at 12 weeks following FSTA placement. At this point in time, the FSTA placed over the maxillary canine has successfully integrated into the recipient site. However, esthetically, it looks like a "tire-patch," which means the new grafted tissue appears bulbous and irregular with respect to the surrounding gingival tissues. The FSTA will need to be followed up by a second surgery, known as a gingivoplasty, to blend the graft with the surrounding gingiva so that its final appearance is more natural and esthetically pleasing. (For this case, the primary objective of performing the free soft tissue autograft was to augment the keratinized tissue, *not* to cover the exposed root surface. Hence, the primary objective was met. The secondary objective will be met following the gingivoplasty. The reader can refer to Figure 30-45.)

Courtesy of Dr. Erik Rose, Indianapolis, IN.

9. **Subepithelial Connective Tissue Graft**

a. **Procedure Description**

1. The **subepithelial connective tissue graft (SCTG)** is another type of periodontal plastic surgical procedure that can augment the width of keratinized gingiva or to cover areas of gingival recession.
2. In addition to gingival augmentation, the subepithelial connective tissue graft can also augment the contour of alveolar ridges to improve the esthetics of some types of dental prostheses.

    3. Unlike a free soft tissue autograft, which retains the overlying gingival epithelium, a subepithelial connective tissue graft is solely composed of connective tissue (and sometime adipose tissue) without the overlying epithelium. A subepithelial connective tissue can be harvested from a variety of intraoral sites, but is usually taken from the patient's palate.

b. **Steps in Performing a Subepithelial Connective Tissue Graft Procedure**
    1. A partial-thickness flap is elevated at the recipient site using sharp dissection; the flap normally extends one half to one tooth to the mesial and distal of the site of recession to be covered.
    2. The exposed tooth root is thoroughly instrumented to remove plaque biofilm, calculus, and root irregularities.
    3. The connective tissue graft is obtained by incising through the epithelium of the palate and excising a segment of connective tissue from beneath the epithelium using sharp dissection. The surface tissues at the donor site can then be sutured to allow for healing by primary intention (**Figure 30-34**). This facilitates improved healing compared to the type of healing that occurs in the palate from a free soft tissue autograft procedure.

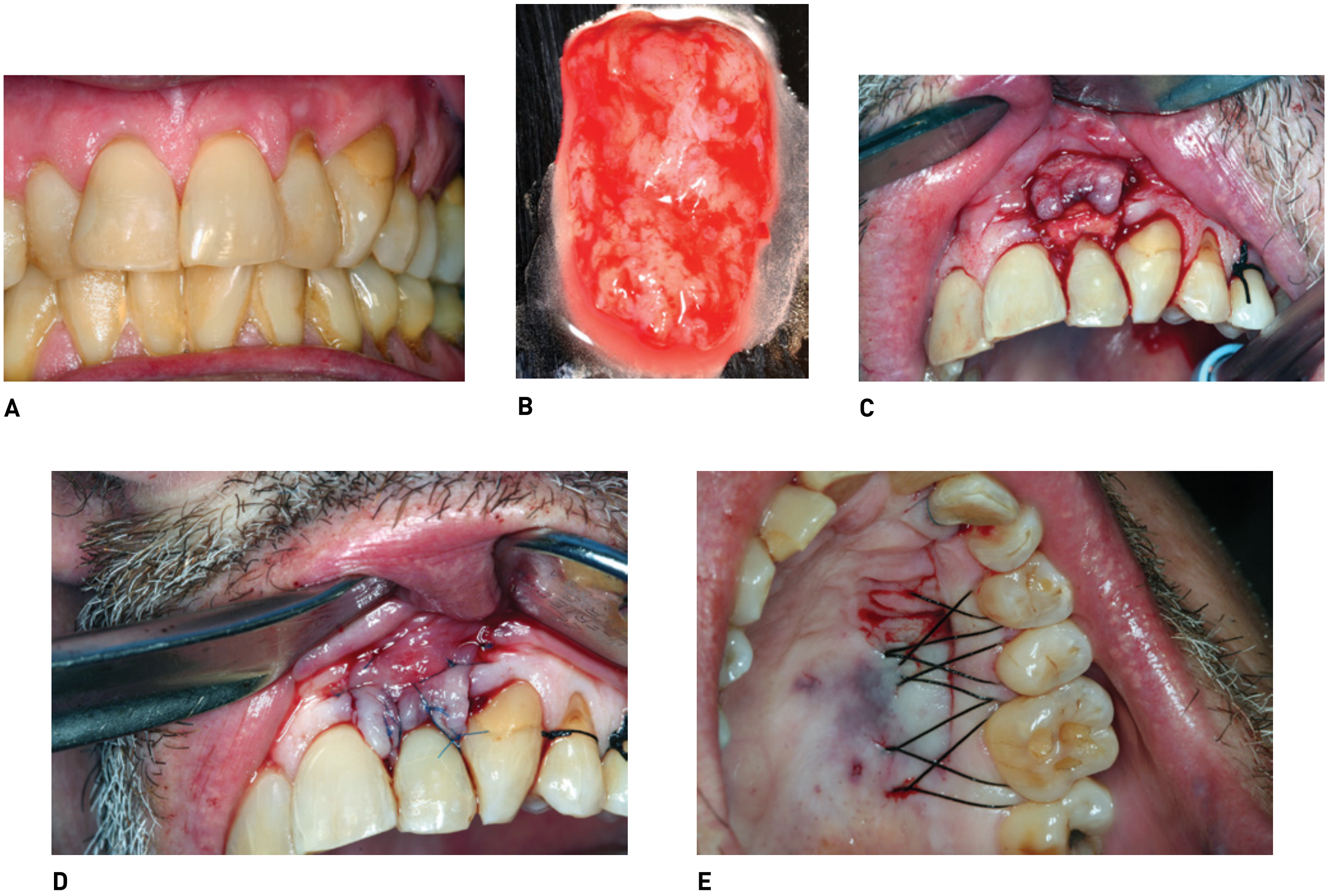

Figure 30-34 Subepithelial Connective Tissue Graft. A. Gingival recession defect on the maxillary left lateral incisor. **B.** Photo of connective tissue autograft harvested from the palate. Compared to the appearance of a free soft tissue autograft (FSTA) (refer to Figure 30-32B), the connective tissue autograft is much more vascular (because the SCTG is composed of thicker connective tissue which has more capillaries). Further, when viewing the connective tissue autograft, it becomes more apparent that there is no overlying epithelium (whereas a FSTA is composed of an overlying epithelial layer and a thin superficial connective tissue layer). **C.** After reflecting a partial-thickness flap over the recipient site, the harvested autogenous donor tissue was placed over the recipient site. **D.** Suturing of the recipient site. The graft and overlying partial-thickness flap was then sutured and secured in place. Note the complete coverage of the connective tissue graft. **E.** Suturing of the donor site. Note that the donor site is completely closed. The wound in the donor site will heal by primary intention. This will aid in a more rapid healing with less patient discomfort compared to the healing that occurs at the donor site from a free soft tissue autograft procedure.

A, C-E: Courtesy of Dr. Jennifer Chang, Indianapolis, IN.

4. The graft tissue is placed over the denuded tooth root and under the partial-thickness flap at the recipient site. The outer portion of the partial-thickness flap is placed over the graft and sutured into place, making sure that at least half of the graft is covered by the outer portion of the flap.
5. Periodontal dressing may be placed to protect the grafted site; because the donor site will heal by primary intention, normally no dressing is needed at the donor site.

c. **Healing Expected After a Subepithelial Connective Tissue Graft**

1. When root coverage is attempted with the subepithelial connective tissue graft, it is reasonable to expect coverage, though not all sites result in complete root coverage.
2. The subepithelial connective tissue graft results in excellent esthetics since the color of the healed tissues often mimics the natural preexisting tissue color precisely (**Figure 30-35**).

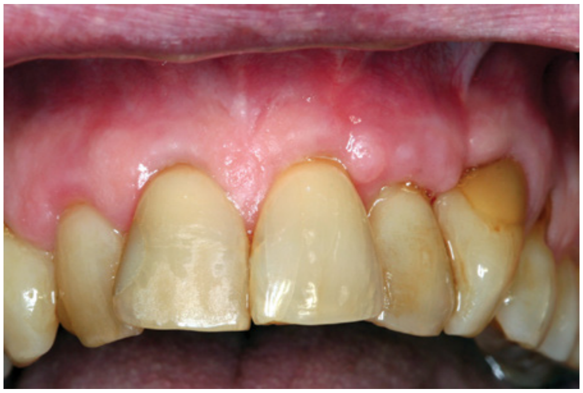

Figure 30-35 Healing of Subepithelial Connective Tissue Graft at 6 Weeks Following Surgery. This is the same case shown in Figure 30-34 at the 6-week postsurgical stage. Note the excellent color matching of the recipient site with neighboring tissues.

Courtesy of Dr. Jennifer Chang, Indianapolis, IN.

d. **Acellular Dermal Matrix Allograft**

1. As a substitute for autogenous tissue, an acellular dermal matrix allograft material is now available. This type of graft material is harvested from donated skin tissue (**Figure 30-36**).

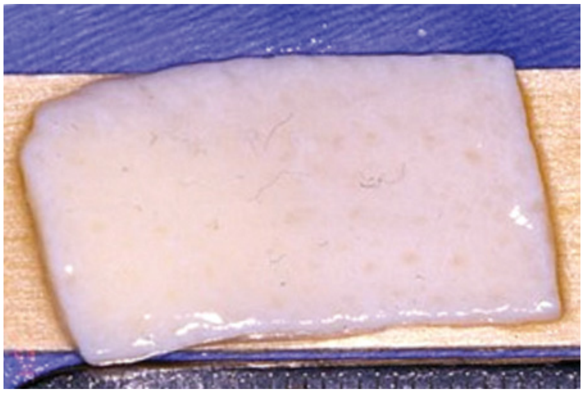

Figure 30-36 Acellular Dermal Matrix Allograft. An acellular dermal matrix (ADM) allograft is a soft, strong, pliable connective tissue graft that conforms readily to the periodontal defect and is easy to handle in the hands of a competent surgeon. In its basic form, ADM is human dermis that has been de-epithelialized (unlike a FSTA which retains the epithelial layer on the graft) and decellularized (unlike a FSTA or SCTG which retains viable cells). Nevertheless, ADM mimics the native tissue environment and acts as a biological scaffold that facilitates host cells to form new tissue over it.

2. Following procurement, the allograft is treated to remove all cellular components. It is also carefully screened to remove the HIV-1 virus and hepatitis C. After it is thoroughly processed, the remaining structural framework consists of a three-dimensional arrangement of blood vessel channels, collagen, elastin, and proteoglycans that stimulates the recipient's own tissue regeneration, integrating and replacing the graft tissue with newly formed dense, collagenous tissue.
3. This dermal matrix allograft material can be used in place of autogenous connective tissue. However, when it is placed over an avascular root surface, it must be covered by as much of the flap as possible to ensure rapid vascularization from the overlying gingival flap (**Figure 30-37**).

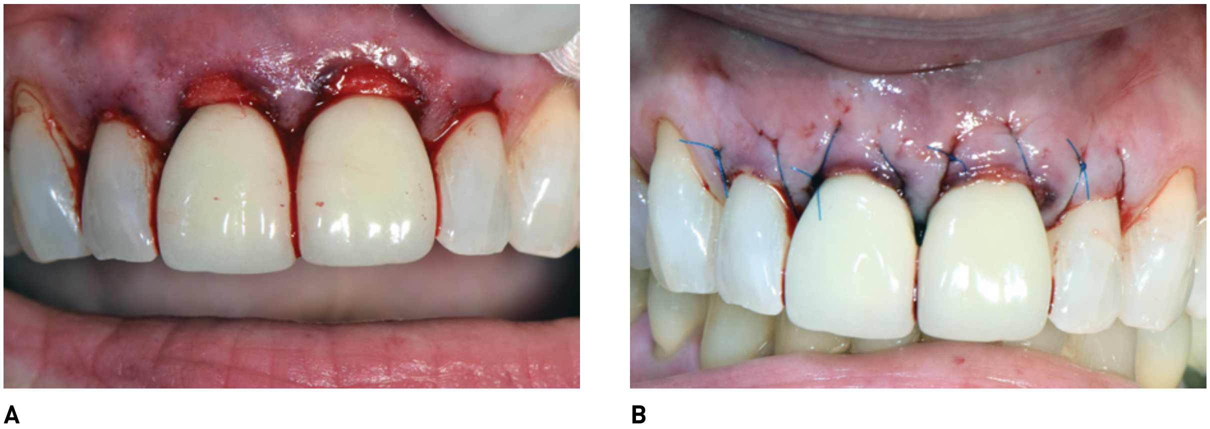

Figure 30-37 Surgical Placement of Acellular Dermal Matrix Allograft. A. Surgical placement of acellular dermal matrix allograft in the recipient site. **B.** Acellular dermal matrix covered by gingival flap.

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4. Advantages of using the acellular dermal matrix allograft include avoidance of needing a palatal donor site, the availability of unlimited donor tissue, and improved patient acceptance due to the benefits of not requiring an intraoral donor site.

10. **Laterally Positioned Flap**
    a. **Procedure Description**
        1. The **laterally positioned flap** is a periodontal plastic surgery technique that can be used to cover root surfaces with gingiva in isolated sites of gingival recession.
        2. The laterally positioned autograft involves a displaced flap (displaced laterally in this case) that "slides" over to cover a neighboring recession defect (**Figure 30-38**). Another term to describe this type of flap is a *lateral displaced pedicle flap*.

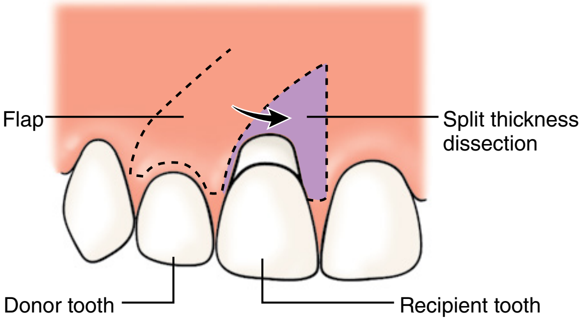

Figure 30-38 Laterally Positioned Flap. The flap is rotated to "slide" over from the donor site to the neighboring recipient site.

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3. The primary prerequisite of this technique is that the donor site must be adjacent to the recipient site so that the flap can "slide" over to cover the recipient site.
4. A secondary prerequisite is that the donor site itself must have a thick, healthy layer of gingiva covering it to prevent postsurgical recession at the donor site.

b. **Steps in a Typical Laterally Positioned Flap**
   1. The recipient site is prepared by thoroughly instrumenting the exposed tooth root and by removing epithelium from the surface of the gingiva surrounding the area of recession, thus exposing some connective tissue to serve as a vascular recipient bed for the displaced flap.
   2. A partial-thickness flap is elevated from the donor site using a series of carefully planned vertical incisions to provide mobility in the flap after elevation.
   3. The elevated flap is rotated laterally so as to cover the recipient site, including both the prepared bed of connective tissue and the prepared root surface.
   4. The flap is stabilized at its new location using a combination of interrupted sutures and sling sutures.
   5. If the surgery was extensive, the clinician may decide to cover the surgical site with aluminum foil and/or periodontal dressing to protect the healing wound.

c. **Expected Healing of a Laterally Positioned Flap**
   1. With careful selection of donor sites and skillful manipulation of the tissues, minimal post-surgical recession will occur on the donor site.
   2. The laterally positioned flap can result in excellent root coverage because the flap maintains part of its own blood supply (unlike the free soft tissue autograft, which is completely severed from its blood supply).

11. **Coronally Positioned Flap**

a. **Procedure Description**
   1. The **coronally positioned flap** is a periodontal plastic surgical procedure that can be used to treat gingival recession if the recession is not severe. Another term to describe this type of flap is a *coronally displaced flap*.
   2. As the name implies, the coronally positioned flap is a displaced flap—the flap is advanced coronally to cover the gingival recession defect.[31] For an example, refer to Figure 30-15.
   3. One advantage to this procedure compared to a FSTA procedure or a SCTG procedure is that it does not require a second surgical site to provide the donor tissue (in other words, the donor site is the recipient site). Another advantage of this procedure compared to other periodontal plastic surgical procedures is that it is simple and predictably provides 2 to 3 mm of root coverage.
   4. One disadvantage to this procedure is that it can be difficult to stabilize and secure the flap with sutures at a more coronal position. Another disadvantage is that there must be a sufficient band of thick keratinized tissue to avoid postsurgical gingival recession.

b. **Steps in a Typical Coronally Positioned Flap**
   1. Exposed tooth roots are instrumented to remove plaque biofilm, calculus, and root irregularities.
   2. Internal bevel and vertical releasing incisions are made at the site so that the flap can be coronally positioned; the vertical releasing incisions extend into the alveolar mucosa to allow for mobility of the flap margin in a coronal direction.
   3. The flap is elevated; the elevation can be full-thickness or split-thickness or a combination of the two depending upon the overall thickness of the tissues being elevated.
   4. The flap is advanced in a coronal direction and sutured using a combination of interrupted and sling sutures.
   5. Periodontal dressing is placed to prevent movement of the flap during healing.

c. **Healing Expected with a Typical Coronally Positioned Flap.** The coronally positioned flap can be used successfully to cover areas of gingival recession when the gingival recession is not severe.

12. **Semilunar Coronal Repositioned Flap**

a. **Procedure Description.** Tarnow proposed a procedure known as the **semilunar coronally repositioned flap** to cover gingival recession where the recession is not far advanced and where the keratinized tissues have an adequate thickness (**Figure 30-39**). The semilunar flap is a variation of a coronally positioned flap.[32]

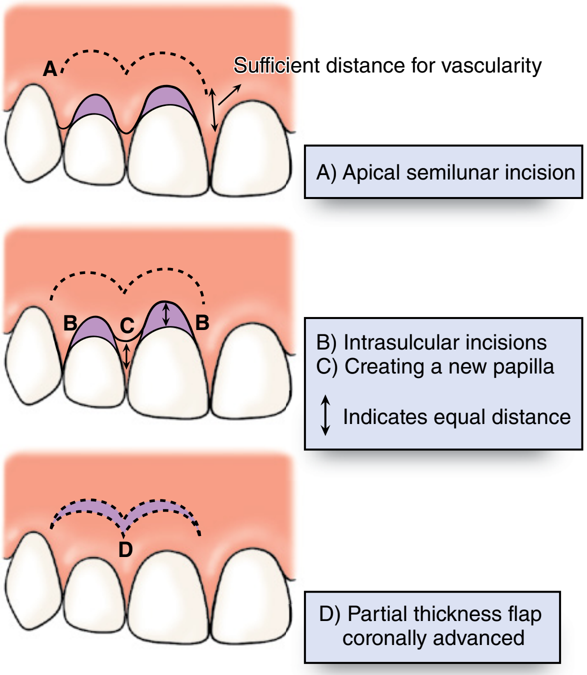
A
Sufficient distance for vascularity
A) Apical semilunar incision
B
C
B
B) Intrasulcular incisions
C) Creating a new papilla
↕ Indicates equal distance
D
D) Partial thickness flap coronally advanced

Figure 30-39 **Semilunar Coronally Positioned Flap.** A semilunar coronally repositioned flap is used to cover gingival recession where the recession is not far advanced and where the keratinized tissues have an adequate thickness.

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- b. **Steps in a Typical Semilunar Coronally Repositioned Flap**
  1. The level of the alveolar bone is located (sounded) to ensure that coronal positioning of the semilunar flap does not inadvertently expose alveolar bone at the base of the flap.
  2. A semilunar, curved incision is made from one interdental area to the adjacent interdental area over the tooth root. The interdental sites for the incisions are selected to be slightly coronal to the position anticipated for the flap advancement. This incision begins in the gingiva and arcs into the mucosa and then back into the gingiva.
  3. A split-thickness flap is performed using sharp dissection to free the surface of the flap from the underlying connective tissue.
  4. The semilunar flap is displaced coronally and stabilized with gentle pressure for several minutes; if needed, the flap can be stabilized with interrupted sutures, but sometimes suturing is not required.

13. **Frenectomy**
    - a. **Procedure Description**
      1. **Frenectomy** is a periodontal plastic surgical procedure that results in removal of a frenum, including removal of the attachment of the frenum to bone (**Figure 30-40**).
         - a. Some authors use the term *frenotomy* to indicate a variation of the frenectomy.
         - b. The frenotomy includes only incision of the frenum, but does not remove the attachment of the frenum from the bone surface.
         - c. A frenum is a fold of mucosal tissue that contains muscle fibers. It attaches the lips and cheeks to the alveolar mucosa.

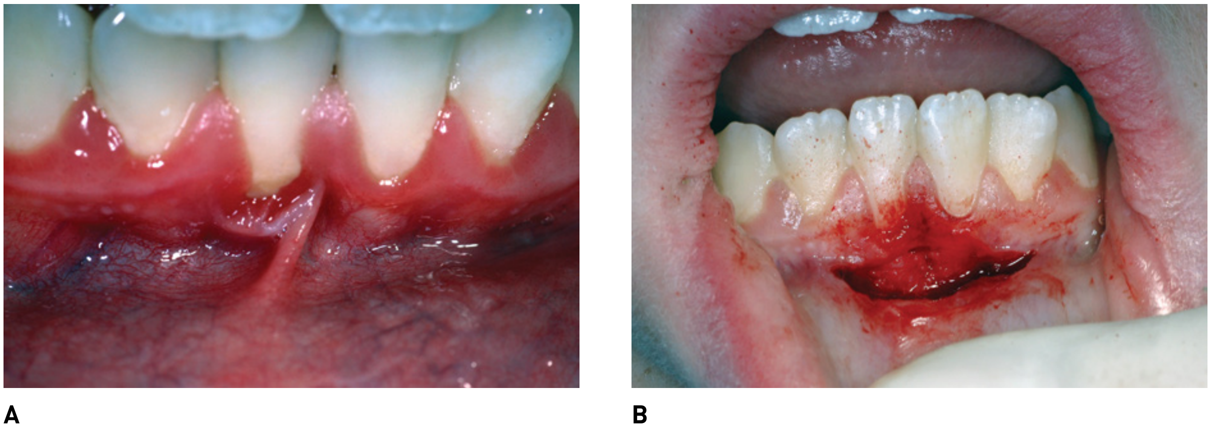

Figure 30-40 **Frenectomy of Mandibular Frenum. A.** Note the plaque-induced inflammation around the mandibular central incisors. The frenum is interfering with proper plaque biofilm control. **B.** Frenum is resected to expose the fibrous attachment to the bone. These fibrous attachments will need to be severed with blunt dissection to the bone.

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2. If a frenum is attached too close to the gingival margin, it will act as a cable that constantly pulls the gingival margin away (apically) from the tooth when the enclosed muscle fibers in the frenum tense up. This can contribute to a buildup of plaque biofilm around the exposed root surface and marginal gingiva and eventually lead to persistent inflammation. In addition, a frenum too close to the gingival margin can interfere with daily self-care because it may be discomforting every time the bristles of the toothbrush contact the exposed root surface and movable frenum.
    a. An aberrant frenum position that requires a frenectomy occurs most often in the frenum between the maxillary central incisors and mandibular central incisors.
    b. An aberrant frenum position can also occur in other locations such as on the facial surface of premolar and canine teeth and on the lingual surface of the mandibular central incisors.
3. If a frenum is present in a fully edentulous patient who is planned to receive a denture, a frenectomy may need to be performed prior to fabrication of the denture. This is because the movable frenum can dislodge the denture from the ridge, thereby affecting its retention. (**Figure 30-41**).

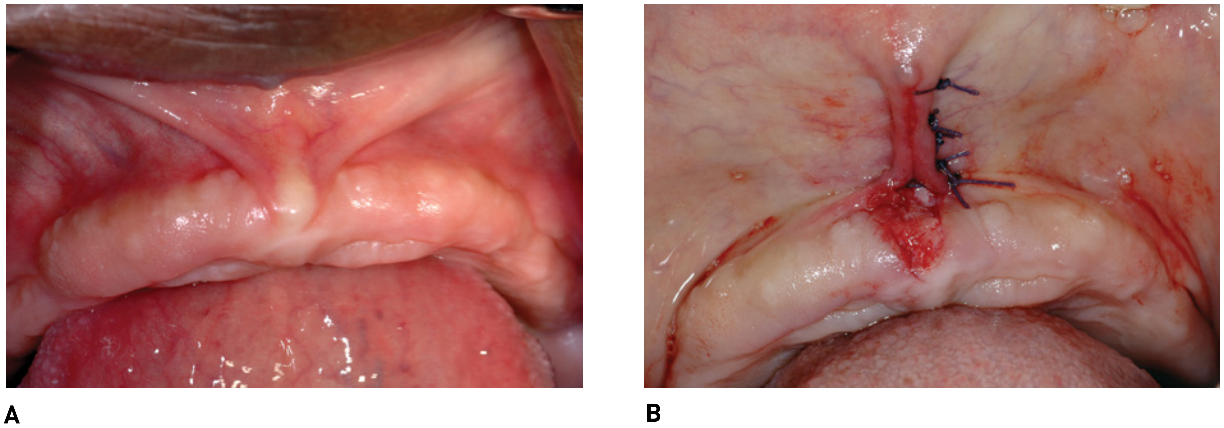

Figure 30-41 **Frenectomy in Fully Edentulous Patient. A.** A broad maxillary labial frenum can interfere with the seating of the denture and can cause discomfort if the denture impinges on the frenum. As such, a frenectomy is indicated. **B.** Note the complete removal of the frenum, including its attachment to the underlying bone.

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- b. **Steps in a Typical Frenectomy**
    - 1. The frenum is grasped with a hemostat placed to the depth of the vestibule.
    - 2. Incisions are made through the tissues on both the under surface and the upper surface of the beaks of the hemostat.
    - 3. The triangular piece of tissue held by the hemostat is removed exposing connective tissue over the surface of the bone.
    - 4. The fibrous attachment covering the bone is incised and dissected with a blunt instrument.
    - 5. Periodontal dressing is applied to the wound.
- c. **Alternative Techniques for the Frenectomy**
    - 1. Frequently, the frenectomy is performed in conjunction with other types of periodontal surgery, and a variety of techniques have been described.
    - 2. Other techniques for performing a frenectomy include removing the tissue of the frenum with electrosurgery or with a laser.
- d. **Healing following a Frenectomy.** Expected healing following a frenectomy is elimination of the gingival margin movement caused by the frenum.

14. **Crown Lengthening Surgery**
    - a. **Procedure Description. Crown lengthening surgery** refers to periodontal plastic surgery designed to elongate the height of the clinical crown at the expense of removing a portion of the gingiva, and, when indicated, some alveolar bone.
    - b. **Terminology.** Two types of crown lengthening procedures are *functional crown lengthening* and *esthetic crown lengthening*.
        - 1. Functional crown lengthening is performed when the remaining tooth structure is inadequate to support a needed restoration.
            - a. Functional crown lengthening augments the outcome of a restorative dental procedure (such as a crown) done on a tooth with inadequate axial tooth height.
            - b. Crown lengthening surgery is necessary when a tooth is decayed or fractured below the gingival margin.
                - 1. When a badly damaged tooth is to be restored, the dentist will evaluate the tooth and surrounding tissues to determine if the final restoration of the tooth will damage the soft tissue attachment (i.e., encroach upon the supracrestal tissue attachment).
                - 2. If such damage can be expected, functional crown lengthening surgery is usually indicated prior to the placement of the final restoration.
        - 2. **Esthetic crown lengthening** refers to crown lengthening performed on teeth to improve the appearance of the teeth where there is excessive gingiva, or a "gummy smile" as it is sometimes called.
            - a. Crown lengthening surgery can be used to improve the esthetics of the gingiva, especially on anterior teeth with short clinical crowns.
                - 1. An individual's smile may be unattractive because of the height or lack of symmetry of the gingiva surrounding the teeth.
                - 2. In some cases, tooth crowns are actually the correct length, but they appear too short in the mouth because there is an excess of gingival tissue covering the teeth.
            - b. During esthetic crown lengthening, the gingival tissues are incised and reshaped to expose more of the natural crown of the tooth; frequently some of the alveolar bone must also be removed to ensure healing of the tissues at a more apical position (**Figure 30-42**).

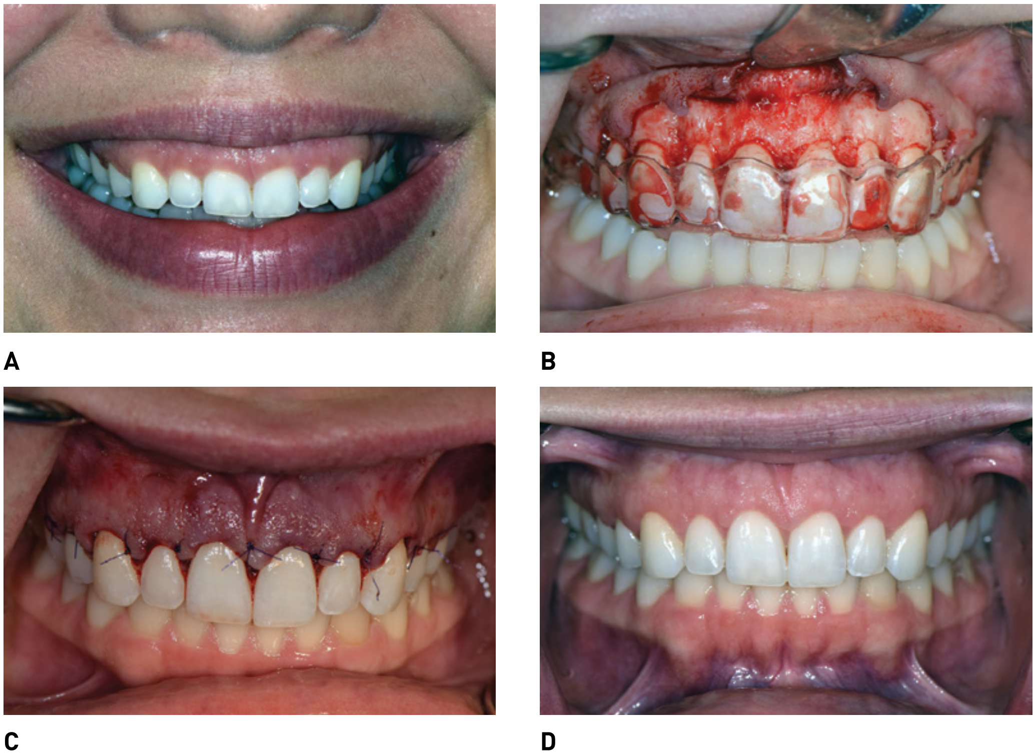

Figure 30-42 **Esthetic Crown Lengthening. A.** The clinical crowns from premolar to premolar appear short. **B.** Following the appropriate incisions and flap elevation, osseous recontouring was performed to expose more of the clinical crown length. **C.** Final suturing of the flap. Note the apical repositioning of the gingival margin. **D.** Three-month postsurgical clinical photograph. Note the increased clinical crown lengths of the maxillary teeth.

Courtesy of Dr. Hawra AlQallaf, Indianapolis, IN.

c. **Surgical Procedure.** The actual surgical procedure during crown lengthening surgery usually involves an apically positioned flap (displaced flap) with osseous resective surgery much like that already discussed.
   1. Unlike the typical indications for an apically positioned flap with osseous surgery, esthetic crown lengthening surgery is indicated in the presence of a perfectly healthy periodontium simply to allow for improved esthetics or exposure of more tooth structure prior to restoration.
   2. Also, unlike the typical apically positioned flap with osseous surgery, esthetic crown lengthening frequently requires the use of a surgical template prepared to guide the surgeon in positioning the tissues during the surgery and to determine the final, most esthetically pleasing apical location of the gingival margin (**Figure 30-43**).

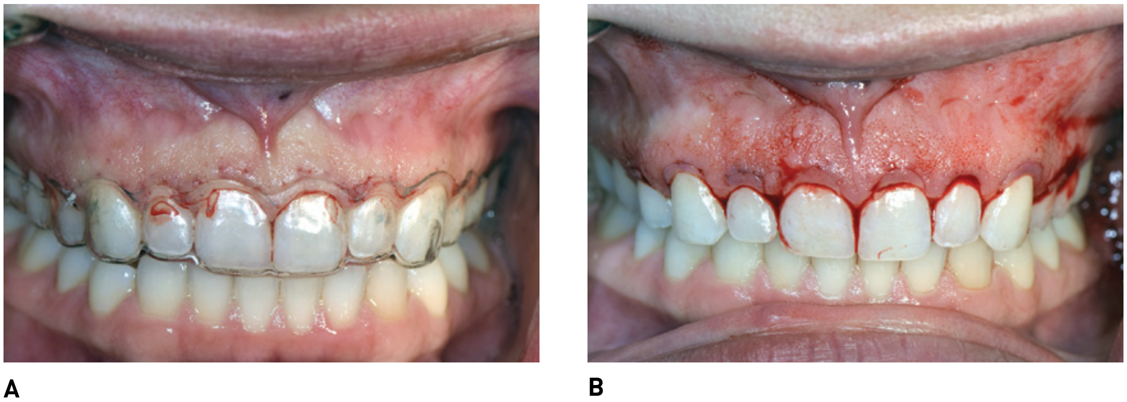

Figure 30-43 **Purpose of the Surgical Template in Esthetic Crown Lengthening. A.** Prior to esthetic crown lengthening surgery, a transparent, acrylic surgical template was fabricated. **B.** The surgical template is used as a guide to help the surgeon position the tissues during surgery.

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3. Occasionally, esthetic crown lengthening may require only a gingivectomy type procedure to be discussed later in this chapter. But, most often, esthetic crown lengthening requires some alveolar bone reduction, so an apically positioned flap with osseous surgery is most often indicated instead of a gingivectomy.

d. **Healing After Crown Lengthening Surgery**
   1. Healing of crown lengthening surgery is similar to that described for the apically positioned flap with osseous surgery.
   2. Final healing of crown lengthening surgery results in a normal attachment (both junctional epithelium and connective tissue attachment) at a position more apical on the tooth root.

e. **Special Considerations for the Dental Hygienist**
   1. Because crown lengthening surgery usually leads to root exposure, temporary dentinal hypersensitivity may occur; it is imperative for the dental team to warn patients in advance that they may experience temporary dentinal hypersensitivity following crown lengthening surgery (**Figure 30-44**).

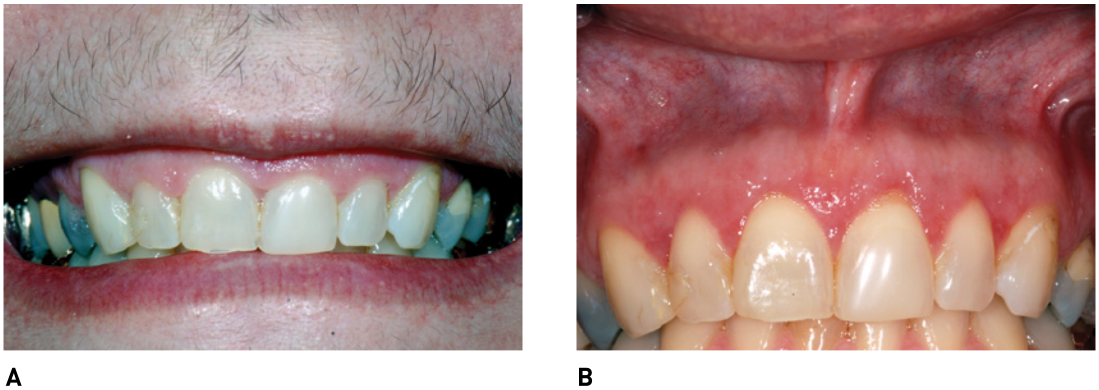

Figure 30-44 Root Exposure Following Esthetic Crown Lengthening. A. Pre-esthetic crown lengthening photograph. Note the gingival asymmetry and the short lengths of the clinical crown of the maxillary anterior teeth. **B.** Two-week post-esthetic crown lengthening photograph. At this time, the patient experienced slight dentinal hypersensitivity due to 1 mm of root exposure on the facial aspect of the maxillary left central incisor. The hypersensitivity subsided within several weeks, and the patient was pleased with the outcome. Note the meticulous plaque control that is achieved by the efforts of both the patient and her dental hygienist.

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   2. As already discussed, when dentinal hypersensitivity results, the dental hygienist may need to institute measures to help the patient deal with the sensitivity.
   3. Control of dentinal hypersensitivity requires meticulous plaque control during the healing phase, and this can be a problem because mechanical plaque control must be restricted following most surgical procedures.
   4. It is the responsibility of the members of the dental team to aid the patient in plaque control until healing allows the patient to resume routine self-care.

15. **Gingivectomy**

a. **Procedure Description**
   1. The **gingivectomy** is a surgical procedure designed to excise some of the gingival tissue. Historically, the gingivectomy was used for many years in periodontics as the primary treatment modality. In modern dentistry, however, the gingivectomy procedure now has a reduced role because this procedure has a higher rate of surgical relapse (the soft tissue may grow back), slower wound healing, and greater postsurgical discomfort and bleeding compared to surgical procedures that heal by primary intention. Also, the gingivectomy procedure fails to conserve keratinized tissue and maintain esthetics.
   2. When a gingivectomy is performed, the tissues are excised (cut away), removing some of the gingiva that would normally be attached to the tooth surface. Thus, the gingivectomy is a resective procedure. It is important to keep in mind that the gingivectomy procedure does not involve any removal of bone.
   3. The gingivectomy results in a more apical position of the marginal gingiva in relationship to the CEJ of the tooth.
   4. Terminology related to gingivectomy can be confusing because of the overlapping use of two terms: *gingivectomy* and *gingivoplasty*.

a. In contrast to the gingivectomy described above, **gingivoplasty** is a term used to describe a surgical procedure that simply reshapes the surface of the gingiva to create a natural form and contour to the gingiva (**Figures 30-45A-E**).

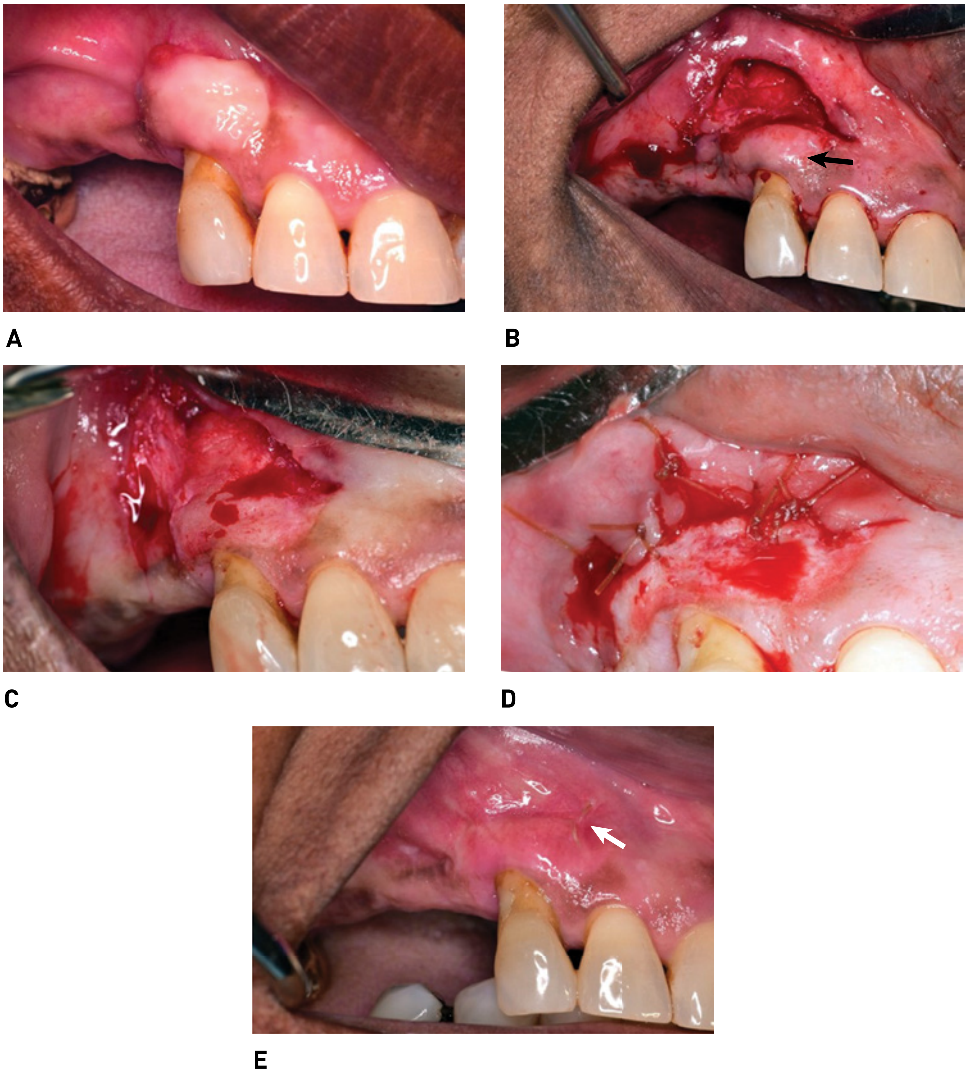

Figure 30-45 **Gingivoplasty Surgical Procedure. A.** "Tire-Patch" Appearance of a Free Soft Tissue Autograft (FSTA). At this point in time, the FSTA placed over the maxillary canine has successfully integrated to the recipient site. However, esthetically, it looks like a "tire-patch" which means the new grafted tissue appears bulbous and irregular with respect to the surrounding gingival tissues. The FSTA will need to be followed up by a gingivoplasty to blend in the FSTA with the neighboring gingival tissues so that the final appearance of the tissue is more natural and esthetically pleasing.
B. An ellipitically-shaped split-thickness incision is made that includes approximately 2 mm of the coronal border of the alveolar mucosa tissue and approximately 2 mm of apical border of the excess FSTA graft. The incised tissue was subsequently removed, but the periosteum was left covering the alveolar bone since the intent of the surgery did not involve bone removal. Note the preservation of the gingival tissue directly attached to the tooth (black arrow).
C. Gingivoplasty is done on the gingival tissue directly attached to the tooth to create a uniform thickness of the graft which blends with the surrounding gingiva. This is done with a combination of scalpel blades and rotary instruments.
D. Absorbable sutures are used to approximate the mucosal margin to the recontoured gingival tissue. **E.** Two-week post-surgical photo. Note the uneventful healing and the more esthetically-pleasing appearance of the graft. Also note that the thickness of the graft blends well with the thickness of the surrounding gingival tissues. Observe a suture (white arrow) remaining which was later removed at this post-surgical visit.

Courtesy of Dr. Erik Rose, Indianapolis, IN.

b. Unlike the gingivectomy, *gingivoplasty* implies reshaping the surface of the gingiva without removing any of the gingiva actually attached to the tooth surface.

c. In reality, a certain amount of gingivoplasty is performed in most gingivectomy-type procedures, so the precise distinction between these two companion terms can be a bit cloudy.

5. There is still a limited place in modern dentistry for the gingivectomy procedure.
    a. Periodontal diseases can produce deformities of the gingiva, including conditions such as gingival enlargements, gingival craters, and gingival clefts.
    b. Occasionally, even in the absence of periodontal pockets these deformities occur and need to be altered to allow for improved esthetics, improved mastication, or enhanced ease of patient self-care.
6. **Figure 30-46** illustrates the types of incisions involved when performing a gingivectomy.

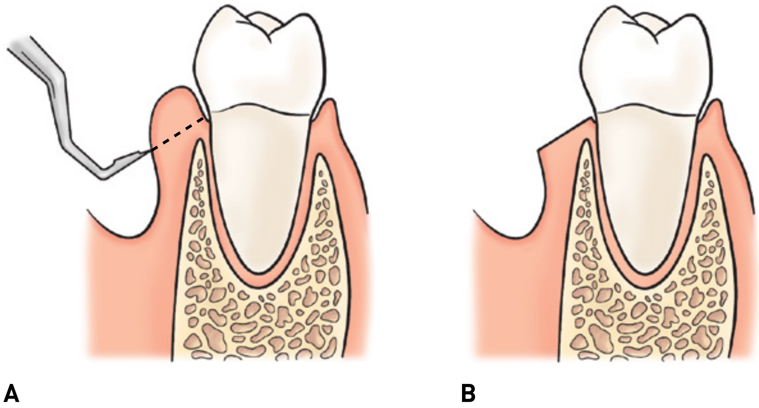

Figure 30-46 Gingivectomy Incisions. A. The placement of a special gingivectomy knife to incise the excess gingival tissue. Note that the direction of the tissue bevel being created is approximately 45 degrees to the tooth surface. This type of incision is known as an external bevel incision. **B.** The excess tissue has been excised and removed creating a more natural level and contour of the gingiva on the tooth surface.

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b. **Disadvantages of Gingivectomy**
    1. In modern periodontal therapy, the gingivectomy is usually limited to removing enlarged gingiva to improve esthetics or to allow for better access for self-care in isolated sites.
    2. Although gingivectomy reshapes enlarged gingiva (as might be seen in drug-influenced gingival enlargement or in hereditary gingival fibromatosis), periodontists have other more effective surgical options today.
    3. As a surgical technique, gingivectomy has several disadvantages:
        a. One disadvantage is that it leaves a large open wound that results in delayed healing compared to most other periodontal surgical procedures; this generally results in the expectation of more discomfort for the patient during the healing phase.
        b. **Figure 30-47** depicts the type of connective tissue wound that occurs following a gingivectomy.

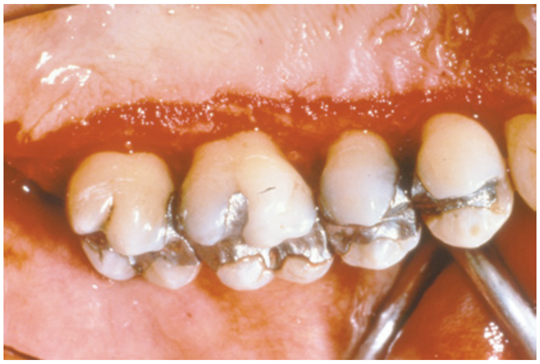

Figure 30-47 Connective Tissue Wound Created by Gingivectomy. Note that the gingivectomy results in a rather large wound that exposes connective tissue. This large wound usually results in protracted healing because the healing requires that the epithelium migrates across the wound created by the gingivectomy.

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        c. Another disadvantage is that it invariably results in a longer-appearing tooth because of the excision of some of the gingiva.
        d. A third disadvantage is that it does not provide access to the underlying alveolar bone. So, when access to the alveolar bone is needed, the surgeon must select another type of surgical approach.
        e. A fourth disadvantage is that it does not conserve keratinized tissue. In many surgical sites, it may be unwise to remove keratinized tissue because it may already be minimal in width.
        f. In spite of these disadvantages, the gingivectomy can be a useful surgical procedure in selected cases.
    c. **Steps in Performing a Typical Gingivectomy**
        1. Existing periodontal pockets are explored and the levels of the bases of the pockets are marked on the surface of the gingiva by punching a hole through the surface of the gingiva.
        2. Using special gingivectomy knives (both broad bladed and narrow bladed), gingiva is excised at the levels of the bases of the pockets.
        3. As the incision is made, care is taken to produce a 45-degree bevel of the gingiva against the tooth to mimic the natural contour of the surface of the gingiva in relationship to the tooth.
        4. The excised portion of the gingiva is removed (which also removes the soft tissue wall of existing periodontal pockets).
        5. The wound surface is inspected, remaining tissue tags are removed, and gingival contours are refined as needed.
        6. The tooth surfaces are inspected and debrided to remove biofilm, calculus, root contaminants, and root irregularities.
        7. The surgical wound is covered with periodontal dressing.
    d. **Healing Expected After a Gingivectomy**
        1. Healing occurs via secondary intention because an exposed connective tissue surface is always left following the procedure.
        2. The approximate rate that gingiva migrates across a connective tissue wound in the oral cavity is 0.5 mm each day. Because the gingivectomy normally leaves many millimeters of connective tissue exposed, the healing time can be protracted.
        3. The final healing of the wound created by a gingivectomy is a normal attachment of the epithelium and connective tissues to the tooth root at a level that is more apical in position than the original gingival level.
        4. Following a gingivectomy, the teeth in the surgical area will appear longer because more of the tooth is exposed following the procedure.
            a. If more tooth exposure is the intended result of the procedure, the outcome is acceptable.
            b. However, if more tooth exposure is not esthetically desirable, another surgical approach would be selected by the surgeon to compensate for the esthetic deficiency.
    e. **Special Considerations for the Dental Hygienist**
        1. As already mentioned, the gingivectomy wound leaves a broad connective tissue surface exposed that can be very uncomfortable for the patient during the healing phase and can be very unsightly.
        2. Postsurgical discomfort can be managed by placing a periodontal dressing over the wound to provide protection and by prescribing analgesics (pain medications) for use following surgery.
        3. At the time of the dressing removal at the first postsurgical visit following a gingivectomy, the dental hygienist will frequently need to replace the periodontal dressing to enhance wound comfort until total epithelialization of the wound has occurred.
        4. Healing of the wound created by a gingivectomy procedure progresses in a predictable manner.
            a. As already discussed, research has shown that oral epithelium grows across the exposed connective tissue at an approximate rate of 0.5 mm per day.
            b. Thus, it is possible for the clinical team to predict approximate healing times by estimating the wound size. This, of course, is useful when counseling patients about what to expect during the postsurgical phase.

16. **Dental Implant Placement.** Dental implants are discussed in Chapter 8. Dental implants are briefly mentioned in this section to provide an overview of the surgical aspects of implant dentistry. The planning and surgical placement of dental implants is quite complex, and the interested reader is directed toward the many excellent textbooks devoted to that topic.

a. **Procedure Description**
   1. A dental implant replaces a tooth root with a screw-shaped post that is surgically placed into the alveolar bone and is strong enough to hold a replacement crown or prosthesis (denture or bridge).
   2. Most conventional dental implants are categorized as endosseous implants, which are implants placed completely within the alveolar bone.
      a. Dental implant placement usually requires exposing alveolar bone using the principles of periodontal flap surgery, drilling a precise hole (termed the *implant osteotomy site*) in the alveolar bone, inserting a dental implant into the site, and then suturing the surgical site (**Figure 30-48**).

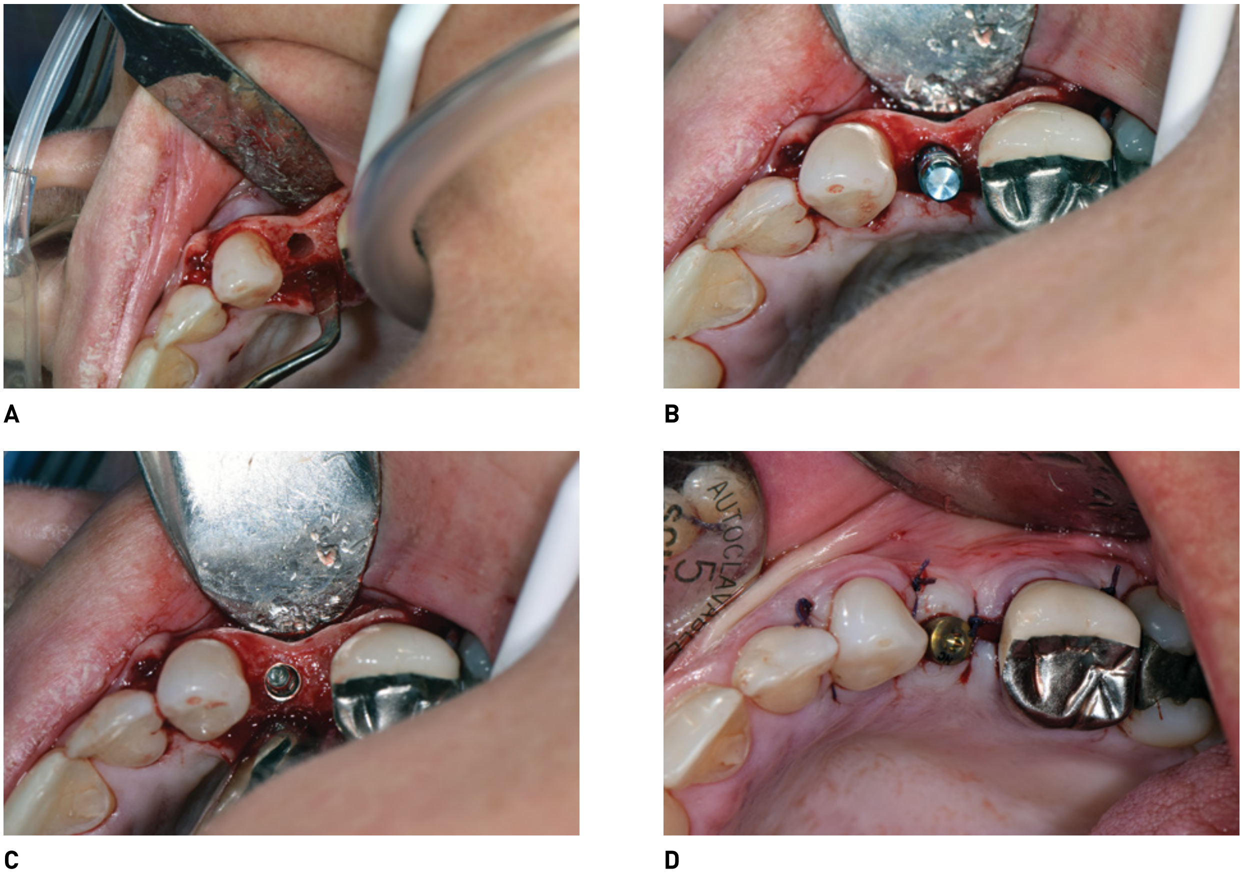

Figure 30-48 **Standard Protocol for the Surgical Placement of an Endosseous Dental Implant. A.** After a full-thickness flap is elevated, the implant osteotomy site is prepared using a surgical stent and series of implant drills. **B.** Osteotomy site alignment and parallelism with adjacent roots is checked with a guide pin. **C.** The implant is inserted into the osteotomy site until the head of the implant (collar) is flush with the alveolar bone. **D.** A healing abutment (or a cover screw) is secured to the top of the implant and the flaps are sutured together.

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      b. Implants may be placed immediately at the time of tooth extraction, known as immediate implant placement (**Figure 30-49**), or they may be placed several months after tooth extraction, known as delayed implant placement. Figure 30-47 is an example of delayed implant placement because the maxillary premolar was extracted several months prior to implant surgery. Many factors influence the decision to either immediately place the implant or delay the placement of the implant for a particular case, including site-specific factors (i.e., amount of available bone and location of anatomic structures), patient-specific factors (i.e., patient expectations and status of medical conditions), and technical factors (such as clinician's experience and overall interdisciplinary treatment plan goals). The decision to place the implant either immediately after tooth extraction or several months after tooth extraction is a balance between the aforementioned factors and the risks associated with complications to the patient.

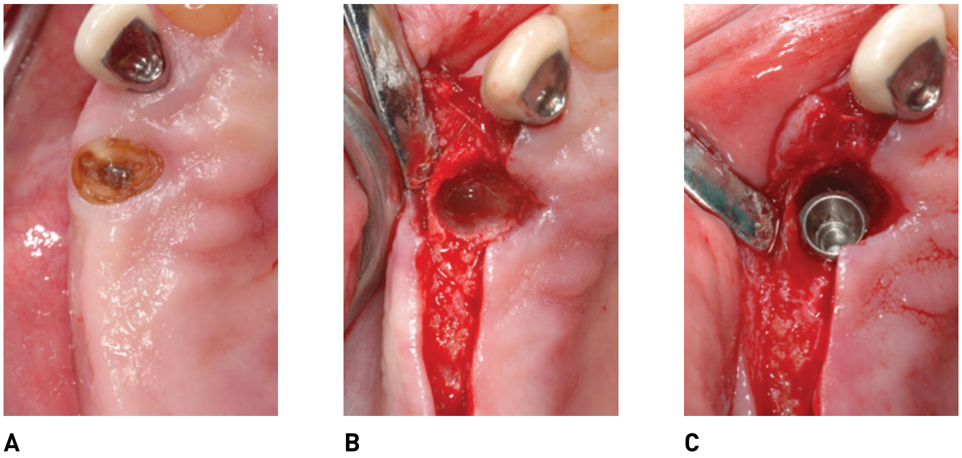

Figure 30-49 Immediate Implant Placement. A. Retained root that is deemed to be nonrestorable. **B.** Extraction of retained root. **C.** Placement of the endosseous implant immediately following extraction of the root.

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c. Additionally, conventional endosseous implants may be placed utilizing either a one-stage (nonsubmerged) or a two-stage (submerged) approach. In the one-stage surgical approach, the implant or abutment emerges from the soft tissue at the time of implant placement (**Figure 30-50**). In the two-stage surgical approach, the implant and cover screw are completely covered by the flap (**Figure 30-51B**). The two-stage surgical approach will require a second surgical procedure following osseointegration to expose the top of the implant in preparation of the restorative phase of the implant. This step is typically performed 3 months after implant placement to allow for sufficient time for osseointegration to take place.

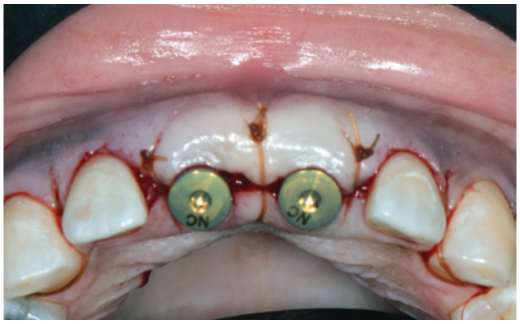

Figure 30-50 One-Stage (Nonsubmerged) Approach. In a one-stage implant protocol, the implant abutment emerges through the gingival tissues at the time of implant placement. Unlike a two-stage implant protocol, a one-stage approach does not require a second surgical procedure to uncover the top of the implant.

Courtesy of Dr. Hawra AlQallaf, Indianapolis, IN.

3. A variety of dental implants are available featuring various lengths, diameters, and designs.
4. If there is an inadequate amount of bone and/or keratinized gingiva, then any of the periodontal regenerative procedures previously discussed can be employed either prior to surgery or at the time of surgery (refer to the arrows in **Figure 30-51A**) to compensate for this deficiency.

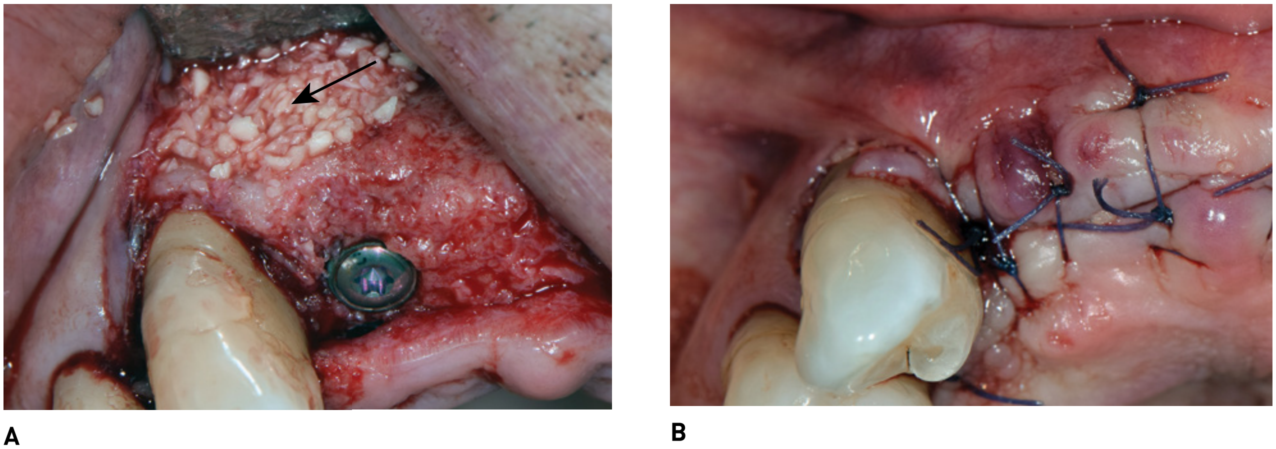

Figure 30-51 **Two-Stage (Submerged) Approach. A.** Surgical placement of implant with cover screw secured to the head of the implant. Note that the cover screw is flush with the alveolar crest to minimize the chance of exposure. Also note that bone graft material was placed at the time of surgery to minimize the effects of postsurgical resorption of the facial plate (black arrow). **B.** Flap closure. Primary closure of the flap is important to ensure that the implant and the bone graft material will remain completely submerged all throughout the healing process.

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b. **Healing Expected Following Dental Implant Placement**
   1. Healing following placement of a dental implant results in bone growth in such close proximity to the implant surface that the implant is stable enough to support a tooth-shaped restoration or a dental prosthetic appliance. Per-Ingvar Branemark (the father of modern dental implants) coined the term *osseointegration* to describe the direct structural and functional connection between living bone and the implant surface. Osseointegration is critical for implant stability and is a prerequisite for the long-term success of the dental implant.
   2. Although dental implants are not surrounded by cementum and do not have periodontal ligament attached to them (as seen in natural teeth), implants are still subject to the same inflammatory process that affects the supporting periodontal tissues around a natural tooth. If not arrested, the inflammatory process will lead to subsequent loss of supporting bone around the osseointegrated implant. This is termed *peri-implantitis*. The eventual outcome, if the peri-implantitis is not appropriately managed, is loss of osseointegration, which leads to implant failure. Because peri-implantitis jeopardizes implant survival, periodic monitoring of the peri-implant tissue, probing depth assessments, and radiographic evaluations are the best means to recognize early implant pathology before it becomes more advanced. Therefore, at each recall visit, it is very important for all members of the dental team to spend time and effort in carefully assessing the health of the implant and determine if interventional therapy is warranted (**Figure 30-52**).

c. **Special Considerations for the Dental Hygienist**
   1. Patient self-care following placement of a dental implant is as critical as it is following every periodontal surgical procedure, and the members of the dental team must assume responsibility for helping the patient with plaque control during the critical healing period.
   2. Once an implant site heals, the gingiva surrounding the implant can be maintained in health using self-care techniques similar to what is required to keep tissues around a natural tooth healthy.
   3. Implant maintenance and the role played by the dental hygienist are discussed in detail in Chapter 8.

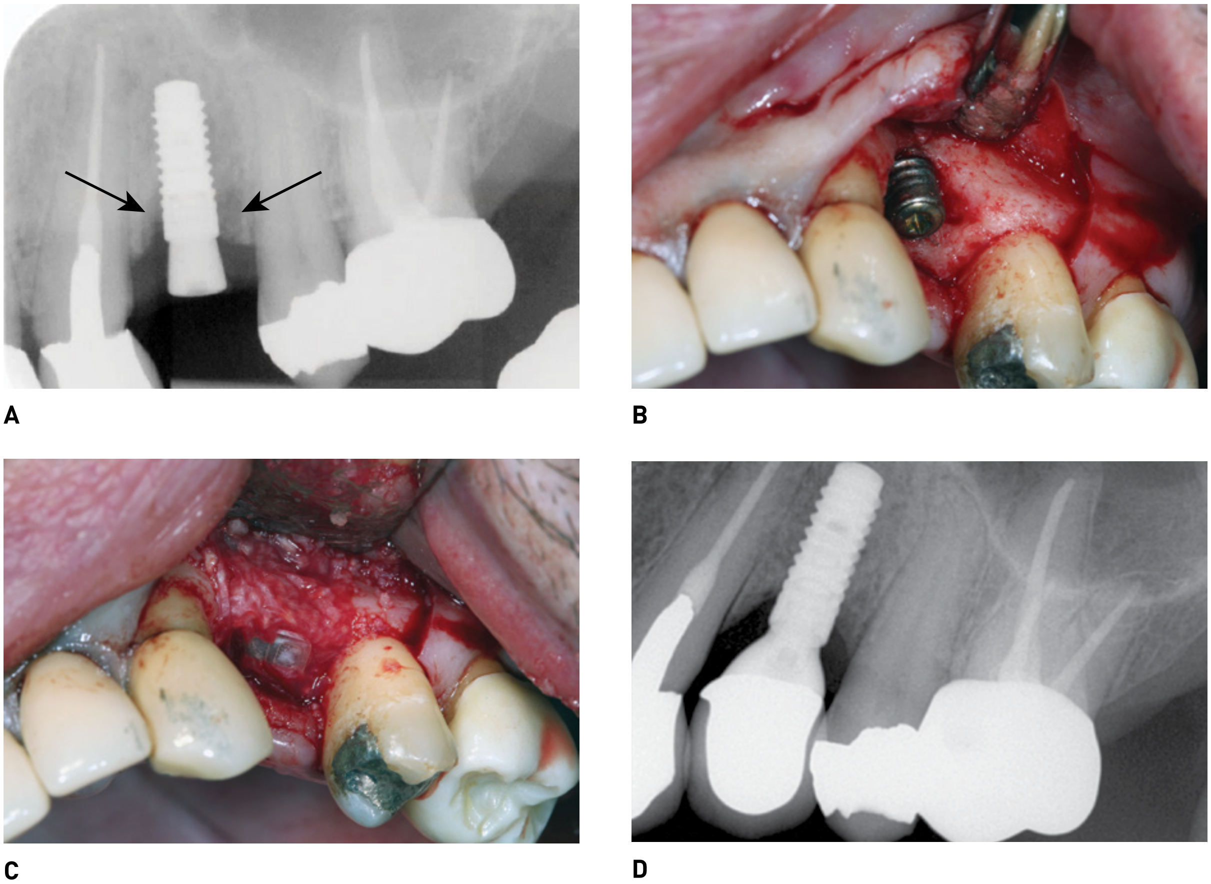

Figure 30-52 Surgical Intervention of Implant with Peri-implantitis. A. Dental radiograph showing alveolar bone loss around a dental implant at the time of periodontal recall visit. **B.** Flap elevated to reveal precise bone contours and to allow for treatment of exposed implant surfaces and grafting of bone defect. **C.** Graft and membrane placement prior to suturing. **D.** Radiograph taken 10 months following surgery showing bone fill.

Courtesy of Drs. Allison Marlow and Apoorv Goel, Indianapolis, IN.

17. **Periodontal Microsurgery. Periodontal microsurgery** is a term used to describe periodontal surgery performed with the aid of a surgical microscope. Principles of microsurgery have had a good deal of influence in medicine and will continue to influence the performance of certain periodontal surgical procedures, especially periodontal plastic surgery. Periodontal surgery performed using microsurgery techniques can result in procedures performed with increased precision on the part of the surgeon.
18. **Laser Therapy.** The use of lasers (Light Amplification by Stimulated Emission of Radiation) to focus a beam of light of a single wavelength at diseased sites has become an important topic in dentistry and periodontics.
    a. Lasers can incise and coagulate soft tissues with efficiency, and this fact makes the use of lasers during some surgical procedures, such as gingivectomy, gingivoplasty, biopsy of soft tissues, ablation of lesions, vestibuloplasty, and frenectomies, useful adjuncts. **Figures 30-53** and **30-54** show pre- and postsurgical cases that involved the use of laser therapy.
    b. Lasers of varying wavelengths have been proposed to be effective in treating periodontitis and peri-implantitis. It has also been proposed that lasers have the capability to stimulate periodontal regeneration. Some of the proposed benefits of laser therapy are (1) the laser beam is bactericidal against pigmented periodontal pathogens; (2) the laser can deliver a precise, focused beam of energy to the target site without collateral damage to the adjacent tissue; and (3) the laser has the ability to seal the pocket orifice with a "thermal fibrin clot," which creates a physical barrier to epithelial downgrowth. However, at this point, due to conflicting studies, it remains inconclusive if laser therapy can effectively and predictably treat periodontitis and induce true periodontal regeneration. Ongoing clinical studies are being conducted to settle this debate.

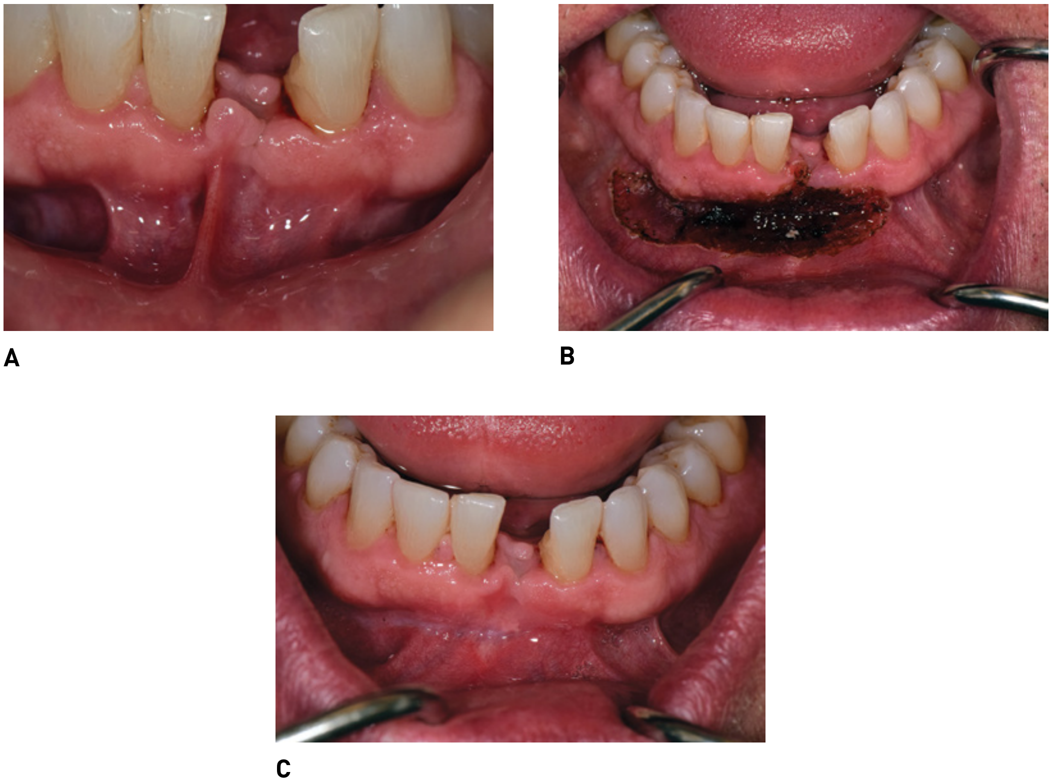

Figure 30-53 **Application of CO_2 Laser for a Frenectomy. A.** Clinical photograph showing excessive frenal pull in the mandibular anterior region. **B.** Laser incision to remove frenum and to perform vestibuloplasty. **C.** Follow-up photograph 10 days after surgery showing excellent healing and minimal inflammation.

Courtesy of Drs. Vidya Prabhu and Yusuke Hamada, Indianapolis, IN.

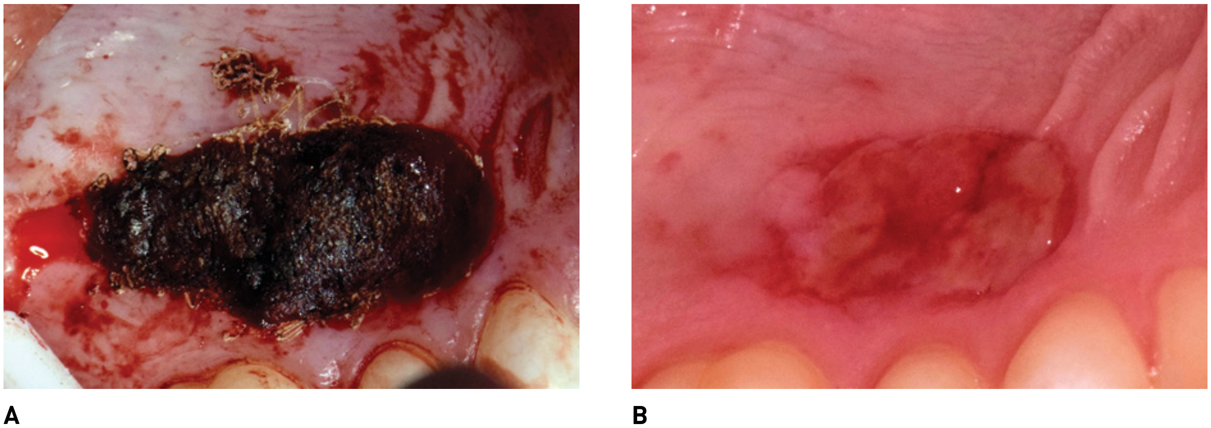

Figure 30-54 **Hemostasis at Free Soft Tissue Autograft Donor Site. A.** After obtaining a free soft tissue autograft from the palate, a CO_2 laser was set on the coagulation setting to gain hemostasis at the donor site. **B.** Seven days after the surgery, note the early signs of healing and re-epithelialization of the donor site.

Courtesy of Drs. Vidya Prabhu and Yusuke Hamada, Indianapolis, IN.

c. Current statements by the American Academy of Periodontology and the American Dental Association caution that there is insufficient evidence to support the use of lasers as a single form of treatment in periodontitis patients at this time.[33,34]

Section 4

Biological Enhancement of Surgical Outcomes

Many attempts have been made to enhance the outcomes of periodontal surgery by using chemical or biologic mediators to influence the healing following periodontal surgical procedures. This section provides a brief overview of some of the chemical and biologic mediators that have been studied. There is much ongoing research into this topic, and it is reasonable to expect that this ongoing research will shine more light on the fundamental mechanisms for enhancing periodontal regeneration.

1. **Root Surface Modification**
   a. **Mechanical Root Preparation**
      1. Many years of observation of the healing of periodontal surgical wounds have demonstrated that gingival tissues adjacent to tooth roots that have previously been exposed heal better when the roots are free of plaque biofilm, calculus, and root contaminants.
      2. These observations have led to the incorporation of mechanical root preparation to aid in regeneration. Hand and ultrasonic instruments have been used extensively for this purpose.
   b. **Chemical Biomodification Mediators.** Chemical biomodification mediators have been used to enhance gingival healing beyond what can be achieved by periodontal instrumentation alone. Several chemical biomodification mediators have been advocated for possible benefits to the gingival healing process.
      1. Ethylenediaminetetraacetic acid (EDTA) is a gel that is applied to treated root surfaces to modify (condition) the surface of the root following mechanical root preparation (**Figure 30-55**).

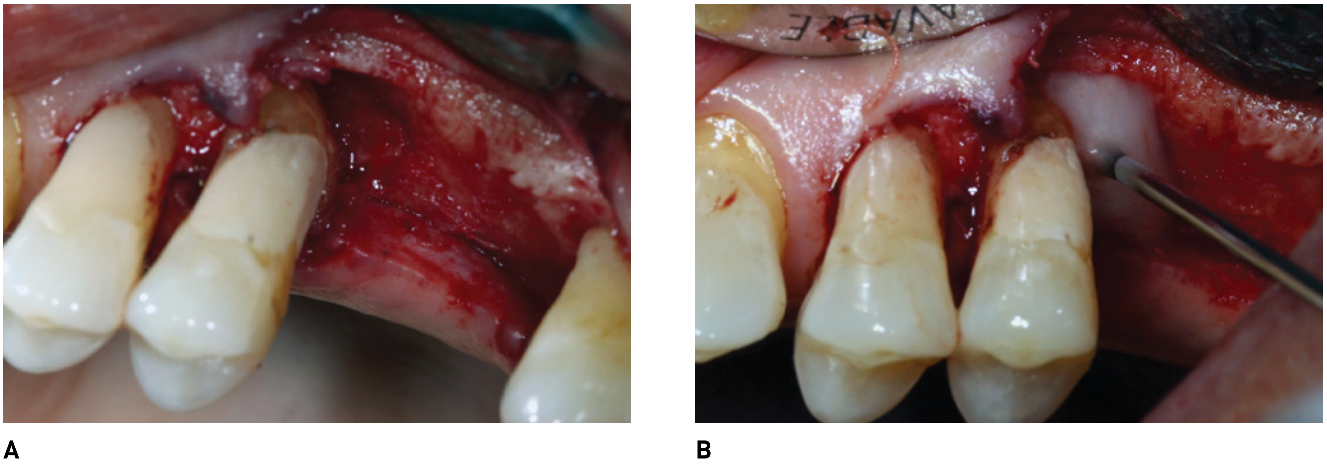

Figure 30-55 Application of EDTA to Treat an Intrabony Defect. A. An intrabony defect is exposed on the distal of the premolar. **B.** Following meticulous root debridement and degranulation of the intrabony defect, the treated root surface is "conditioned" by using EDTA. From a theoretical standpoint, EDTA "biomodifies" the root, thus facilitating attachment of connective tissue fibers to the treated root surface.

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         a. Possible benefits of using EDTA on roots include removal of the dentin smear layer, exposure of ends of embedded collagen fibers in remaining cemental surface, and removal of endotoxin buried deeper below the root surface.
         b. Although some clinicians have used EDTA to enhance root preparation, most of the evidence indicates that there is a questionable positive effect on the outcomes of any surgery by the use of this chemical to prepare tooth roots.
      2. A second chemical agent that has been referred to as a biologic mediator, again to enhance the outcomes of periodontal root preparation, is tetracycline (**Figure 30-56**).

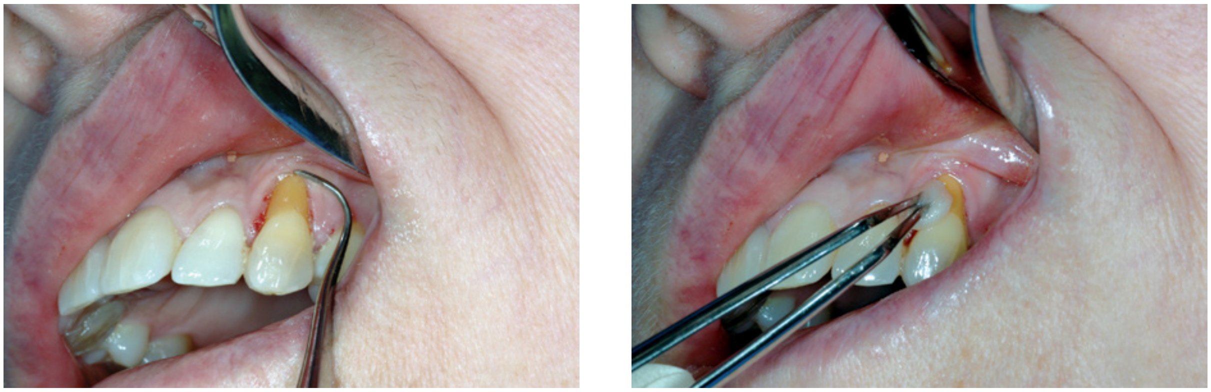

Figure 30-56 Application of Tetracycline. After meticulously instrumenting the exposed root surface, tetracycline is burnished onto the root surface to remove the smear layer and to selectively remove mineral from the dentin or cementum surface exposing a collagenous matrix.

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 a. Tetracyclines are a family of antibiotics with varied properties in addition to their antimicrobial effects. It has been suggested that tetracyclines applied to roots during surgery may enhance the migration of fibroblasts to the root surfaces during healing in addition to slightly decalcifying the root surfaces.
 b. Most studies indicate that using this biologic mediator on root surfaces during surgery has a questionable effect on the outcomes of the surgical procedures.
3. Both EDTA and tetracyclines are also known as root conditioning agents.

2. **Growth Factors.** Growth factors are naturally occurring proteins that regulate both cell growth and development. Several growth factors are being studied for their effect in enhancing the predictability of periodontal regeneration.[35,36] These growth factors include platelet-derived growth factor (PDGF) and insulin-derived growth factor (IGF). It is reasonable to anticipate that continued research into the use of growth factors might expand its application in periodontal regeneration.
3. **Enamel Matrix Derivative**
   a. **Periodontal Regeneration Factors**
      1. Periodontal regeneration depends upon the type of cells that first populate the periodontal surgical wound.
      2. As already discussed, using barrier materials to ensure that cells of the periodontal ligament enter the healing surgical wound without the premature interference of epithelial cells can result in more predictable regeneration outcomes.

   b. **Protein Preparations.** Research into using protein preparations and growth factors to enhance periodontal regeneration by mimicking natural healing processes has shown some promising results.[37–41]
      1. Enamel matrix derivative (EMD) is a Food and Drug Administration approved biologically-active gel that is applied to treated root surfaces to promote healing and regeneration through angiogenesis and osteogenesis.
      2. Enamel matrix derivative is a preparation of proteins extracted from porcine tooth buds. Enamel matrix derivative is a preparation of proteins extracted from porcine tooth buds mixed with a propylene glycol alginate carrier.
      3. Although it is unclear as to what the exact mechanism of action of enamel matrix derivative is, research has proposed several biological effects of enamel matrix derivative that may potentially promote periodontal regeneration: (1) increased attachment of periodontal ligament to the treated root surface, (2) increased differentiation of periodontal ligament stem cells into cementoblasts and osteoblasts, (3) decreased proliferation of epithelium (blocks the downgrowth of the long junctional epithelium), and (4) stimulation of the release of important growth factors, such as transforming growth factor beta (TGF-β).
      4. The major constituents of this extract appear to be proteins called amelogenins and enamelin.
      5. At this point, it appears that enamel matrix derivative may indeed enhance periodontal regeneration and that the effectiveness of this material is quite high when used in conjunction with different types of graft materials and/or barrier membranes (**Figure 30-57**).

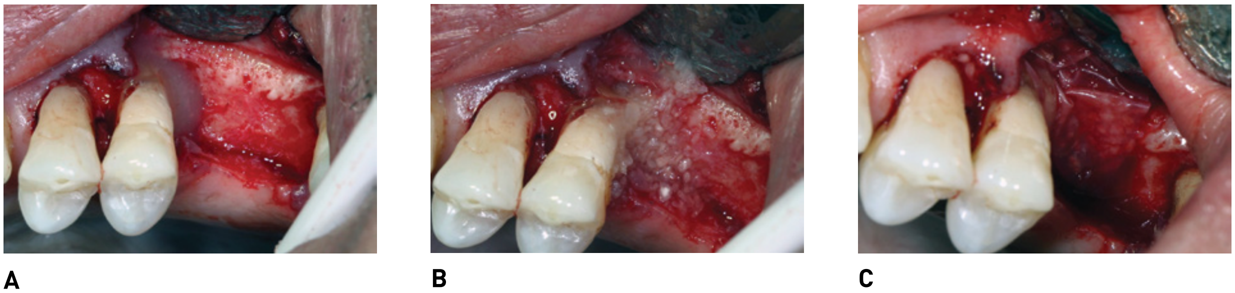

Figure 30-57 Application of EMD, Bone Graft, and Resorbable Membrane. This is a continuation of the case shown in Figure 30-54. **A.** After root conditioning with EDTA, EMD gel is placed in the defect. **B.** DFDBA (bone graft) is packed into the defect. **C.** A resorbable barrier membrane is placed to cover and contain the grafted site.

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6. Enamel matrix derivative is currently being used by clinicians in an attempt to improve outcomes of some types of periodontal surgery.
7. Studies into the precise constituents in this protein extract that can enhance healing are continuing.

4. **Platelet-Rich Plasma (PRP).** Another example of a biologic mediator is platelet-rich plasma. Platelet-rich plasma is classified as a first-generation platelet concentrate. The purported advantages of platelet-rich plasma are that (1) it is derived from an autogenous source (from the patient's own blood), thus eliminating concerns associated with disease transmission or immunogenic reactions, and (2) it is a practical source of growth factors needed for periodontal regeneration.[42,43]
   a. **Multistep Process for Platelet-Rich Plasma Development**
      1. Developing platelet-rich plasma is a multistep process (**Figure 30-58**). First, blood is drawn from the patient through a process known as venipuncture.

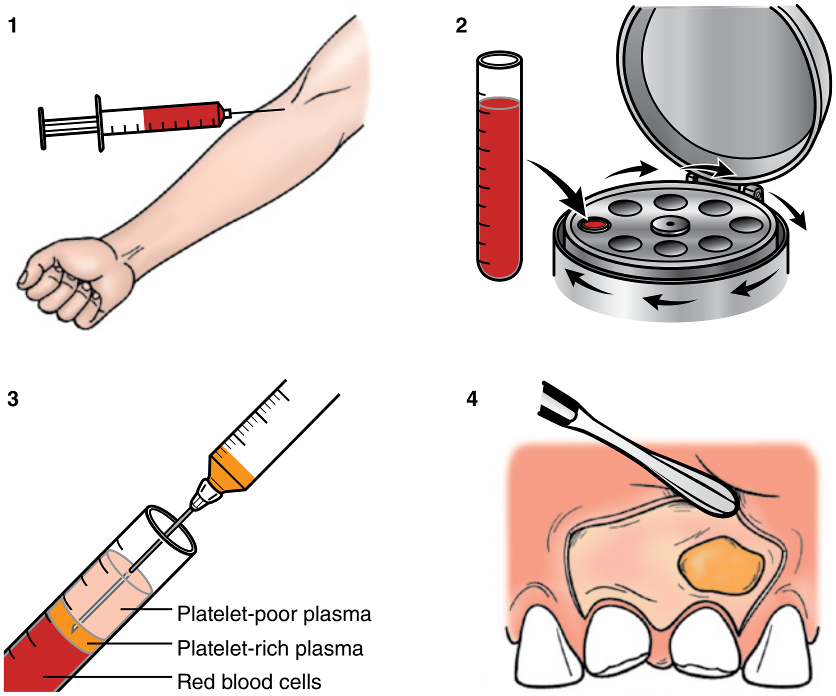

Figure 30-58 Process of Platelet-Rich Plasma Therapy. 1. Blood is first drawn from the patient's arm. **2.** The collected blood is then placed and spun in a centrifuge. The centrifuge separates the platelets from the rest of the blood components. **3.** PRP is extracted from the glass tube coated with an anticoagulant. **4.** After mixing the prepared PRP fraction with bovine thrombin and calcium, which activate the platelets, the PRP is now ready to be applied to the periodontal defect.

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        2. The blood sample is then mixed with an anticoagulant (to prevent spontaneous blood clot formation) and separated through a centrifugation process into three separate fractions: platelet-rich plasma, red blood cells, and platelet-poor plasma.
            a. The red blood cells and platelet-poor plasma fractions have no clinical benefit and are therefore discarded.
            b. The platelet-rich plasma fraction, in contrast, is a rich source of platelets which produce and release a cocktail of multiple growth factors that work synergistically to initiate the wound healing cascade and enhance the regenerative process.
        3. Next, the platelet-rich plasma fraction is mixed with bovine thrombin and calcium to biochemically activate the platelets so that they degranulate and release their growth factors.
        4. Finally, the platelet-rich plasma is ready to be delivered to the surgical site via an applicator.
    b. **Research on the Regenerative Role of Platelet-Rich Plasma**
        1. Although some studies indicate a potential enhancement of the healing process following the use of this preparation, it is still unclear if the growth factors are present in high enough concentrations to have much effect on the actual surgical outcomes. Additional studies of this material are underway.
        2. Furthermore, platelet-rich plasma has several undesirable adverse effects that can pose problems. First, the platelet-rich plasma fraction contains anticoagulants that block the full coagulation cascade, which plays a critical role in tissue wound healing. Second, platelet-rich plasma is mixed with bovine thrombin, which can induce an allergic reaction in individuals sensitive to bovine materials.

5. **Platelet-Rich Fibrin (PRF).** Platelet-rich fibrin is a second-generation platelet concentrate that was first developed in France in 2001 by Choukroun et al. Like platelet-rich plasma, platelet-rich fibrin is obtained from the patient's own blood and subjected to centrifugation. However, platelet-rich plasma and platelet-rich fibrin have several key differences.
    a. First, anticoagulants and bovine thrombin are not added to platelet-rich fibrin, thereby avoiding the potential adverse effects associated with platelet-rich plasma. Collecting blood without anticoagulants allows for the natural coagulation process to take place so that the desired platelet fibrin clot forms.
    b. Second, platelet-rich fibrin is a three-dimensional fibrin matrix that can be used as a scaffold for a variety of procedures, including serving the function of a barrier membrane in guided bone regeneration and guided tissue regeneration procedures (**Figure 30-59**).

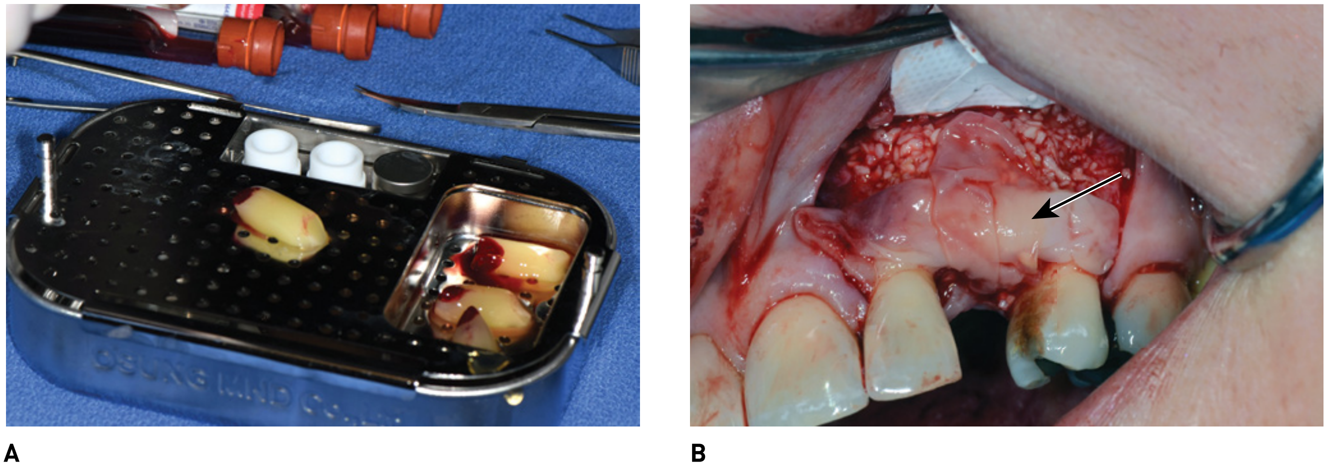

Figure 30-59 Platelet-Rich Fibrin Therapy for a Periodontal Defect. A. After centrifugation, the PRF clot is removed from the test tube using sterile tweezers and placed on a sterile metal tray. Each PRF clot releases its serum (exudate) and is ready for compression into the barrier membrane. **B.** The PRF clot (arrow) can be compressed and molded to form a strong fibrin matrix that covers the bone graft and serves as the barrier membrane. The fibrin matrix is remodeled in a way comparable with a natural clot and does not dissolve rapidly after application.

Courtesy of Drs. Jennifer Chang and Yusuke Hamada, Indianapolis, IN.

   c. Third, platelet-rich fibrin has been demonstrated to contain even higher levels of growth factors over an extended period of time compared to platelet-rich plasma.

6. **Bone Morphogenetic Proteins (BMP).** Bone morphogenetic proteins are a group of regulatory glycoproteins that have been studied for possible use in the field of periodontal regeneration because of their known osteoinductive effects. Both purified and recombinant BMPs are currently being studied, and early results indicate possible enhancement of regeneration in some treated sites. Unfortunately, using BMPs to enhance surgical outcomes carries the risk of tooth ankylosis and much further study of this material is indicated. Investigations into the use of BMPs to enhance periodontal regeneration are continuing.

Section 5

Patient Management Following Periodontal Surgery

The dental hygienist plays a critical role in managing patients following periodontal surgery. This section discusses components of postsurgical management, including use of sutures, use of periodontal dressings, delivery of postsurgical instructions, and organizing postsurgical visits. It is important to realize that the management of the patient during the healing phase following periodontal surgery can be as important to the surgical outcomes as the skill of the surgeon performing the surgery.

Use of Sutures in Periodontal Surgical Wounds

1. **Overview of the Use of Sutures in Periodontal Wounds.** Many periodontal surgical procedures require the placement of sutures to stabilize and secure the position of the soft tissues during the early phases of healing; a suture, or "stitch" as it is sometimes called, is a material placed by a surgeon to hold tissues together during healing.
   a. **Characteristics of Suture Material**
      1. The ideal suture material is biocompatible, nontoxic, flexible, and strong.
      2. Sutures are classified as either multifilament or monofilament. A multifilament suture, such as black silk suture, is composed of smaller strands that are braided or twisted together, whereas a monofilament suture, such as a polypropylene suture or nylon suture, is composed of a single strand. One undesirable property of multifilament sutures is their capacity to absorb and retain fluid and/or bacteria within the suture fibers (i.e., bacteria can accumulate in between the multiple smaller strands and travel down the entire length of the suture to contaminate the surgical wound). This effect, called "wicking," may have a negative effect on surgical outcomes because it triggers an inflammatory reaction. In contrast, wicking is rarely associated with a monofilament suture because it is composed of a single strand.
      3. When placing sutures, the surgeon takes great care not to put tension on a flap with sutures (i.e., to ensure that the flap lies passively at the intended position prior to suturing); sutures are used to stabilize the flap in its passive position.
      4. If a suture places tension on a flap, the suture material will pull out of the tissues during healing and will fail to serve the purpose of stabilizing the tissues. Another adverse consequence of suturing the flap with tension is that it may traumatize the soft tissue and compromise vascularization to the flap. This will ultimately result in flap necrosis and compromise surgical outcomes. Tension-free closure is especially important when suturing fine, delicate gingival tissues, such as the papilla.
   b. **Types of Suture Material.** In general, two types of suture material are used: nonabsorbable and absorbable. **Box 30-7** provides an overview of some of the suture materials available for use in periodontal wounds.
      1. **Nonabsorbable suture**, also referred to as nonresorbable suture, is a suture made from a material that does not dissolve in body fluids. A clinician must remove the nonabsorbable sutures after some healing of the wound has occurred.
      2. **Absorbable suture**, also referred to as resorbable suture, is a suture made from a material designed to dissolve harmlessly in body fluids over time; though absorbable sutures do not normally require removal by the dental team, some absorbable sutures do not dissolve particularly well in saliva.

Box 30-7 Examples of Suture Materials

Nonabsorbable
- Braided silk
- Monofilament nylon
- Polytetrafluoroethylene
- Braided polyester
- Polypropylene

Absorbable
- Plain and chromic gut
- Polyglactin 910 (Coated Vicryl)
- Poliglecaprone 25 (Monocryl)

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2. **Suture Placement Techniques.** Familiarity with some general techniques for suturing can guide the hygienist assigned to the task of suture removal at a postsurgical appointment. **Box 30-8** provides an overview of general indications for some of the more common suturing techniques for periodontal surgical wounds. Three of the most common suture placement techniques are discussed and illustrated below.

Box 30-8 Overview of General Indications for Common Suture Techniques

1. Interrupted suture: closure of vertical incisions, closure of nondisplaced flaps
2. Sling suture: closure of displaced flaps
3. Continuous sling suture: closure of displaced flaps, closure of nondisplaced flaps

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a. **Interrupted Interdental Suture**
   1. In most periodontal flap surgeries, flaps are elevated on both the facial and lingual (or palatal) surfaces of the teeth. At the completion of the surgery, an interrupted interdental suture is typically used to suture the facial and lingual papillae together. This technique is also ideal for closing vertical incisions.
   2. When interrupted interdental sutures are placed to close a periodontal flap that involves several teeth, a separate suture is placed and tied in each of the interdental sites. The interrupted suture ensures that the flap remains closed in the event that one suture fails.
   3. An interrupted interdental suture that might be utilized during a periodontal flap for access procedure is illustrated in **Figures 30-60** and **30-61**.

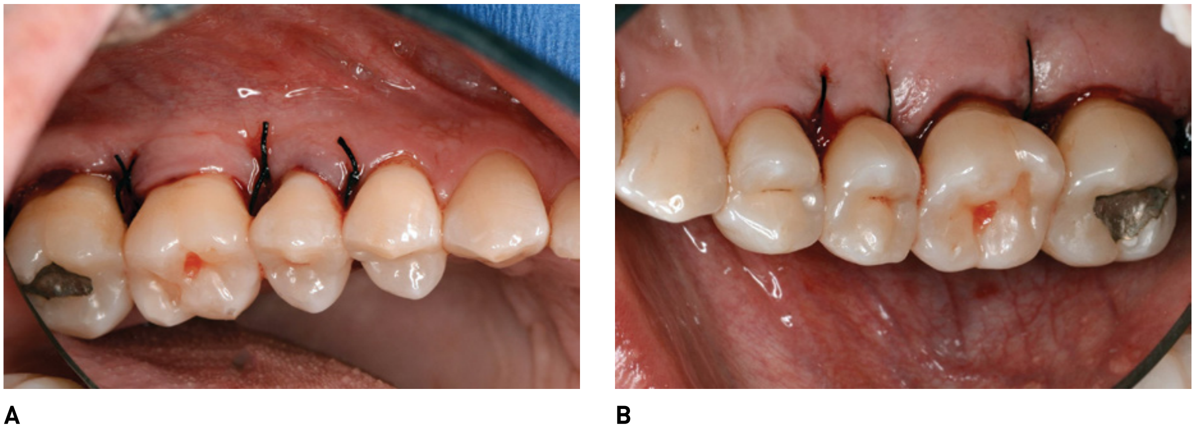

Figure 30-60 Interrupted Interdental Sutures. A. Buccal view. **B.** Accompanying palatal view. Observe that each papilla is closed up by an individual suture that is not connected to the neighboring sutures. Each individual suture has its own independent knot that will need to be cut prior to removal.

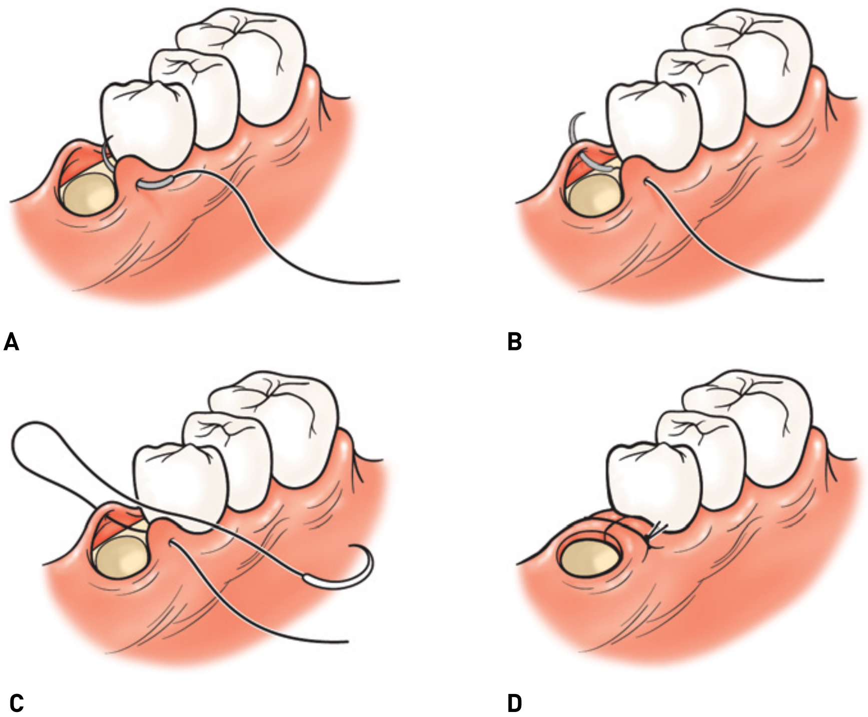

Figure 30-61 Interrupted Interdental Suture. In these drawings, the most mesial tooth has been removed to allow for visualization of the path of the interrupted interdental suture. **A.** Suture is drawn through facial papilla. **B.** Suture is then drawn through the lingual papilla. **C.** Suture passes passes back to facial side, without re-engaging the papillae. **D.** Suture knot tied on the facial side.

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4. When removing interrupted interdental sutures, the clinician must cut each of the interdental sutures prior to pulling out the suture material.

b. **Continuous Sling Suture**

1. The continuous sling suture is preferred by many clinicians for suturing many types of periodontal flaps.
2. The continuous sling suture engages only the facial papillae (without engaging the lingual papillae), then loops around each tooth to engage the adjacent facial interdental site; the suture is tied following placement of the loop around the terminal tooth.
3. A continuous sling suture that might be utilized during an apically positioned flap is illustrated in **Figures 30-62** and **30-63**.

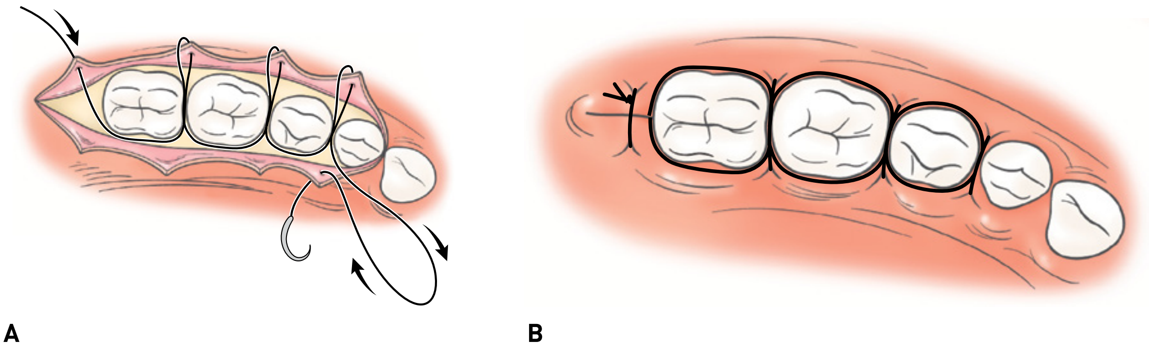

Figure 30-62 Continuous Sling Suture. Note that the suture loops around the cervical areas of each tooth, then passes through the tissues associated with each interdental area (**A**). The suture knot is located distal to the most terminal tooth (**B**).

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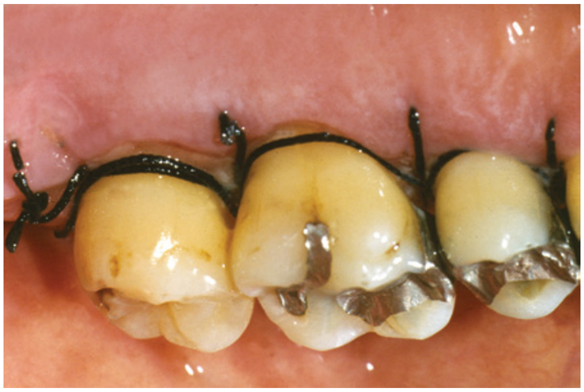

Figure 30-63 **Continuous Sling Suture.** Continuous loop suture on the maxillary arch. Note that only a single knot is tied distal to the most terminal tooth.

Courtesy of Dr. Don Rolfs, Periodontal Foundations, Wenatchee, WA.

4. Removal of a continuous sling suture can frequently be accomplished with a single cut through the suture near the knot prior to pulling out the suture material.

c. **Sling Suture**

1. Some periodontal surgical wounds require the placement of a sling suture; the sling suture is used to sling or suspend the tissues around the cervical area of a tooth rather than to tie soft tissue to other soft tissue.
2. The sling suture is frequently used when a flap is displaced in an apical direction. **Figure 30-64** illustrates a sling suture.

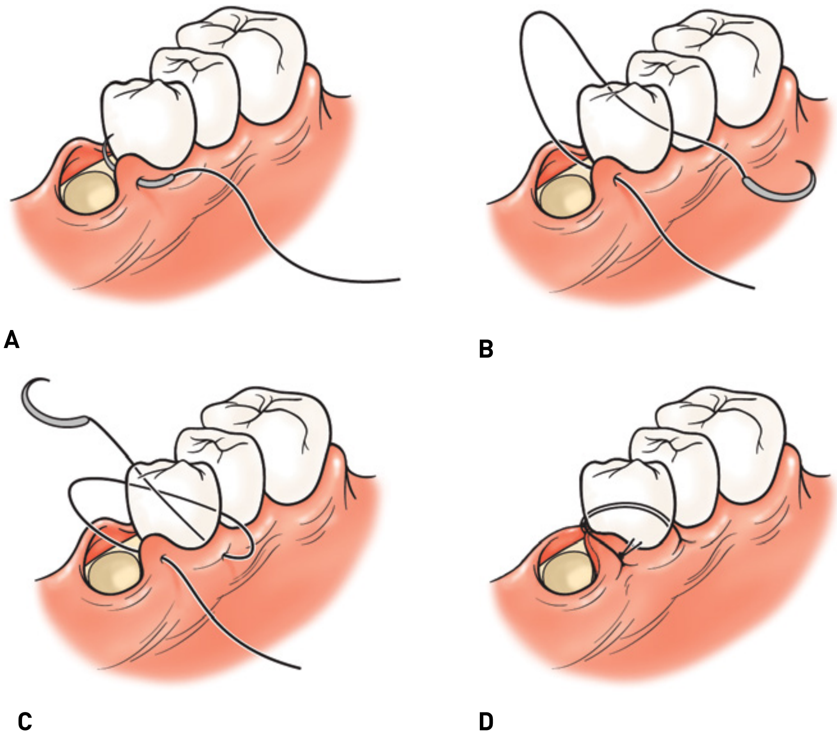

Figure 30-64 **Sling Suture.** In these drawings, the most mesial tooth has been removed to allow for visualization of the path of the sling suture. **A.** Suture is drawn through the facial papilla on the mesial side of the tooth. **B.** Suture loops around the lingual surface of the tooth without engaging the lingual papilla. **C.** Suture engages the next facial papilla on the distal side of the tooth. **D.** Suture loops back around the tooth, then tied where the suture started.

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        3. Note that when facial and lingual flaps are sutured using the sling suture technique a separate sling suture must be placed on both the facial and lingual surfaces.
        4. Removal of the sling suture only requires locating the individual knots, cutting the suture near the knot, and careful extraction of the suture.

3. **Suture Removal**
    a. **Removal of Sutures**
        1. Nonabsorbable sutures placed during surgical procedures are removed as part of routine postsurgical visits. Frequently, remnants of absorbable sutures can also be removed at the routine postsurgical visits to avoid unnecessary tissue inflammation that can occur if the retained absorbable suture is not dissolved in a timely manner.
        2. Guidelines for timing of removal vary. But, in general, sutures should be removed when wound healing has progressed to the point at which the sutures are no longer needed to stabilize the tissues. Many sutures are loose and no longer needed to stabilize the tissues at the time of the 1-week postsurgical visit.
        3. Most periodontal sutures should not be left in place longer than 2 weeks because they can act as plaque retentive irritants if the suture material remains in the tissues too long. It should be noted that sutures in some periodontal cases are routinely left in place for much longer periods.
        4. Each periodontal surgical procedure is unique, and removal procedures can vary. Some general guidelines for suture removal are outlined below in **Box 30-9**.

Box 30-9 General Guidelines for Suture Removal

Guideline 1: Remove sutures in a timely manner.
Guideline 2: Read the surgical note in the patient's chart.
Guideline 3: Understand the typical sizing system for sutures.
Guideline 4: Never allow the knot to be pulled through the tissues.
Guideline 5: Always visually confirm that all sutures have been removed.

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    b. **General Guidelines for Suture Removal**
        1. **Guideline 1: Remove sutures in a timely manner.** Nonabsorbable sutures are generally removed after 1 week of healing; most absorbable sutures can be left in place 1 to 3 weeks.
        2. **Guideline 2: Read the surgical note in the patient's chart prior to suture removal.**
            a. The number and type of sutures placed should be a routine part of the chart entry recorded for each periodontal surgery.
            b. Knowing the number of sutures placed during the actual surgical procedure can help the dental hygienist confirm that all sutures have been located and removed during a postsurgical visit.
        3. **Guideline 3: Understand the typical sizing system used for periodontal sutures.**
            a. Although there are numerous sizes of sutures used in a medical setting, typical designations for suture sizes used in periodontal surgery are sizes 3-0, 4-0, and 5-0.
                1. In this sizing system, the 3-0 size is larger than the 4-0 size, and 4-0 is larger than 5-0.
                2. In the mouth, 5-0 can be more difficult to locate than a 4-0 size, especially in the posterior part of the mouth.
            b. The dental hygienist should learn the precise abbreviations used in the chart entries in the individual clinical setting. A typical example of an abbreviation would be "4-0 BSS." This would mean the size of the suture is 4-0 while BSS stands for black silk suture, a commonly used nonabsorbable suture material.
            c. **Table 30-5** outlines designations for typical suture sizes used during periodontal surgery as they might appear in a patient's chart.

Table 30-5 Typical Designations for Suture Sizes Used in Periodontal Surgery

Suture Size	Approximate Diameter of Suture
3-0	0.20 mm
4-0	0.15 mm
5-0	0.10 mm

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4. **Guideline 4: Never allow the knot to be pulled through the tissues.**
    a. Grasp the knot with sterile tissue forceps.
    b. Use scissors to cut the sutures under the knot as close as possible to the tissue. This should be done to avoid passage of the plaque-contaminated outer portion of the suture back through the tissue.
    c. Gently pull the suture with the sterile forceps away from the tissue.
        1. When the suture is gently pulled from the tissue, care should be taken not to force the knot itself through the tissue. This technique is illustrated in **Figure 30-65**.

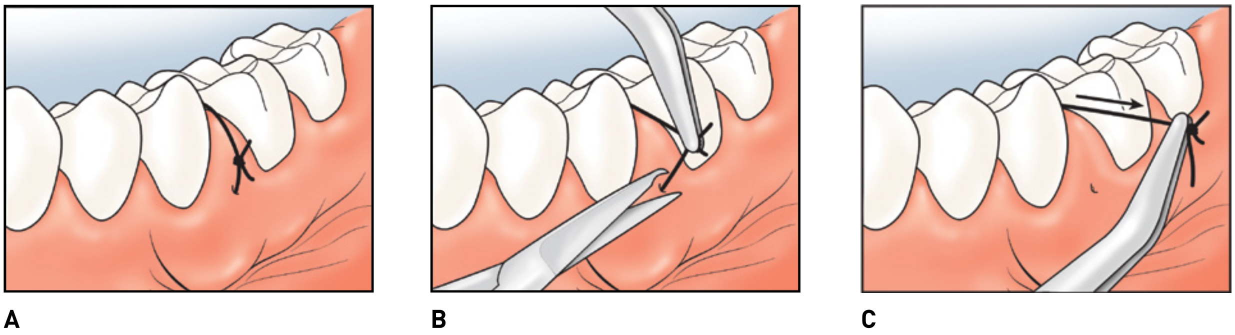

Figure 30-65 **Suture Removal. A.** Suture in place. **B.** Grasp the suture material with forceps and cut under the knot as close as possible to the tissue. **C.** Pull the suture material gently from the tissue taking care not to pull the knot through the tissue.

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        2. It should be noted that suture removal is rarely painful for the patient if care is taken not to create unnecessary tissue movement or tissue trauma.
5. **Guideline 5: Always visually confirm that all sutures have been removed.**
    a. Following suture removal, it is imperative to visually inspect the wound with care to ensure that all sutures have indeed been located and removed.
    b. Remember that the patient chart entry made on the day of the surgery usually will contain information about how many sutures were actually placed.

Use of Periodontal Dressing

1. **Purpose of Periodontal Dressing**
    a. **Periodontal dressing**, or periodontal pack as it sometimes called, is a protective material applied over a periodontal surgical wound (**Figure 30-66**). Periodontal dressings are used somewhat like a bandage covering a finger wound. Periodontal dressing provides mechanical protection for the surgical wound and therefore facilitates healing, enhances patient comfort by isolating the area from external irritations or injuries, prevents postsurgical bleeding by maintaining the initial clot in place, and maintains a debris-free area of the protected site.

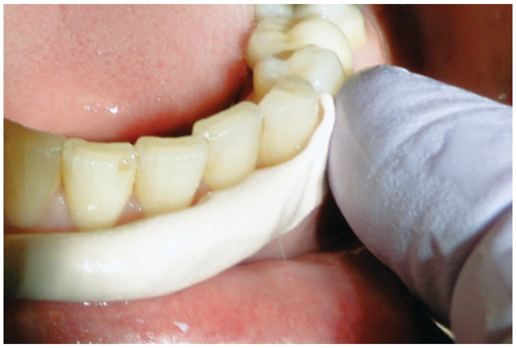

Figure 30-66 **Periodontal Dressing.** Periodontal dressing is placed over a periodontal surgical site. Note that gentle finger pressure is applied to mechanically push the pack around the necks of the teeth so that it interlocks around each tooth in the interproximal areas.

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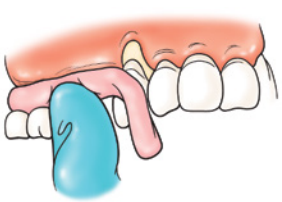
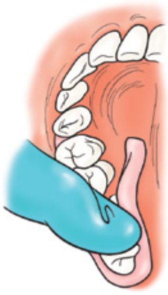
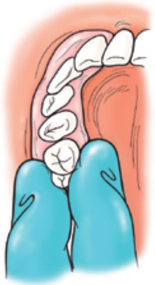
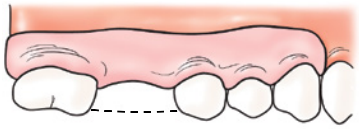
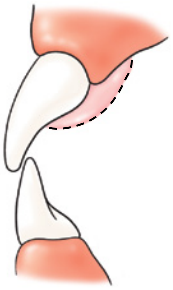
- b. Although the placement of periodontal dressings following periodontal surgery used to be routine, modern surgical techniques may or may not require placement of a periodontal dressing.
    1. The surgical wound created by the gingivectomy procedure leaves a raw connective tissue surface exposed that always requires a periodontal dressing.
    2. Periodontal flaps that are well adapted to the alveolar bone and tooth roots may not always require a periodontal dressing.
    3. Periodontal dressings can be placed to facilitate flap adaptation and are frequently indicated when the surgical procedures have created varying tissue levels or when displaced flaps are used.
    4. The periodontist will determine the need for dressing placement at the time of the surgical procedure.

2. **General Guidelines for Management of Periodontal Dressings**
    - a. **Proper Placement**
        1. Periodontal dressing is retained primarily by pushing some of the material into the embrasure spaces to lock the dressing around the necks of the teeth mechanically.
        2. Using less periodontal dressing is better than using more of the material during placement; the proper amount of dressing is only enough to cover the wound. The dressing should be placed so that there is no contact between the dressing and the teeth in the opposing arch when the patient bites down; occlusal contact with teeth in the opposing arch will quickly dislodge the dressing. **Table 30-6** and **Figures 30-67A-E** illustrate the proper placement of a periodontal dressing.

Table 30-6 **Steps in Periodontal Dressing Placement**

 **Figure 30-67A** © Wolters Kluwer	Dressing is pressed into the interdental spaces with gentle finger pressure on the facial.
 **Figure 30-67B** © Wolters Kluwer	Dressing is looped around the most distal tooth and pressed into the interdental spaces on the palatal.

 **Figure 30-67C** © Wolters Kluwer	Gentle finger pressure is continued against the dressing to join it interdentally on the facial and palatal aspects.
 **Figure 30-67D** © Wolters Kluwer	Dressing can be bridged across edentulous areas.
 **Figure 30-67E** © Wolters Kluwer	Dressing amount should be minimal to avoid contact of the dressing with the teeth in the opposite arch.

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    3. Periodontal dressings should be replaced every 5 to 7 days until the surgical wound is healed enough to be exposed. It should be noted that suture material could accidentally become trapped within the periodontal dressing. When removing dressings, it may be necessary to loosen the dressing slightly and cut the suture before completely removing the dressing from the necks of the teeth.

3. **Types of Periodontal Dressing.** Two types of modern periodontal dressings are commonly available for use today. Both types are held in place primarily by mechanical retention around the necks of the teeth.
   a. **Chemical Cure Paste.** One type of periodontal dressing is a two-paste chemical cure material that requires the mixing of paste from two tubes to form a dressing with a putty-like consistency that has a soft texture but still has enough flexibility to facilitate its placement and adaptation over the surgical wound area.
      1. This type usually contains zinc oxide, mineral oils, and rosin plus a bacteriostatic or fungicidal agent.
      2. Mixing of these two-paste dressings is either by hand (**Figure 30-68**) or in an auto-mix cartridge.
      3. Examples of two-paste dressings are Coe-Pak manufactured by GC America, Inc. and PerioCare manufactured by Pulpdent Corp.

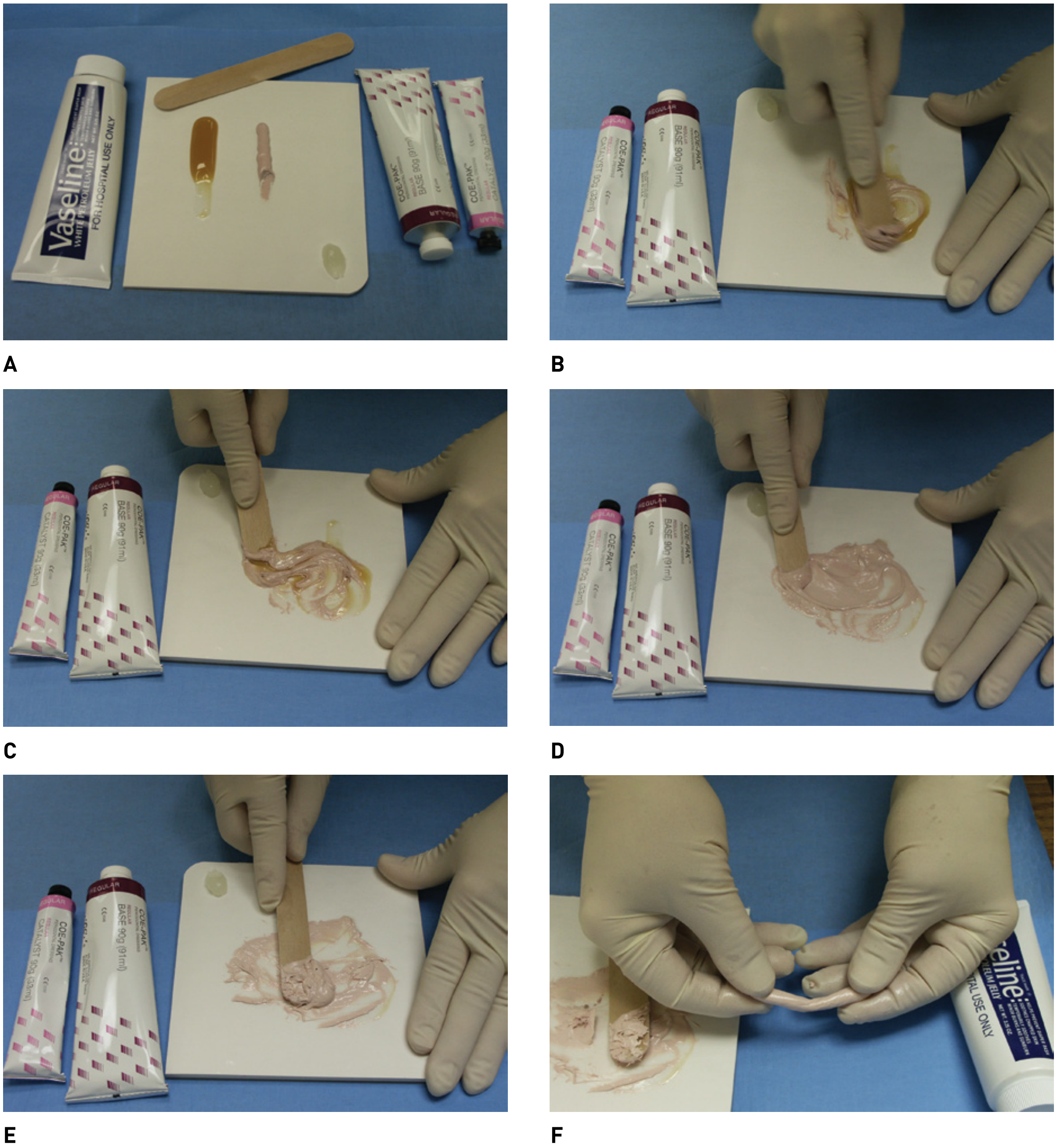

Figure 30-68 **Two-Tube Chemical Cure Paste. A.** Equal lengths of paste from the catalyst and the base tube are extruded onto the mixing pad. **B–E.** A wooden tongue depressor (or a metal spatula) is used to mix the paste until a thick consistency and uniform color is reached. The setting time can be altered by adding a few drops of warm water during mixing or by immersing the pack into a bowl of warm water just after mixing. **F.** Once the paste loses its tackiness, it can be handled and molded using gloves lubricated with water or petroleum. The pack is then formed into uniform pencil-sized rolls that have a smooth consistency.

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b. **Light-Cured Paste.** A second type is a light-cured gel that contains polyether urethane dimethacrylate resin (**Figure 30-69**).
   1. The dental hygienist must study the manufacturer's instructions with care and must practice placement of the dressing on a typodont (model) before using it in a patient's mouth.
   2. This type of dressing is available as a clear, translucent material that is preferred for use by some clinicians in esthetic areas of the dentition.
   3. An example of a light-cured gel periodontal dressing is Barricaid VLC periodontal surgical dressing manufactured by Dentsply Caulk Co.

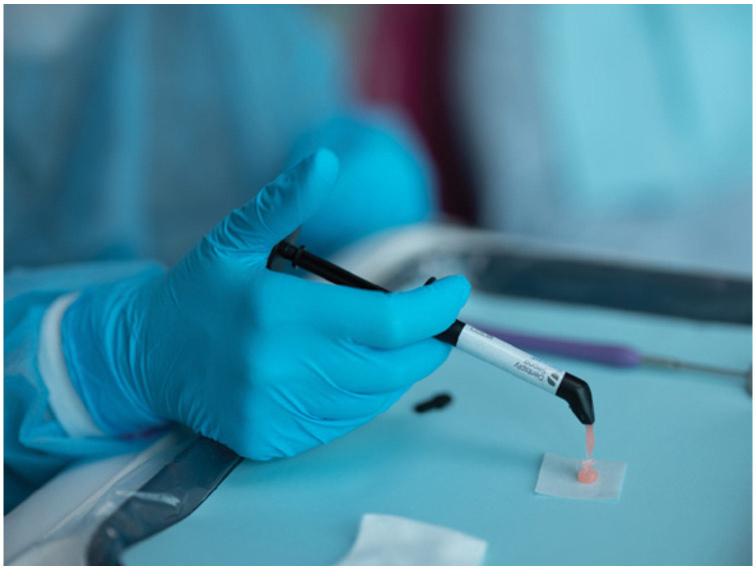

Figure 30-69 Light-Cured Dressing Material. Barricaid by Dentsply is an example of a periodontal dressing available in prefilled syringes. The dressing is applied over the surgical site and light-cured to form an elastic protective covering that is pink, tasteless, and allows for superior esthetics. Unlike chemical-cure pastes, light-cured dressing material eliminates the time-consuming process of mixing pastes together.

Courtesy of Dentsply International.

Postsurgical Instructions and Follow-up Visits

A member of the dental team should provide postsurgical instructions to the patient following periodontal surgery. Usually, the patient is provided with both written and verbal instructions to minimize confusion and to maximize compliance. Typical postsurgical instructions are outlined in **Box 30-10**. For patients who require sedation, the companion who has accompanied the patient to the office is included when postsurgical instructions are given.

Box 30-10 Typical Postsurgical Instructions

1. If you have questions or concerns, call the office or the office emergency number right away. Office: 555-1111; emergency 555-2222.
2. Do take medications as prescribed. Report any problems with the medications immediately.
3. Do take it easy for several days. Limit your activity to mild physical exertion.
4. Expect some bleeding following the procedure. If heavy bleeding persists, call the office emergency number.
5. Expect some swelling. Intermittent use of an ice pack on the face in the area of the surgery during the first 8 to 10 hours following surgery can minimize swelling.
6. **Diet Recommendations**
   a. Soft food only on the day of the surgery.
   b. No hot beverages on the day of surgery.
   c. Avoid chewing on the surgical site.
7. **Oral Self-Care**
   a. Rinse with recommended mouth rinse starting the day after surgery.
   b. If dressing was placed, it may also be brushed lightly.

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1. **Postsurgical Instructions to the Patient**
    - a. **Restrictions on Self-Care.** Most periodontal surgical procedures require some restrictions on self-care during the early phase of healing.
        1. It is common practice to prescribe 0.12% chlorhexidine mouth rinse to be used twice daily to aid with self-care until the patient can safely resume mechanical plaque control.
        2. In most cases following any periodontal surgery, manual self-care can be resumed by the patient in 10 to 14 days.
        3. For selected surgical procedures (such as GTR or bone-grafting procedures), the surgical sites should not be cleaned with routine mechanical plaque control for up to 4 to 6 weeks.
        4. Areas of the dentition not involved by the periodontal surgery may be cleaned with routine self-care techniques.
    - b. **Postsurgical Medications.** Patients should be encouraged to take medications as prescribed.
        1. If systemic antibiotics are prescribed, it is particularly important for the patient to understand that all of the prescribed antibiotic medication should be taken.
        2. Common postsurgical medications include either nonsteroidal anti-inflammatory agents or narcotic pain medications. Usually, these pain medications should only be taken as long as needed.
    - c. **Dietary Changes.** Chewing must be limited to areas not involved in the surgery until healing has progressed to an acceptable level.
        1. Many of these periodontal procedures require that the surgical site be undisturbed for an extended period of time.
        2. Recommendations for a soft or liquid diet for 24 to 48 hours are routine following most periodontal surgical procedures.
        3. Chewing should be limited to the side of the mouth not involved in the surgery, especially during the early phases of healing.
2. **Postsurgical Complications**
    - a. **Facial Swelling.** It is common for the patient to experience slight facial swelling following most types of periodontal surgery. This is an expected post-surgical event that usually peaks 2–3 days after surgery, then naturally subsides.
        1. Swelling can arise from the tissue trauma incurred during the procedure and can even occur during the second and third day following the surgery.
        2. Although this swelling can be disconcerting to the patient, it is usually not a sign that healing is compromised.
        3. Swelling can be minimized by the intermittent use of ice packs for the first 8 to 10 hours following the surgery.
    - b. **Postsurgical Bleeding.** Slight bleeding following periodontal surgery is to be expected.
        1. Patients should be reassured that minor bleeding is not a cause for alarm.
        2. Postsurgical instructions should clearly state that if excessive bleeding occurs, the emergency number (like 911 in the United States and Canada) should be contacted immediately.
    - c. **Smoking.** Surgical patients who have elected to continue smoking should be counseled to suspend their habit during the healing phase.
3. **Organizing Postsurgical Visits.** It is the dentist's responsibility to manage postsurgical problems, such as extreme pain or infection. The dental hygienist, however, can perform much of the routine postsurgical patient management. Following periodontal flap surgery, the patient is most often reappointed in 5 to 7 days for the first postsurgical visit. Postsurgical care for the various types of periodontal surgery varies; however, steps to be followed at a typical postsurgical visit are outlined below.
    - a. **Steps Involved in a Typical Postsurgical Visit**
        1. **Step 1: Patient Interview.** An interview is conducted with the patient to determine what the patient experienced during the days following the surgery. The patient interview should be detailed enough to provide the dental hygienist with an overview of possible problems to investigate and solve at the postsurgical visit. It is also imperative that the dental hygienist alerts the dentist if any unusual conditions are reported by the patient or are observed during the postsurgical visit. The following are some of the items that would normally be included in this interview.

   a. **Analgesics.** Following periodontal surgery, analgesics (pain control medications) are used to control patient discomfort. The patient should be asked about the current level of discomfort and if another prescription is needed.
   b. **Antibiotics.** If antibiotics were needed following a surgical procedure, remind the patient that all of the antibiotic tablets should be taken. It is also important to find out if the patient experienced any unusual reactions to the antibiotic.
   c. **Antimicrobial Mouth Rinse.** An antimicrobial mouth rinse, such as 0.12% chlorhexidine gluconate, may have been prescribed for the patient to use during healing because mechanical plaque control must be restricted at the surgical site following periodontal surgery. Ask about the amount of mouth rinse remaining. During the course of the visit, it may be necessary to provide the patient another prescription for this mouth rinse.
   d. **Swelling.** Following periodontal surgery, it is common for the patient to experience slight facial swelling. Remember that although slight swelling can be disconcerting to the patient, it is a common post-surgical event that subsides 2–3 days after surgery and usually not a sign that healing is compromised. However, if the swelling persists past 72 hours, enlarges rapidly in size, or impedes the airway, the oral health care provider should immediately refer the patient to an emergency physician (also known as an "ER doctor") for prompt management of a potential medical emergency.
   e. **Postsurgical Bleeding.** Inquire about postsurgical bleeding. It is common for patients to experience a little bleeding following periodontal surgery, but heavy bleeding is not routinely observed following periodontal surgery. However, if abnormal bleeding is suspected, the dentist should be alerted prior to planning any additional periodontal surgical intervention.
   f. **Sensitivity to Cold.** Sensitivity to cold following postsurgical root exposure is quite common. Although this is an annoying postsurgical occurrence, the sensitivity normally dissipates within the first few weeks following the surgery if excellent plaque control is maintained.

2. **Step 2: Vital Signs.** The patient's vital signs including blood pressure, pulse, and temperature are assessed. An elevated temperature at the first postsurgical visit can indicate a developing infection.
3. **Step 3: Periodontal Dressing.** Any periodontal dressing placed at the time of surgery is removed so that the surgical site can be examined. The surgical site is rinsed with warm sterile saline, and cotton-tipped applicators are used to remove any debris adherent to the teeth, soft tissues, or sutures. Suture material can become trapped within the periodontal dressing. When removing dressings, it might be necessary to loosen the dressing slightly and cut the suture before completely removing the dressing from the necks of the teeth. **Figure 30-70** shows interrupted interdental sutures ready for removal at the 1-week postsurgical visit.

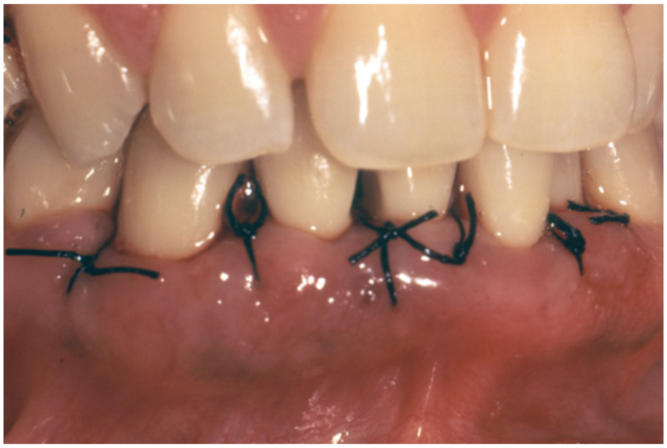

Figure 30-70 Interrupted Interdental Sutures Ready for Removal. At the 1-week postsurgical visit, the periodontal dressing has been removed, the sutures have been cleaned with sterile saline, and the sutures are ready for removal.

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4. **Step 4: Examination of Surgical Site.** Examine the surgical site with care. Tissue swelling or exudate such as pus can indicate a developing infection. Excessive granulation tissue that occasionally forms in the surgical site should be removed with a sharp curette.
5. **Step 5: Suture Removal.** The sutures are cut and removed using sterile scissors and tissue forceps. Remember to pull the suture out of the tissue without drawing the knot through the tissue.
6. **Step 6: Plaque Biofilm Removal.** All plaque biofilm on the teeth in the area of the surgery is removed. It is typical for patients to be unable to perform good plaque control during the days following periodontal surgery, so plaque accumulation is likely. Part of the responsibility of the dental team is to help the patient with plaque control at each postsurgical visit during the critical stages of healing.
7. **Step 7: Replacement of Periodontal Dressing.** If indicated, the periodontal dressing is replaced. For most surgical procedures, the periodontal dressing should be discontinued as soon as the patient can resume some mechanical plaque control. In a few instances, the tissues may not be well adapted to the necks of the teeth, and replacement of the periodontal dressing should be considered to protect the wound for at least another week.
8. **Step 8: Self-Care Instructions.** The patient is instructed in self-care. Mechanical plaque control should be resumed as soon as possible following periodontal surgery, but special instructions may be necessary during the first few weeks following the surgery.
    a. Special self-cleaning aids, such as postsurgical toothbrushes with very delicate and flexible bristles, may be recommended during early stages of healing.
    b. During postsurgical healing, it is frequently necessary to continue monitoring the patient's plaque control as the tissues heal and mature. Gingival margin contours usually are altered to some degree by the surgery, and this may necessitate the introduction of additional self-care aids that were not necessary prior to the surgery. Monitoring and modification of the patient's self-care efforts during the healing phase is one of the most important responsibilities of the dental team and can help ensure success of the surgical procedure.
9. **Step 9. Reappointment.** The patient is reappointed for the second postsurgical visit. This second visit should occur 2 to 3 weeks following the surgery.

b. **Follow-up Visits**
    1. Following the initial postsurgical visit, additional postsurgical visits may be scheduled based on the extent of healing of the surgical wound.
    2. Professional tooth polishing should be performed every 2 weeks until the patient can safely resume routine self-care.
    3. When healing is complete, the patient is always placed on a program of periodontal maintenance. Enrolling the patient in a periodontal maintenance program is critical in preserving and sustaining the long-term benefits of periodontal surgery.
    4. Attachment of the flap back to the alveolar bone is usually complete within 3 weeks following the surgery, and for many surgical procedures it is safe to proceed with restorative care in the surgical site after at least 6 weeks of healing. Note that some periodontal surgical procedures (such as bone replacement graft and periodontal regeneration) will require much longer periods of healing prior to restoration placement.
    5. Remodeling of the soft tissue can continue, however, for up to 6 months. So, the dentist may wait quite a while prior to the placement of definitive restorations in esthetic zones, such as on anterior teeth.

Chapter Summary Statement

Periodontal surgery is a critical element in the care of most patients with moderate to severe periodontitis and the foundation in the care of many patients in need of restorative dental procedures. The periodontal flap is a fundamental part of most periodontal surgical procedures, and a basic understanding of the principles of periodontal flap surgery is important to the dental hygienist. The healing of periodontal surgical wounds is a complex process, and the terminology that has been used to describe the various types of healing that can occur in the periodontium can be confusing. A variety of specific types of periodontal surgery are being used; these techniques include procedures such as flap for access, osseous resective surgery, bone replacement grafting, periodontal regeneration, and periodontal plastic surgery, among others. The dental hygienist should be familiar with the common types of periodontal surgery employed. Current research into enhancing the outcomes of periodontal surgery by using chemical and biologic mediators is ongoing. Postsurgical care following periodontal surgery is vital to maintaining the successful results of surgical outcomes, and dental hygienists play a key role in the management of patients following periodontal surgery.

Section 6

Focus on Patients

CASE 1

You are assigned the task of providing nonsurgical therapy for a periodontitis patient. During routine nonsurgical periodontal therapy, you encounter multiple sites where the probing depths exceed 6 mm. During periodontal instrumentation, you are unable to instrument the root surfaces thoroughly in the areas of the deepest pockets. What should you tell the patient related to this clinical observation?

CASE 2

During nonsurgical periodontal therapy, a patient with periodontitis informs you that the dentist had previously discussed the possibility of periodontal surgery. The patient expresses deep concern and fear over the thought of agreeing to any periodontal surgery. The patient tells you about an aunt who had periodontal surgery many years ago and had many problems following the surgery. How should you proceed?

CASE 3

At the time of the first-week postsurgical visit, you note that a patient who had undergone flap for access surgery has a temperature of 101.5°F (38.6 degrees Celsius) and a pulse rate of 70 beats per minute. Clinical examination of the surgical site reveals that the sutures are in place, but there appears to be a good deal of swelling in one part of flap. How should you proceed?

CASE 4

You are assigned the task of managing the first-week postsurgical visit for a patient who had an apically positioned flap with osseous resective surgery. Following removal of the periodontal dressing and removal of the sutures, you note several areas where the healing is progressing by secondary intention because the flap could not be adapted to the teeth perfectly at the time of surgery. Although healing is progressing satisfactorily, it is apparent that not all of the connective tissue wound around the teeth is completely covered by epithelium yet. How should you proceed?

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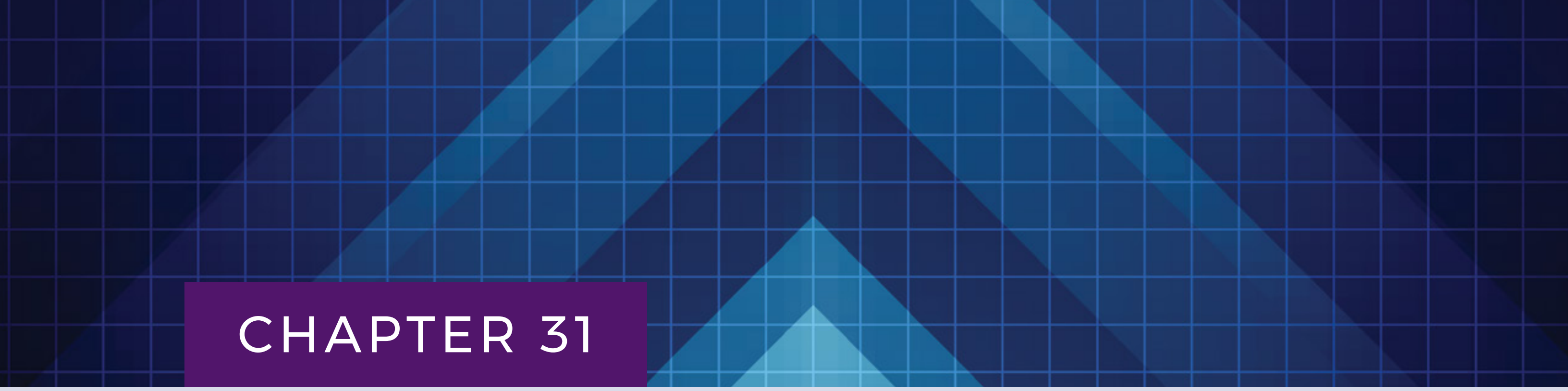

CHAPTER 31

Maintenance for the Periodontal Patient

- Section 1 Introduction to Periodontal Maintenance Therapy
- Section 2 Planning and Implementing Periodontal Maintenance Therapy
- Section 3 Periodontal Disease Recurrence
- Section 4 Patient Compliance with Periodontal Maintenance
- Section 5 Root Caries as a Complication During Maintenance
- Section 6 Focus on Patients
  - Clinical Patient Care

CLINICAL APPLICATION

Given the chronic nature of periodontitis, an appropriate periodontal maintenance program performed at regularly timed intervals is the most integral part of the entire periodontal treatment approach. A periodontal maintenance program is especially important in preventing future progression or recurrence of the disease after the periodontium has been successfully stabilized following active treatment. At each periodontal maintenance, members of the dental team continuously monitor the patient's oral health, diagnose any incipient signs of recurrent disease, and intercept any unstable/deteriorating sites with the appropriate supportive therapy. The success of periodontal maintenance care hinges on two major factors: (1) the patient's willingness to maintain plaque biofilm levels that are consistent with health and stability and to adhere to the recommended periodontal maintenance regimen and (2) the clinician's ability to deliver the appropriate management. As such, the dental hygienist must have a firm understanding of the goals, benefits, and objectives of periodontal maintenance care and the importance of continually reinforcing and motivating the patient to follow the recommended periodontal maintenance regimen at each recall visit. This chapter outlines the fundamental aspects of periodontal maintenance for all members of the dental team to consider during patient care.

LEARNING OBJECTIVES

- List three objectives of periodontal maintenance.
- Describe how periodontal maintenance relates to other phases of periodontal treatment.
- List the typical steps performed during an appointment for periodontal maintenance.
- Define the term *baseline data*.
- Describe guidelines for determining whether the general practice office or the periodontal office should provide periodontal maintenance.
- Describe how to establish an appropriate interval between maintenance appointments.
- Define the term *periodontal disease recurrence*.
- List clinical signs of periodontal disease recurrence.

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- List reasons for periodontal disease recurrence.
- Define the term *compliance*.
- Explain the role compliance plays in maintaining periodontal health and stability.
- List reasons for noncompliance with periodontal maintenance recommendations.
- Explain some strategies that can be used to improve patient compliance.
- Define the term *root caries* and list recommendations for use of fluorides in the prevention of root caries.
- Describe the various types of assessment tools an oral health care practitioner can use to evaluate a patient's risk of developing caries.

KEY TERMS

Periodontal maintenance therapy
Baseline data
Periodontal disease recurrence
Refractory periodontitis
Compliance
Compliant patient
Noncompliant patient
Root caries
Caries Management by Risk Assessment (CAMBRA)

Section 1

Introduction to Periodontal Maintenance Therapy

1. **Description of Periodontal Maintenance Therapy.** Long-term successful management of periodontitis requires involvement of patients in posttreatment appointments at well-timed intervals known as periodontal maintenance therapy. **Periodontal maintenance therapy** is a term that refers to the continued care provided by members of the dental team to help a patient maintain periodontal health following the successful completion of nonsurgical or surgical periodontal therapy.[1–5]
   a. Periodontal maintenance is performed at appropriately timed intervals to assist the patient in maintaining oral health; the precise intervals are selected to meet the needs of the individual patient.
   b. Once initiated, periodontal maintenance is routinely continued throughout the life of the natural dentition (or the life of the dental implant).
   c. Periodontal maintenance can be discontinued temporarily if surgical or nonsurgical therapy must be reinstituted because of the recurrence of periodontitis.
   d. Periodontal maintenance is performed on both natural teeth and dental implants.
   e. Although *periodontal maintenance* is the preferred term, other terms that have been used for periodontal maintenance are *supportive periodontal therapy* (SPT) and *periodontal recall*.
   f. It is important to appreciate that periodontal maintenance is not synonymous with a dental prophylaxis.
2. **Importance of Periodontal Maintenance**
   a. Periodontal maintenance is one of the most important phases of periodontal treatment. A periodontal maintenance program is critical to ensuring that the periodontal tissues are maintained in a state of health, with an acceptable degree of disease stability, patient comfort, and function.
      1. With good periodontal maintenance, most periodontitis patients can retain their teeth or implants in function and comfort throughout their lives.[3,5]
      2. In the absence of periodontal maintenance, patients frequently exhibit a recurrence of periodontitis.[6–10]
      3. Periodontal maintenance can be successful regardless of the specific type of periodontal treatment (surgical or nonsurgical) needed by the patient.
      4. The success of periodontal maintenance in reducing tooth loss is well documented in the literature.[9,11–13] **Figure 31-1** illustrates the importance of enrolling a patient on a well laid-out periodontal maintenance program as a function of attachment gain vs attachment loss.

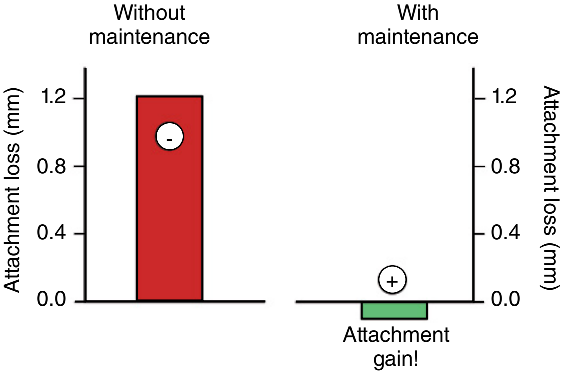

Figure 31-1 Attachment Loss with Maintenance Versus Attachment Loss without Maintenance. A classic study assessed the efficacy of a periodontal maintenance program to prevent the recurrence of disease in patients treated for advanced periodontitis. Patients who were not in a periodontal maintenance program had progressive attachment loss over a 6-year period. In contrast, patients who were placed on maintenance every 2 to 3 months over the 6-year period exhibited attachment gain.

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3. **Goals of Periodontal Maintenance Therapy.** Periodontal maintenance has several overlapping goals (**Box 31-1**).

Box 31-1 Goals of Periodontal Maintenance

- Minimize the recurrence and progression of periodontitis.
- Reduce the incidence of tooth loss.
- Increase the likelihood of detecting and treating other oral conditions.

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a. **To Minimize the Recurrence and Progression of Periodontitis**
   1. The primary risk factor for inflammatory periodontal diseases is bacterial plaque biofilm.
      a. Despite a patient's best self-care efforts, it is common for plaque biofilm and/or calculus to reform at some sites. Periodic professional biofilm and calculus removal is an important step in periodontal maintenance therapy.
      b. It is common for a patient's self-care efforts to become less effective over time. Hence, reinforcement and review of self-care techniques at each periodontal visit is critical in renewing the patient's motivation to adhering to good self-care habits.
   2. Secondary risk factors for inflammatory periodontal diseases include plaque-retentive areas (such as restorations with overhangs), smoking, and certain systemic factors.
      a. Whereas these secondary risk factors should always be addressed as part of nonsurgical periodontal therapy, the condition of most patients changes over time.
      b. The dental practitioner should be vigilant for any newly emerging secondary risk factors (e.g., recently placed plaque-retentive restorations or changes in medical history) that might put the patient at a higher risk of disease recurrence.[11,14–16]

b. **To Reduce the Incidence of Tooth Loss.** One of the primary overall goals of all phases of periodontal therapy (including periodontal maintenance) is to reduce the incidence of tooth loss (or reduce the incidence of implant loss).
   1. In a study by Wilson et al. evaluating tooth loss in maintenance patients in a private periodontal practice, tooth loss was shown to be inversely proportional to the frequency of periodontal maintenance.[17]
   2. Other studies have also shown that patients who are compliant with a regular periodontal maintenance regimen lose fewer teeth than patients who are not as compliant to undergoing frequent periodontal maintenance.[9,12,18]
   3. Even a patient who has lost teeth during the active stage of treatment can prevent additional tooth loss if he or she adheres to the recommended periodontal maintenance protocol.[1,2,11,18]

c. **To Increase the Likelihood of Detecting and Treating Other Oral Conditions**
   1. As members of the health care community, the dental team must be vigilant for the development of any oral condition that can affect patient welfare. For example, at each periodontal maintenance visit, an oral cancer screening is performed. If oral cancer is identified at its early stage, then interceptive treatment of the pathologic lesion can improve the long-term prognosis of the patient and treatment outcomes. A thorough oral cancer screening examination is an important step that should be undertaken by the oral health care provider at each recall visit, irrespective of risk factors (like smoking, alcohol consumption, and prior cancer history) present. (**Figure 31-2**)
   2. Periodontal maintenance allows for ongoing monitoring of the overall oral health of a patient.

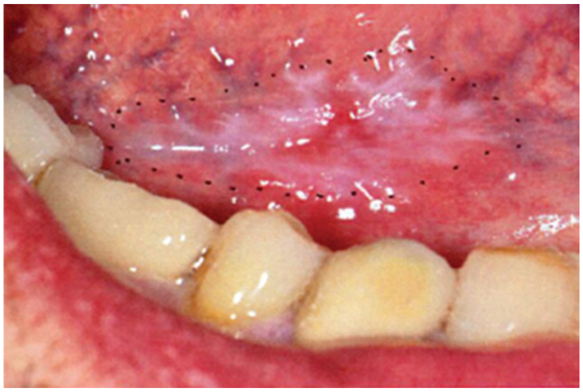

Figure 31-2 Oral Cancer Screening Examination as a Critical Component of Care Performed at Every Recall Visit. An ASA I 40-year-old patient with no known risk factors for cancer (e.g., negative smoking habit, negative alcohol consumption, and negative history of cancer) presented for a dental check-up and periodontal maintenance. Upon routine oral cancer screening evaluation, the dental hygienist discovered a suspicious asymptomatic leukoplakic, ulcerated patch on the right ventrolateral surface of the tongue (outlined by black dashes). Due to the suspicious nature and the location of the lesion, the dental hygienist immediately referred the patient to the periodontist who performed a biopsy of the suspicious tongue lesion. Subsequent pathology report later confirmed a diagnosis of oral squamous cell carcinoma.

4. **Patient and Clinician Roles in Periodontal Maintenance**
   a. Periodontal maintenance is a team effort that requires commitment from everyone involved. The periodontal maintenance team includes the members of the dental team plus the patient and occasionally other health care providers, such as the patient's physician or nurse practitioner.
   b. Periodontal maintenance requires considerable effort from the patient in sustaining meticulous self-care and cooperating with regular ongoing professional periodontal maintenance care. Patients must be made aware of the need for this ongoing effort even prior to receiving nonsurgical periodontal therapy.
   c. In addition, periodontal maintenance requires considerable effort on the part of the dental health team for professional care at regular intervals, renewal of patient motivation, instruction in self-care techniques, and elimination or reduction of primary and secondary risk factors.

5. **Relationship of Periodontal Maintenance to Other Phases of Therapy.** It is important for a clinician to understand how periodontal maintenance relates to other phases of comprehensive periodontal therapy; this relationship is outlined in **Figure 31-3**.

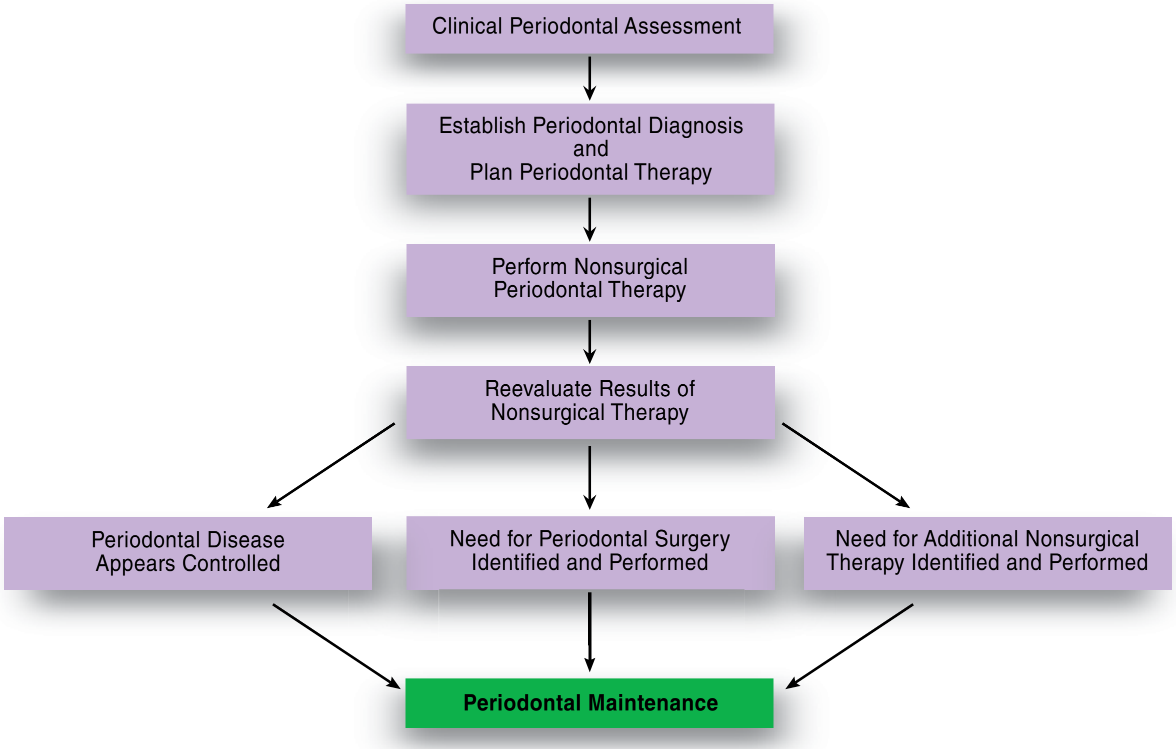
Clinical Periodontal Assessment
Establish Periodontal Diagnosis and Plan Periodontal Therapy
Perform Nonsurgical Periodontal Therapy
Reevaluate Results of Nonsurgical Therapy
Periodontal Disease Appears Controlled
Need for Periodontal Surgery Identified and Performed
Need for Additional Nonsurgical Therapy Identified and Performed
Periodontal Maintenance

Figure 31-3 Phases of Periodontal Therapy. This flowchart illustrates how periodontal maintenance relates to other phases of periodontal therapy that are frequently provided for patients with periodontal disease.

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Section 2

Planning and Implementing Periodontal Maintenance Therapy

1. **When to Implement Periodontal Maintenance.** Following nonsurgical periodontal therapy, a reevaluation of the patient's periodontal status is performed with a particular emphasis on the results obtained from nonsurgical therapy.
    a. Based upon the findings of this reevaluation, additional therapy such as periodontal surgery might be recommended.
    b. At the reevaluation, the dental team also must determine the most appropriate time interval for future periodontal maintenance recall visits.
    c. For most patients, periodontal maintenance will begin following this reevaluation because for many patients no further active therapy is needed, at least in selected sites in the dentition that exhibit periodontal stability.
    d. Even though periodontal surgery may be recommended in some sites, the maintenance phase begins following nonsurgical therapy at least for those sites where no further active periodontal therapy is indicated.
2. **Procedures Performed During Periodontal Maintenance.** Successful periodontal maintenance requires the active participation of the patient as well as all members of the dental team. In most dental offices, the dental hygienist plays a major role in each periodontal maintenance visit. A typical patient office visit for periodontal maintenance includes the steps discussed below and outlined in **Box 31-2**.[19–21]

Box 31-2 Overview of the Steps Performed During a Periodontal Maintenance Appointment

- Update medical status
- Patient interview
- Clinical assessment
- Evaluation of effectiveness of patient self-care
- Identification of treatment needs
- Periodontal instrumentation
- Patient counseling
- Application of fluorides

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a. **Update of Medical Status.** An update of a patient's medical status is always the first step in any clinical appointment.

b. **Patient Interview**
   1. A patient interview is part of a periodontal maintenance appointment; during the interview, changes in the medical, dental, and social status of the patient should be explored and documented.
   2. Examples of social status issues that should be explored would be changes in lifestyle, bereavement, and work status.
   3. The patient interview should also include a review of dental care provided by other clinicians since the previous maintenance visit.
   4. In addition, the patient interview should clarify the patient's perception of his or her oral status, including the patient's thoughts about problems encountered during self-care efforts.

c. **Clinical Assessment**
   1. Following the patient interview, a thorough clinical assessment should be performed. Results of the clinical assessment should then be compared with previous baseline data. The term **baseline data** refers to clinical data gathered at the initial visit that is subsequently used for comparison at future maintenance visits.
   2. The actual clinical assessment usually includes steps such as those listed below:
      a. Extraoral and intraoral examination
      b. Dental examination
      c. Radiographic examination (if indicated)
      d. Periodontal examination, including the following features:
         1. **Probing Depths**
            a. Probing depths should be recorded with the same attention to detail that was employed during the initial examination.
            b. Disease progression (continuing attachment loss) should be suspected when a 2-mm increase in probing depth is noted at a site.
         2. **Bleeding on Probing**
            a. When present, bleeding on probing is generally visible within a few seconds after gentle periodontal probing.
            b. Research suggests that after a few years of maintenance, a high frequency of bleeding on probing is a predictor of increased risk for progressive attachment loss.[22]
            c. Bleeding sites should be charted because these sites may need more attention during periodontal instrumentation.
         3. **Attachment Level**
            a. Attachment levels should be recorded at all sites in the dentition.
            b. The most reliable way to evaluate periodontal disease control is by sequential comparison of clinical attachment level measurements.
            c. Disease progression is thought to be indicated by a 2-mm increase in clinical attachment loss at a specific site as measured with a manual periodontal probe.

        4. **Tooth Mobility**
            a. Tooth mobility can be stable or increasing. Increasing mobility over time is one of the important clinical features to note.
            b. The more severe the mobility measured in a tooth, the greater the risk of eventual tooth loss.
        5. **Furcation Involvement**
            a. Periodontal disease control is more difficult in areas of furcation involvement.
            b. The more advanced the furcation involvement, the greater the risk of tooth loss over time.
        6. **Mucogingival Involvement**
        7. **Levels of Plaque Biofilm and Calculus**

d. **Evaluation of Effectiveness of Self-Care**
    1. Patients often spend considerable time on self-care and justifiably expect to be informed about the effectiveness of their efforts.
        a. Plaque scores recorded after using a disclosing solution are good indications of the patient's level of self-care compliance and disclosing solution can be used to allow patients to view the biofilm accumulation.
        b. Plaque scores may reveal that the patient is in need of renewed instruction in self-care methods.
    2. Biofilm accumulation can be related to many factors; examples of these factors are listed below.
        a. Patients may lack the manual dexterity needed to carry out the self-care regimen that was recommended previously. When manual dexterity is a problem, alternative self-care techniques should be considered. Older patients can lose skills that they were perfectly capable of performing at a previous maintenance visit.
        b. Unfortunately, it is common for patients to discontinue the use of one or more of the self-care methods recommended previously.
        c. Gingival recession or shrinkage may have occurred following periodontal surgery; introduction of new interdental aids may be indicated for plaque biofilm removal on proximal root surfaces (refer to Chapter 26 to review the many types of interdental aids available to help remove plaque biofilm from interproximal areas).

e. **Identification of Treatment Needs**
    1. It is a routine part of periodontal maintenance to perform thorough periodontal instrumentation to remove biofilm and calculus, but other treatment needs can also be identified.
    2. Examples of other treatment needs can include local delivery of antimicrobials, restorative therapy, and reinstitution of active periodontal therapy.
    3. Selective tooth polishing may be indicated to remove stains that are visible when the patient smiles.

f. **Biofilm Removal**
    1. Biofilms are resistant to topical chemical control; therefore, frequent professional removal of plaque biofilm is an essential component of successful nonsurgical periodontal therapy.
    2. Professional biofilm control may be accomplished by hand and/or ultrasonic instrumentation or subgingival glycine powder air polishing.

g. **Periodontal Instrumentation**
    1. The goal of periodontal instrumentation is to create an environment that is biologically acceptable to the tissues of the periodontium.
    2. Thorough removal of biofilm and calculus deposits should be accomplished using hand and/or ultrasonic instrumentation.
    3. The main adverse effect of frequent mechanical instrumentation of the root surface is disturbance of the epithelial attachment and cumulative, irreversible root substance removal,[23–28] and gingival recession.[29,30]
        a. Hard tissue loss is one of the major causes of dentin sensitivity to hot and cold stimuli, as well as sensitivity to toothbrushing.[31–34]
        b. Because periodontal instrumentation is a routine part of nonsurgical periodontal therapy, it is important that removal is accomplished in an efficient manner with minimal hard tissue damage.[35]
            1. Following periodontal therapy, some patients will present for periodontal maintenance with little to no subgingival calculus deposits. In these patients, firm stroke pressure with the instrument against the tooth is not necessary and should be avoided.

2. Ultrasonic instrumentation with a precision thin tip has been shown to remove less root substance than hand instrumentation and offers the added benefit of the antimicrobial effect created by the vibrating ultrasonic tip.[36,37]
3. Plastic curettes (such as those used to debride dental implants) can be effective for deplaquing root surfaces and minimizing trauma to the root during maintenance care when no calculus is present or when implants are involved.

h. **Patient Counseling**
   1. As already discussed, maintenance patients should always be counseled of the importance of self-care efforts since biofilm control by the patient is a critical element in preventing recurrence of periodontitis.
      a. Failure to provide this information may give a patient the impression that the dental team is not truly interested in the patient's dental health status.
      b. Most patients will need some reinforcement of motivation for biofilm control in addition to retraining in the complex skills involved. Vatne et al. found that providing the patient with customized information on the pathogenesis, prevention, and treatment as well as maintenance of periodontal diseases resulted in a high degree of patient compliance with maintenance therapy.[38]
   2. It is also prudent to include counseling that explains the need for compliance with the periodontal maintenance regimen, because compliance with the periodontal maintenance regimen will always remain a challenge for some patients.
   3. Other counseling may be indicated for specific patients. Examples of counseling that may be needed are caries prevention counseling, smoking cessation, or dietary changes.

i. **Application of Fluorides**
   1. Professional application of fluoride treatments during periodontal maintenance care is normally indicated to promote remineralization of tooth surfaces, minimize the risk of root caries on exposed root surfaces, and manage dentinal hypersensitivity. Additionally, patients who are deemed to be highly susceptible to caries (i.e., due to medication-induced hyposalivation) may benefit by routine application of fluoride at each periodontal maintenance visit.
   2. Research studies suggest that high concentrations of topical fluorides may also have some antimicrobial properties and may be of some benefit in decreasing plaque biofilm accumulation.
   3. The use of fluorides is discussed in detail in Section 5 of this chapter.

3. **Decisions Related to Periodontal Maintenance Therapy**
   a. **Office Guidelines for Provision of Periodontal Maintenance.** For patients treated in a periodontal office, members of the dental team should discuss guidelines for how periodontal maintenance should be provided (i.e., either in the general dental practice office or in the periodontal practice). This decision is based on the combined clinical judgments of the general practice dental team and the periodontal practice team in determining how to best maintain the health of the patient. Some general guidelines are outlined below:
      1. Patients who have been treated previously for Stage I periodontitis and currently display a stable periodontium can usually receive periodontal maintenance in a general dental practice.
      2. Patients who have been treated previously for Stage II periodontitis and currently display a stable periodontium can usually be managed by alternating periodontal maintenance visits between the general dental practice and the periodontal practice.
      3. Patients with Stage III (severe periodontitis with potential for additional tooth loss) and Stage IV (advanced periodontitis with extensive previous tooth loss and potential for loss of remaining dentition) periodontitis should be monitored and should receive periodontal maintenance in a periodontal practice because of the high risk of tooth loss. In addition, annual or semiannual visits should be scheduled with a general dentist who will provide restorative and other general dental care.
      4. Patients who have been treated for Stage IV, Grade C periodontitis should receive all phases of periodontal therapy, including periodontal maintenance in a periodontal practice. In addition, annual or semiannual visits should be scheduled with a general dentist who will provide restorative and other general dental care.
   b. **Establishing Appropriate Periodontal Maintenance Intervals**
      1. Establishing a periodontal maintenance interval that is appropriate for the patient can be challenging. The frequency of periodontal maintenance visits must be determined on an individual basis.[39,40] A single recall interval (e.g., 6 months) is not suitable for all patients. Some factors to consider in determining the interval between maintenance visits include the following:

   a. **Severity of Periodontitis.** In general, the more severe the periodontitis, the shorter the intervals should be between periodontal maintenance visits.
   b. **Adequacy of Patient Self-Care.** In general, the more effective the patient's self-care, the less frequently the patient needs to be seen. For patients with less-than-optimal self-care, the intervals between maintenance visits should be shorter.
   c. **Host Response.** Systemic or genetic factors may negatively affect the host response. For example, a patient who continues to smoke or one with poorly controlled diabetes should be seen at shorter intervals.
2. An important guide for determining the frequency of maintenance care is based on the time interval for the repopulation of periodontal pathogens following thorough periodontal instrumentation.
   a. Studies indicate that following periodontal instrumentation, the subgingival pathogens return to pre-instrumentation levels in approximately 9 to 11 weeks in most patients, though times can vary.[41]
   b. Research shows that periodontal maintenance should be performed at least every 3 months or less for the removal and disruption of subgingival periodontal pathogens. This 3-month interval is the one most frequently recommended, though this interval may need to be adjusted based on sound clinical judgment.[40]
   c. Patients who comply with frequent periodontal maintenance experience less attachment loss and tooth loss than patients who have less frequent maintenance care.

Section 3

Periodontal Disease Recurrence

1. **Understanding Periodontal Disease Recurrence**
   a. **Periodontal Disease Recurrence Defined**
      1. The term **periodontal disease recurrence** refers to the return of the disease in a patient that has been previously, successfully treated for periodontitis.
      2. The term *disease recurrence* implies that the periodontitis was indeed brought under control following active periodontal therapy (nonsurgical periodontal therapy alone or nonsurgical periodontal therapy plus periodontal surgery), but that at some later time the periodontitis once again results in progressive attachment loss.
      3. Note that in spite of having received excellent treatment, patients who have been treated for periodontitis are at risk for future recurrence of periodontitis for as long as teeth (or implants) are present.
      4. Periodontal disease recurrence can occur at specific sites only (not necessarily throughout the dentition). For example, it would be possible for a patient previously treated for periodontitis to exhibit disease recurrence on the mesial surface of a maxillary first premolar tooth (due to the plaque-retentive mesial root concavity) while all other sites on the same aforementioned tooth or all the teeth in the dentition exhibit periodontal stability. It is also possible for disease recurrence to be more generalized and affect the entire dentition.
   b. **Clinical Recognition of Periodontal Disease Recurrence.** To date, the most effective way to identify sites of recurrence of periodontitis (i.e., sites of progressive attachment loss) is through thorough periodic clinical assessments. The usual clinical signs of periodontal disease recurrence are listed in **Box 31-3**.

Box 31-3 Clinical Signs of Periodontal Disease Recurrence

- Progressive clinical attachment loss
- Pockets that get deeper over time
- Pockets that bleed upon probing
- Pockets that exhibit exudate
- Radiographic evidence of progressing bone loss
- Increasing tooth mobility

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c. **Reasons for Periodontal Disease Recurrence.** Periodontitis recurs in patients for a variety of reasons, and the members of the dental team should be aware that it is not always possible to determine a specific reason for disease recurrence. However, the most common reasons for recurrence of periodontitis are:
    1. Inadequate self-care by the patient and/or poor patient adherence to the recommended periodontal maintenance regimen.
    2. Incomplete professional treatment.
        a. Incomplete periodontal instrumentation.
        b. Failure to control all local risk factors.
    3. Failure to control systemic factors.
    4. Inadequate control of occlusal contributing factors.
    5. Improper periodontal surgical technique.
    6. Attempting to treat teeth with a poor prognosis.

d. **Refractory Form of Periodontal Disease Versus Recurrent Periodontal Disease.** Refractory periodontal disease should be differentiated from recurrent periodontal disease. Unfortunately, periodontitis in certain patients is difficult or impossible to control even with all the modern therapies currently available and in spite of the best efforts of the most skilled clinicians.
    1. As already discussed, the term *periodontal disease recurrence* refers to the return of the disease in a patient who has been previously successfully treated for periodontitis.
    2. The term **refractory periodontitis**, however, refers to periodontitis that is resistant to treatment from the outset of therapy even with what appears to be appropriate periodontal therapy.
    3. Referral to a periodontal practice is usually indicated when refractory periodontitis is suspected in a patient.

2. **Options for Management of Patients with Periodontal Disease Recurrence.** The members of the dental team should be alert for the need for retreatment that may be identified during any periodontal maintenance visit. When periodontal disease recurrence is identified, several factors should be considered when planning the periodontal retreatment.
    a. If inadequate patient self-care appears to be the fundamental cause of the disease recurrence, then nonsurgical therapy should be reinstituted followed by a reevaluation of the patient's periodontal status after an appropriate healing time.
    b. If failure to comply with the schedule of periodontal maintenance appears to be the fundamental cause of the disease recurrence, then nonsurgical therapy should be reinstituted along with further patient education about the need for maintenance followed by a reevaluation of the patient's periodontal status after an appropriate healing time.
    c. If there appears to be disease recurrence in limited individual sites in the presence of adequate patient self-care, treatment options can include localized periodontal instrumentation, local delivery of antimicrobial agents, or localized surgical therapy.
    d. If there appears to be disease recurrence in multiple sites in the presence of adequate patient self-care, periodontal surgical therapy is frequently indicated.
    e. If generalized attachment loss has recurred, the systemic condition of the patient should be reassessed with emphasis on the possible need for periodontal surgical intervention, for possible microbial analysis, or for possible local or systemic antimicrobial therapy. Patients of this type should be managed by a periodontist.

Section 4

Patient Compliance with Periodontal Maintenance

1. **Overview of Patient Compliance.** The term **compliance** is defined as the extent to which a person's behavior coincides with medical or health advice. Compliance also is called adherence or therapeutic alliance, but *compliance* is the most common term used in the literature.
    a. A patient is described as a **compliant patient** if he or she adheres to the recommendations set forth by the health care provider. Examples of "this" would be a patient who faithfully takes antihypertensive medications as prescribed by a physician or a patient who cooperates in meeting regularly scheduled periodontal maintenance appointments as recommended by the dental team.

   b. A patient is described as a **noncompliant patient** if he or she does not follow recommendations set forth by the health care provider. Examples of this would be a patient who does not take prescribed medications daily to control diabetes or a patient who does not perform adequate daily self-care that has been recommended as part of the maintenance regimen.

2. **Patient Compliance During Periodontal Maintenance.** Patient compliance with a program of periodontal maintenance is not easy to achieve. It requires that a patient faithfully adhere to a strict program of recall appointments several times each year and follow very specific recommendations for meticulous daily self-care.
   a. Overall, patient compliance with most medical advice is poor, and it should not be surprising that patient compliance with periodontal maintenance is also poor.
   b. Studies of compliance exhibited by periodontal maintenance patients indicate that only 16% to 30% of the patients are fully compliant with periodontal maintenance.[42–44]
   c. Reasons for noncompliance with periodontal maintenance are complex, and those reasons can be different for each patient and even for the same patient at different times.[18,45–49]
   d. Examples of some reasons that have been suggested for noncompliance with recommended programs of periodontal maintenance include the following:
      1. Patient fear of receiving dental treatment.
      2. The expense of the dental treatment involved.
      3. The low priority for dental care for some patients in the face of competing demands for time.
      4. Denial on the part of some patients related to the periodontal challenges they face.
      5. Failure for some patients to understand the implications of noncompliance.
      6. Perceived indifference on the part of the dental health care providers.
3. **Strategies of Improving Compliance.** The thoughtful dental team will investigate and adopt strategies for improving patient compliance with periodontal maintenance.[45] Some strategies for improving compliance are discussed below and outlined in **Figure 31-4**.

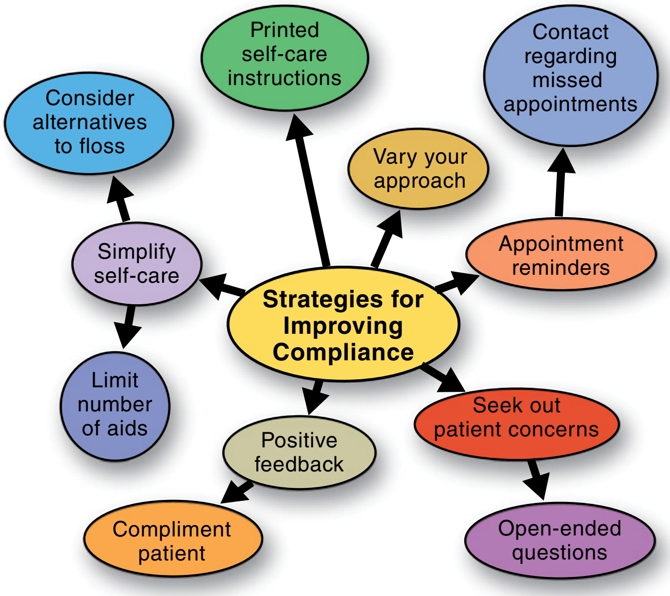
Strategies for Improving Compliance
Printed self-care instructions
Contact regarding missed appointments
Consider alternatives to floss
Vary your approach
Simplify self-care
Appointment reminders
Limit number of aids
Positive feedback
Seek out patient concerns
Compliment patient
Open-ended questions

Figure 31-4 Suggestions for Improving Patient Compliance. An idea map of various strategies for improving patient compliance with recommendations for periodontal maintenance.

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   a. Give a patient printed self-care instructions, and make sure to supply the instructions written in the patient's native language.
   b. Simplify self-care recommendations as much as possible for each patient.
      1. Patients often perceive self-care instructions as being difficult to follow and as too time-consuming in their busy lives.

    2. Self-care instructions should be as clear and as simple as possible while addressing the specific needs of the patient.
    3. Caution should be exercised when recommending multiple types of self-care aids. Patients are less likely to comply with self-care when they are instructed to use multiple aids daily.
    4. When possible, alternatives to traditional dental floss should be considered since compliance with flossing is generally poor.
  - c. Vary the office approach to patient education and self-care instructions from appointment to appointment. Patients often complain about having to listen to the "same old lecture" from the dental hygienist at each periodontal maintenance appointment.
  - d. Seek out patient concerns and provide opportunities for communication by asking patients open-ended questions. Examples of open-ended questions appear below.
    1. "What are your concerns about this suggestion or treatment?"
    2. "How do you think you will fit this self-care recommendation into your daily schedule?"
    3. "How would you compare using this powered flossing device to using traditional dental floss?"
  - e. Accommodate the individual patient's needs whenever possible. A satisfied patient is more likely to comply with self-care and maintenance appointments.
  - f. Keep patients fully informed about their periodontal condition.
    1. At each visit, counsel the patients about their periodontal health status.
    2. Explain the benefits of having regularly scheduled periodontal maintenance visits and the risks of infrequent professional care.
  - g. Monitor compliance with the maintenance appointments and contact patients promptly when compliance seems to become a problem.
  - h. Provide positive feedback to patients as frequently as possible. Positive reinforcement can help improve compliance.
    1. Areas of improvement should be pointed out to the patient (e.g., such as less biofilm accumulation, fewer bleeding sites, or less inflamed tissue).
    2. Positive reinforcement should be used to convey a motivational message rather than criticism.

Section 5

Root Caries as a Complication During Maintenance

1. **Introduction to Root Caries**
  - a. **Occurrence of Root Caries in Patients with Periodontitis**
    1. Whereas coronal caries occurs on enamel, the term **root caries** refers to tooth decay that occurs on the root surfaces of the teeth.
    2. According to data extrapolated from the 2017–2020 National Health and Nutrition Examination Survey, root caries is a substantial public health concern affecting 11.5% of adults aged 50 to 59, 12.5% of adults between 60 to 69 years, and 12.5% of adults >70 years of age exhibiting unrestored root caries.[50]
    3. In 2013, the NIDCR-funded research network known as the Northwest Practice-based REsearch Collaborative in Evidence-based DENTistry (PRECEDENT) reported:
      - a. A total of 19.6% of adults had root caries.
      - b. Risk factors associated with elevated risk of root caries in middle-aged adults are being of the male sex, dry mouth, root surfaces exposed to the oral environment, and increased frequency of eating or drinking between meals.[51]
    4. A 2004 systematic review on root caries incidence found that 23.7% of older adults develop at least one new lesion annually.[52]
    5. Root caries occurs only if the root surface is exposed to the oral environment due to loss of attachment.[53]
      - a. In health, the root surface is protected by the periodontal attachment apparatus and is not exposed to the oral environment.
      - b. However, in disease or in cases of gingival recession due to non-periodontitis reasons, the root may be exposed to the contaminants (specifically, plaque biofilm) in the oral environment.

b. **Clinical Appearance of Root Caries**
   1. Active root caries lesions usually appear yellowish to light brown and may be covered with biofilm. Inactive lesions appear dark brown or black.[53,54] **Figure 31-5** shows a typical clinical appearance of root caries.
   2. Root caries usually begins at or slightly coronal to the free gingival margin. The carious lesions can spread laterally and can even extend circumferentially around the root surface.[55]

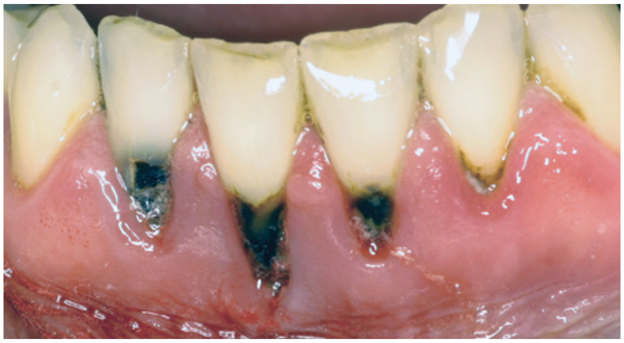


Figure 31-5 **Root Caries.** Root caries on the mandibular incisors of an individual with periodontitis.

Courtesy of Dr. Richard J. Foster, Educational Solutions and Design, Greensboro, NC.

c. **Etiology of Root Caries**
   1. A 2020 systematic review reported that higher counts of *Streptococcus mutans* or *S. lactobacilli* were associated with higher prevalence and extent of root caries; no correlation was found between the presence of *Actinomyces* spp. in plaque and root caries. In addition, a positive correlation was reported between counts of yeast and root caries, but no correlations were found between counts of *S. sobrinus, S. sanguis*, and *S. salivarius*.[56]
   2. Like coronal caries, root caries requires a susceptible tooth surface, plaque biofilm, and time to initiate and progress. However, root caries differs from coronal caries in some ways.
      a. Root surfaces are more vulnerable to demineralization than enamel surfaces. Root surfaces demineralize at a pH of 6.2 to 6.7.[57]
      b. Mineral loss for the root surface during the process of demineralization is up to 2.5 times greater than enamel.[58]
   3. Risk factors for the development of root caries include attachment loss, inadequate patient self-care, a cariogenic diet, infrequent dental visits, past caries experience, inadequate salivary flow, lack of fluoride exposure, and removable partial dentures. A recent review found past root caries, number of surfaces at risk, poor oral hygiene, gender, age, and periodontal disease as the factors most associated with root caries incidence.[59,60]
   4. In addition, individuals who have coronal/enamel caries are 2 to 3.5 times more likely to develop root caries.[61]

2. **General Recommendations for the Prevention of Root Caries.** Root caries is a common problem in patients with periodontitis due to the clinical attachment loss associated with the disease. Managing root caries from a restorative standpoint can be quite difficult. Thus, the best strategy for managing root caries is to prevent the onset of root caries.
   a. **Prevention of Periodontitis.** The prevention of periodontitis and its associated attachment loss is the most effective way to prevent root caries. In patients with existing periodontal disease, prevention of further attachment loss will reduce the surface area susceptible to decay.
   b. **Fluoride for the Prevention of Root Caries**
      1. Root lesions can be arrested by remineralization. A 2007 systematic review of fluoride interventions for root caries concluded that fluoride appears to be a preventive and therapeutic treatment for root caries.[62,63]
      2. A variety of fluoride products can be helpful in preventing root caries. Depicts some of these products. A 2020 systematic review recommended 1.1% to 1.5% fluoride toothpastes/gels and fluoride varnishes as the most effective means to stimulating remineralization of root caries.[64]
         a. **Fluoridated Drinking Water.** Several studies have demonstrated that the presence of fluoridated drinking water throughout the lifetime of an individual reduces the development of root surface caries.[65]

b. **Fluoride Toothpaste**
   1. The use of an 1100 ppm sodium fluoride (NaF) dentifrice results in a significant decrease in root surface caries by 67%.[66]
   2. A randomized clinical trial concludes that a prescription-strength fluoride toothpaste containing 5000 ppm NaF is significantly more effective in controlling root caries lesion progression and promoting remineralization compared to lower-prescription strength fluoride toothpaste.[67]
   3. Patients using fluoride toothpastes should avoid rinsing their mouth with water soon after using fluoride toothpaste.[68]

c. **Fluoride Mouth Rinses.** Fluoride mouth rinses containing 0.05% NaF have been shown to significantly reduce the incidence of root caries.[69]

d. **Professional Application of Fluoride**
   1. A large long-term clinical study showed that semiannual applications of 1.23% APF gel significantly reduced the formation of new root caries. The number of remineralized lesions was significantly increased by daily rinsing with a 0.05% NaF rinse.[70]
   2. Fluoride varnish applied every 3 months has been shown to reduce new root caries formation by over 50%.[64,71]

e. **Silver Diamine Fluoride**
   1. Silver diamine fluoride (SDF) has been widely used outside the United States since 1970. Initially, the U.S. Food and Drug Administration (FDA) approved SDF as a desensitizing agent. However, in 2016, SDF was granted “breakthrough therapy status” by the FDA as a noninvasive, easy-to-apply, inexpensive, and safe caries arresting agent. (The designation “breakthrough therapy status” means that the drug in question will undergo expedited review by the FDA because preliminary clinical evidence shows that it has a reasonable chance of providing more effective treatment than the standard of care. However, this does not mean that SDF is specifically FDA-labeled for use for caries control and management. Instead, as of the publication of this textbook, SDF as an anticaries agent is an off-label use.)
   2. The downsides to SDF are that it leaves behind a dark stain as it arrests the caries lesion, it has the potential to irritate the gingival tissue, and it reportedly leaves behind an unpleasant metallic taste. Nevertheless, aside from these limitations, SDF may be an effective tool in arresting caries progression in children and adults without having to drill into the tooth.
   3. SDF is a colorless liquid composed of 24.4%-28.8% (weight/volume) silver and 5.0-5.9% fluoride.
   4. When topically applied over a carious lesion, the silver has antimicrobial activity which potentially neutralizes the cariogenic bacteria, thereby halting the progression of the caries lesions without removal of sound tooth tissue. The fluoride promotes remineralization of decayed dental tissue.
   5. In summary, the combined effect of the silver and fluoride in SDF a) promotes remineralization, b) greatly reduces caries progression in enamel, dentin, and cementum, and c) reduces the risk of caries on adjacent tooth surfaces.
   6. Indications for SDF include individuals who are at high caries risk, individuals who are unable to access dental treatment or tolerate conventional dental care, such as individuals with physical and/or cognitive disabilities, individuals with limited manual dexterity, or young children who may not tolerate conventional surgical removal of caries.[72,73]

c. **Antimicrobial and Supplemental Remineralization Therapies**
   1. A recent systematic review found no benefit from the use of chlorhexidine varnish in reducing root caries.[74] There is limited evidence of benefit from the use of a chlorhexidine mouthrinse.[75] High-caries-risk adults should rinse with 10 mL of 0.12% chlorhexidine once daily for 1 week per month.
   2. Xylitol-containing gums and mints are recommended for high-caries-risk patients.
      a. The therapeutic dose of xylitol is 6 to 10 g spread throughout the day.
      b. A recent randomized clinical trial found 40% fewer root caries lesions in the xylitol group compared to placebo. The xylitol group received five lozenges containing 1 g of xylitol. The lozenges were consumed across the day.[76]
   3. Casein phosphopeptide (CPP)-amorphous calcium phosphate (ACP) pastes are recommended for high-caries-risk patients.
      a. The paste can be applied with a fingertip daily.

  - b. Most research on CPP-ACP is laboratory based rather than in vivo. However, CPP-ACP may promote remineralization in patients with low salivary flow.
  - c. Several systematic reviews conclude that there is insufficient evidence to make conclusions regarding the effectiveness of CPP-ACP in preventing caries.[77,78]
- 4. In a clinical practice guideline from the American Dental Association, application of a 1:1 mixture of chlorhexidine and thymol varnish (Cervitec Gel) every 3 months was recommended to reduce the incidence of root caries.[79]

d. **Summary and Best Practices**

1. Improved oral hygiene, good dietary habits, regular periodontal maintenance visits, and locally applied preventive agents are important to prevent root caries and periodontal disease progression.[80]
2. Research supports the use of fluoridated water, fluoride toothpastes, fluoride mouth rinses, xylitol, and professional topical fluorides for the prevention of root caries.[81]

3. **Caries Risk Assessment and Management**

a. **Caries Risk Assessment**

1. Research clearly demonstrates that a caries risk assessment is an easy, proactive preventative chairside tool that can identify at-risk individuals and uncover potential risks factors which may predispose that individual to developing dental caries.[82]
2. Subsequent to this research, protocols for clinical management of caries risk were developed and employed at a number of dental schools.[83]Although complete consensus on these protocols continues to develop, there is strong agreement that assessing each patient for dental caries based on individual risk profile(s) is an important component of preventive dental care.[83]
3. Several methods for the assessment of caries risk are available. These include **Caries Management by Risk Assessment (CAMBRA)**, the ADA Caries Risk Assessment Form, and Cariogram.[82,84] CAMBRA and the ADA Caries Risk Assessment Form are questionnaires that can be presented to the patient. Cariogram is an internet-based educational program that illustrates the caries risk of an individual and possible interactions between various caries-related factors. All three of these assessment methods intend to provide the patient with educational information that may help them reduce their caries risk over time and offer the oral health care provider a means to evaluate an individual patient's caries risk status.
4. The assessment methods seek to provide practical clinical guidelines to managing dental caries based upon individual risk profile. These methods are based upon the best evidence at this time and can be used to implement evidence-based caries management for any patient.[80,82]
5. Example of A Caries Risk Assessment Form
   - a. In 2007, a group of experts from across the United States laid out the basis of a caries risk assessment system which outlines the step-by-step procedures and philosophies, known today as "Caries Management by Risk Assessment" (CAMBRA).[82]
   - b. In 2021, the same original group of experts released an update on the most current evidence-based protocol for caries risk assessment (CRA) and for caries management for children ages 0-6 years and adults above the age of 6 years.[85] Much of the current evidence substantiates that CRA is a valuable assessment tool that identifies certain risk factors which may predispose an individual to developing carious lesions, aids the oral health care provider in making patient-centered decisions for successful caries management, and empowers the patient with the knowledge to make careful informed decision(s) based on the most current evidence.
   - c. The CAMBRA system includes a caries risk assessment (CRA) that is routinely performed as part of the patient assessment. Since dental caries is a multifactorial disease, the caries risk assessment system is based on the individual's (1) caries risk profile (the likelihood of the patient developing new carious lesion), (2) protective factors (such as an individual's consumption of fluoridated drinking water, saliva production, the use of fluoridate tooth paste), (3) risk factors (such as frequent snacking of fermentable carbohydrates and amount of visible plaque present in the mouth), and (4) disease indicators (which is past and current level of caries destruction that is apparent in an individual's mouth). Based on the information ascertained from a CRA, the clinician uses the CAMBRA system to determine the individual's caries risk level (low, moderate, or high), which in turn guides the clinician to tailor a personalized caries management approach for each individual patient. **Figures 31-6A** and **Figure 31-6B** show the CAMBRA Caries Risk Assessment guide for children 0-6 years and for individuals above the age of 6 through adult, respectively.

Table 1 (Part 1). Updated CAMBRA Caries Risk Assessment form# for ages 0–6 years (January 2021)##. Refer to the second page of this form (part 2) for instructions for use as guidelines for caries risk assessment.

Patient Name:	**Reference Number:**		
Provider Name:	**Date:**		
Caries risk assessment component	**Column 1 Score: –1**	**Column 2 Score: +2**	**Column 3 Score: +3**
Biological or environmental risk factors* Question items		Check if Yes**	
1. Frequent snacking (more than 3 times daily)		▨	
2. Uses bottle/non-spill cup containing other than water or milk		▨	
3. Parent/primary caregiver or sibling has current decay or a recent history of decay (see high risk description below)		▨	
4. Family has low socioeconomic &/or low health literacy status		▨	
5. Medications that induce hyposalivation		▨	
Protective factors - Question items	Check if Yes**		
1. Lives in a fluoridated drinking water area	▨		
2. Drinks fluoridated water	▨		
3. Uses fluoride (F)-containing toothpaste at least two times daily—a smear for ages 0–2 years and pea size for ages 3–6 years of 1,000 ppm F.	▨		
4. Has had fluoride varnish applied in the last 6 months	▨		
Biological risk factors - Clinical exam*		Check if Yes**	
1. Heavy plaque on the teeth		▨	
Disease indicators – Clinical exam			Check if Yes**
1. Evident tooth decay or white spots			▨
2. Recent restorations in last 2 years (new patient) or the last year (patient of record)			▨
Column total score (Columns 2 + 3 –1):	**Column 1 Total:**	**Column 2 Total:**	**Column 3 Total:**
Yes's in columns 1 and 2 only: use the caries balance-below Yes or yes's in column 3 likely indicates high or very high risk			

Final Overall Caries Risk Assessment Category (check) determined as per guidelines:

LOW ☐ **MODERATE** ☐ **HIGH** ☐ **VERY HIGH** ☐

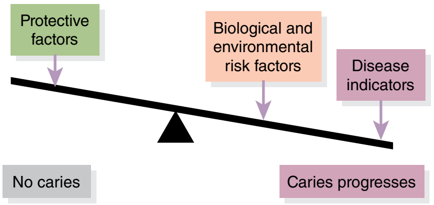
Table 1 (Part 2). Caries Risk Assessment Guidelines 0–6 years. Assessing the caries risk as low, moderate, high or very high.

The dental caregiver has the responsibility of making a caries risk assessment and then deciding on a caries management plan for the patient that leads from the risk assessment and a personalized assessment of the needs of the individual patient. These guidelines can assist in the process.

1. ***Low risk.*** If there are protective factors, very few or no risk factors, no disease indicators, and the protective factors prevail, the patient is at low risk.
2. ***Moderate risk.*** If there are no disease indicators and the risk factors and protective factors appear to be balanced then a moderate caries risk determination is appropriate. If in doubt move the moderate to a high classification.
3. ***High risk.*** If there is a "YES" in column 3 (one or both disease indicators) the patient is very likely at high risk. Even if there are no "yes" disease indicators the patient can still be at high risk if the risk factors definitively outweigh the protective factors. Parent or caregiver with current or recent dental decay most likely indicates high caries risk for the child.
4. ***Very high risk.*** If the above process indicates high risk and the existing or recent decay is severe and/or extensive a designation of "very high" caries risk is appropriate and will guide a more aggressive caries management plan.

Any items checked "yes" should also be used as topics to modify behaviour or determine additional therapy.

Use the following modified caries balance to visualize the overall result and determine the risk level. It may be helpful to allocate scores for each "yes" checked on the risk assessment form with a score of –1 for yes's in column 1, and +2 and +3 respectively for yes's in columns 2 and 3. The final total will help guide the risk level decision. **Low** = –4 to –1; **Moderate** = 0 to +3; **High** = +4 to +13; **Very high** = +14 to +18 and/or is a high risk level plus extensive and/or severe recent or existing decay.



Additional caries-related components for caries management and caregiver/patient counseling. Record in patient chart at each visit.

Dietary counseling to reduce frequency and amount of fermentable carbohydrates, especially sucrose, fructose (high fructose corn syrup) and continual fruit juice (e.g., apple juice). Record number and type of daily snacks, drinks and juices used.

Bottle used continually, bottle used in bed or nursing on demand. Record details provided.

Fluoride (F) toothpaste use. Note frequency and amount used at each visit.

Record all recommended therapy such as F toothpaste, F varnish, use of silver diamine fluoride in appropriate cases. Record usage provided by parent/caregiver.

Record medications at each visit and check for changes.

Record participation in assistance programs such as "school lunches," "head start," appropriate to the state or country.

Child has developmental problems/child has special care needs (CHSCN).

Inadequate saliva flow and related medications, medical conditions, or illnesses.

Discuss self-management goals with caregiver/patient and set two goals together at each visit. Provide in writing.

**Biological and environmental risk factors are split into a) question items, b) clinical exam*

***Check the yes answers in the appropriate column. Shading indicates which column to place the appropriate yes. Assess the caries risk as per instructions in Table 1 (part 2).*

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Figure 31-6A Caries Risk Assessment Form.

Table 2 (Part 1). Updated CAMBRA Caries Risk Assessment form[#] for ages 6 year through adult (January 2021)[##]. Refer to the second page (part 2) for instructions for use as guidelines for caries risk assessment.

Patient Name:	**Reference Number:**		
Provider Name:	**Date:**		
Caries risk assessment component	**Column 1 Score: −1**	**Column 2 Score: +2**	**Column 3 Score: +3**
Protective factors – Question items	**Check if Yes***		
1. Fluoridated water			
2. F toothpaste at least once a day			
3. F toothpaste 2X daily or more			
4. 5,000 ppm F toothpaste			
5. F varnish last 6 months			
6. 0.05% sodium fluoride mouthrinse daily			
7. 0.12% chlorhexidine gluconate mouthrinse daily 7 days monthly			
8. Normal salivary function			
Biological or environmental risk factors–Question items		**Check if Yes***	
1. Frequent snacking (>3 times daily)			
2. Hyposalivatory medications			
3. Recreational drug use			
Biological risk factors – Clinical Exam			
4. Heavy plaque on the teeth			
5. Reduced salivary function (measured low flow rate)**			
6. Deep pits and fissures			
7. Exposed tooth roots			
8. Orthodontic appliances			
Disease Indicators – Clinical exam			**Check if Yes***
1. New cavities or lesion(s) into dentin (radiographically)			
2. New white spot lesions on smooth surfaces			
3. New non-cavitated lesion(s) in enamel (radiographically)			
4. Existing restorations in last 3 years (new patient) or the last year (patient of record)			
Column total score (Columns 2 + 3 −1):	**Column 1 Total:**	**Column 2 Total:**	**Column 3 Total:**
Yes in column 3 likely indicates high or extreme risk Yes's in columns 1 and 2: use the caries balance-below **Hyposalivation plus high risk factors = extreme risk			

Final Overall Caries Risk Assessment Category (check) determined as per guidelines below

LOW ☐ **MODERATE** ☐ **HIGH** ☐ **VERY HIGH** ☐

Table 2 (Part 2). Caries Risk Assessment Guidelines for ages 6 years through adult. Assessing the caries risk as low, moderate, high or extreme.

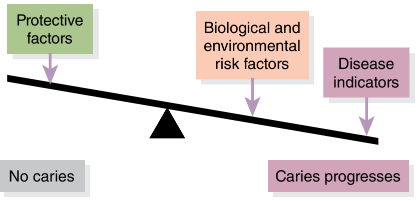
The dental caregiver has the responsibility of making a caries risk assessment and then deciding on a caries management plan for the patient that leads from the risk assessment and a personalized assessment of the needs of the individual patient. These guidelines can assist in the process.

Determining the caries risk as low, moderate, high or extreme - guiding principles

1. ***Low risk.*** If there are no disease indicators, very few or no risk factors and the protective factors prevail, the patient is most likely at low risk. Usually this is obvious.
2. ***Moderate risk.*** If the patient is not obviously at high, or extreme risk and there is doubt about low risk, then the patient should be allocated to moderate risk and followed carefully, with additional chemical therapy added. An example would be a patient who had a root canal as a result of caries 4 years ago, and has no new clinical caries lesions, but has exposed tooth roots and only uses a fluoride toothpaste once a day.
3. ***High and extreme risk.*** One or more disease indicators most likely signals at least high risk. If there is also hyposalivation the patient is likely at extreme risk. Even if there are no positive disease indicators the patient can still be at high risk if the risk factors definitively outweigh the protective factors. Think of the caries balance: visualize the balance diagram as illustrated below.

Any items checked "yes" should also be used as topics to modify behavior or determine additional therapy.

Use the following modified caries balance to visualize the overall result and determine the risk level. It may be helpful to allocate scores for each "yes" checked on the risk assessment form with a score of −1 for yes's in column 1, and +2 and +3 respectively for yes's in columns 2 and 3. The final total will help guide the risk level decision. **Low** = −8 to −2; **Moderate** = −1 to +2; **High** = +3 to +17; **Extreme** = +18 to +30 and/or is a high risk level plus measured or observed hyposalivation. Use the caries balance to visualize the overall result and determine the risk level for the individual patient.



Additional caries-related components for caries management and caregiver/patient counseling. Record in patient chart at each visit.

Dietary counseling to reduce frequency and amount of fermentable carbohydrates. Record number and type of daily snacks, drinks and juices used.

Oral hygiene and fluoride (F) toothpaste use. At each visit note frequency and amount used.

Record all recommended therapy such as F toothpaste, F varnish, chlorhexidine and usage by patient.

Record medications at each visit and check for changes.

Record participation in assistance programs such as "school lunches," "head start," appropriate to the state or country.

Child or adult has developmental problems or special care needs (CHSCN).

Inadequate saliva flow and related medications, medical conditions, or illnesses.

Discuss self-management goals with caregiver/patient and set two goals together at each visit. Provide in writing.

**Check the yes answers in the appropriate column. Shading indicates which column to place the appropriate yes. Assess the caries risk as per instructions in Table 2 (part 2) below.*

***Hyposalivation plus high risk factors = extreme risk.*

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Figure 31-6B *(Continued)*

Modified from Featherstone JDB, Crystal YO, Alston P, et al. Evidence-based caries management for all ages–practical guidelines. *Front Oral Health.* 2021;2:Article 657518. doi:10.3389/froh.2021.657518. This material may be used free of charge for the purposes of patient care, education, academic works, research, health promotion, health policy and related activities. However, permission must be obtained before this material is used for commercial purposes. Copyright © 2022 The Regents of The University of California. CAMBRA® is a trademark of the Regents of The University of California. Except where otherwise noted, this content is licensed under a Creative Commons 4.0 International license (CC BY-NC-ND 4.0).

b. **Caries Risk Determination and Management**
   1. **Caries Risk Determination.** Assigning a "caries risk level" to a patient is the initial step in managing the disease process.
      a. Low caries risk is assigned based on clinical judgment following an evaluation of the risk factors and protective factors of the patient. Salivary testing is optional based on the patient's risk profile.
      b. Moderate caries risk is assigned based on clinical judgment following an evaluation of the risk factors and protective factors of the patient and/or presence of caries or exposure of root surfaces. Salivary testing is optional based on the patient's risk profile.
      c. High caries risk is signified by the presence of any one of the following: clinical (visible) evidence of cavities or radiographic evidence of caries extending past the dentinoenamel junction and into the dentin, presence of multiple root caries lesions, radiographic interproximal enamel lesions, white spots on smooth surfaces, or restorations in the last 3 years, specifically if the lesions/restorations are on root surfaces. Salivary and bacterial testing is recommended.
      d. Extreme caries risk is high caries risk and severe salivary gland hypofunction (salivary flow rate of less than 0.5 mL/minute for stimulated saliva or less than 0.2 mL/minute for unstimulated saliva). Salivary and bacterial testing is recommended.
   2. **Evidence-Based Caries Management.** Following caries risk determination, the next step is to tailor a patient-centered treatment plan based on the individual's risk level.
      a. Low-caries-risk patients should use fluoride toothpaste twice daily; professional topical fluoride applications are optional. Bacterial and salivary tests are not necessary so long as the patient remains in the low-risk category. Most periodontal maintenance patients are not considered low risk because exposed roots are a primary risk factor for root caries.
      b. Moderate-caries-risk patients should use fluoride toothpaste twice daily, rinse with a 0.05% sodium fluoride mouthrinse, and receive fluoride varnish applications at each 3- to 4-month recall visit. Bacterial and salivary tests may be optional so long as the patient remains in the moderate-risk category.
      c. High-caries-risk patients should use a prescription of 1.1% to 1.5% sodium fluoride toothpaste twice daily and receive one to three topical fluoride varnish applications every 3 months. Fluoride varnish should be applied at 3-month intervals. Bacterial and salivary tests are recommended.
      d. Extreme-caries-risk patients receive the same CAMBRA therapies as high-risk patients. In addition, baking soda rinses, 0.5% sodium fluoride rinses, and calcium/phosphate pastes may be recommended.

Chapter Summary Statement

The intent of periodontal maintenance is to ensure that periodontal health is preserved following successful completion of nonsurgical and/or surgical periodontal therapy of a periodontitis patient. In most dental offices, the dental hygienist plays a major role during each periodontal maintenance visit. At each periodontal maintenance visit, the dental hygienist performs a thorough patient interview that reviews and updates the patient's medical history, clinical assessment, evaluation of effectiveness of self-care, identification of treatment needs, periodontal instrumentation, patient counseling, and application of fluorides. Additionally, a thorough oral cancer screening evaluation should be part of any dental check-up due to the importance of early detection. Currently, the most frequently recommended interval for periodontal maintenance is every 3 months. However, the recall interval may be customized to the treatment goals of an individual patient, depending on the patient's compliance level and individual periodontitis risk profile. Recurrence of periodontitis in treated patients with the need for additional active periodontal treatment is always a possibility. Patient compliance with periodontal maintenance recommendations is poor, but strategies can be employed to improve compliance. Root caries is a complication in many treated periodontitis patients. Several caries risk assessment methods are available to the oral health care provider to use to identify the caries risk profile of an individual patient. Based on the individual's caries risk assessment, the oral health care provider can then determine the appropriate steps to ensure effective management of the carious lesion(s).

Section 6

Focus on Patients

Clinical Patient Care

CASE 1

Your dental team has just completed a reevaluation of the results of nonsurgical therapy for a patient with generalized Stage I periodontitis. The findings of the reevaluation reveal that the periodontitis appears to be under control and that periodontal maintenance is the next logical step. When should the first maintenance appointment be scheduled, and what factors should be considered when assigning this maintenance interval?

CASE 2

One of your dental team's Stage II periodontitis patients has recently undergone periodontal surgery and now has several sites of gingival recession exposing tooth roots. Unfortunately, this patient has had a high incidence of both coronal and root caries over the past few years. What measures might your team take to minimize the risk of further root caries in this patient?

CASE 3

A patient who has been treated for Stage II periodontitis by your team has been followed for periodontal maintenance for more than 3 years. During each maintenance visit, there have been no indications of recurrence of the periodontitis. The patient calls you before her next maintenance visit to inform you that she has just been diagnosed with diabetes mellitus. She looked up diabetes on the Internet and now wants to know if this will affect her periodontal condition. How should your dental team respond to the patient's concern?

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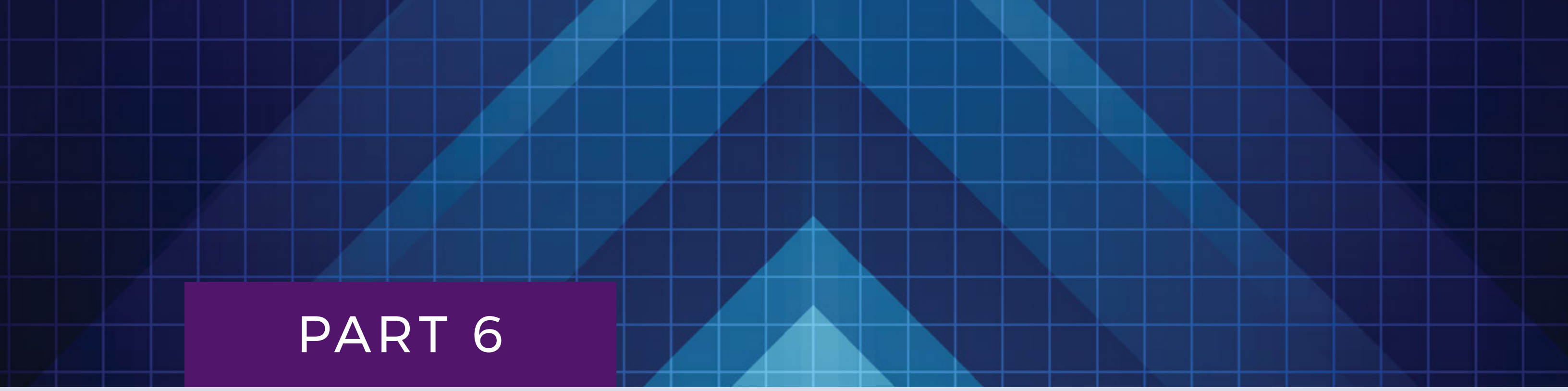

PART 6

Other Aspects of the Management of Patients with Periodontal Diseases and Peri-Implant Diseases/Conditions

CHAPTER 32 Documentation and Insurance Reporting of Periodontal Care 737

CHAPTER 33 Future Directions for Management of Periodontal Patients 755

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CHAPTER 32

Documentation and Insurance Reporting of Periodontal Care

- Section 1 Legal Issues in the Provision and Documentation of Care
  - Concepts of Malpractice and Tort Law
  - Areas of Potential Liability
- Section 2 Documentation of Periodontal Care
  - Principles for Thorough Documentation
  - Pitfalls in Documentation
- Section 3 Computer-Based Electronic Health Records
  - Advantages of Computerized Patient Records
  - Cautions Regarding Computerized Records
- Section 4 Procedural Codes for Periodontal Treatment
  - Procedural Coding Overview
  - Procedural Codes for Nonsurgical Periodontal Services
- Section 5 Focus on Patients
  - Clinical Patient Care
  - Ethical Dilemma

CLINICAL APPLICATION

The maxim "*Sloppy documentation is equated with sloppy care*" rings true when it comes to the ethical and professional responsibility of all dental clinicians to properly record a thorough and accurate written or typed report in each patient's record as a routine part of periodontal care. A complete and accurate entry in the patient's records is essential for fostering continuity of care and ensuring the continual delivery of quality dental care. It also provides a standard means of communication between two different providers and/or between a provider and patient about the health status, past treatment rendered, recommended treatment options, and anticipated outcomes of treatment. Moreover, legal ramifications related to the quality of the documentation in a patient's records require dental clinicians to have a thorough understanding of how to document patient interactions accurately. Additionally, this chapter provides guidance in the use of American Dental Association (ADA)–approved terminology for procedural coding.

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LEARNING OBJECTIVES

- Explain the foundations of tort law and how it applies to the profession of dentistry.
- Define the term *liability* as it applies to the provision of periodontal care.
- Describe situations in the dental office that trigger liability for dental hygienists.
- Define the terms *intentional torts* and *negligence* and provide examples of each.
- In a clinical setting, be able to thoroughly document a patient's dental record that includes updated health history, treatment options, history of patient noncompliance and cancellations, refusal of treatment, and follow-up conversations about the proposed treatment, potential risks and benefits of each option, and treatment alternatives.
- Explain the use of procedural codes and forms in periodontal care.
- Be able to explain the advantages and disadvantages of computer-based electronic health records.

KEY TERMS

Standard of care
Liability
Malpractice
Tort
Intentional torts
Negligence
Upcoding

Section 1

Legal Issues in the Provision and Documentation of Care

It is important for every dental clinician to practice to the highest established standards of care, not only to ensure the safety of the patient receiving treatment but also to avoid costly malpractice litigation.[1] Potential liability is a reality for every health care provider. Although patients can sue any health care provider for a variety of reasons, the success of such a suit often depends on the quality of the chart notes. Hence, the dental practitioner has a moral and ethical obligation not only to deliver high-quality care, but also to maintain thorough and accurate chart notes for each patient visit to protect the practice against liability.

Concepts of Malpractice and Tort Law

1. **Standard of Care.** The legal definition of **standard of care** varies in North America. In general terms, dental health care providers are required to exercise the same degree of skill and care as could reasonably be expected of a prudent dental health care provider of the same experience and standing.[1,2]
2. **Liability.** In the context of health care, **liability** is a health care provider's obligation or responsibility to provide services to the patient. The health care provider's liability should account for the possibility of being sued if the individual receiving the services feels as if he or she has been treated improperly or negligently.
3. **Malpractice.** **Malpractice** is the improper or negligent treatment by a health care provider that results in injury or damage to the patient.[3,4]
4. **Tort.** The legal basis for most lawsuits in dental and dental hygiene practice is founded on tort law. A **tort** is a civil wrong where a person has breached a duty to another. A tort is the law that permits an injured person to recover compensation from the person who caused the injury.
5. **Intentional Torts.** **Intentional torts** are actions designed to injure another person or that person's property. There are many specific types of intentional torts, including the following:
    a. Battery is the unlawful and unwanted touching or striking of one person by another, with the intent of bringing about a harmful or offensive contact. Forceful discipline of unruly children in the dental chair could be construed as battery.

   b. Assault is an unlawful threat or attempt to do bodily injury to another. A doctor who treats a minor patient without proper parental or guardian informed consent could be charged with assault or battery.
   c. Infliction of emotional distress. An example is talking in a loud or harsh voice to an unruly child.
   d. Fraud is deception carried out for the purpose of achieving personal gain while causing injury to another party.
   e. Misrepresentation occurs when a health care provider deliberately deceives a patient about possible outcomes.
   f. Defamation is communication to third parties of false statements about a person that injure the reputation of or deter others from associating with that person. For example, a dental hygienist learns that another hygienist has been making disparaging comments about the quality of care that he or she provides. The hygienist being disparaged could sue for defamation.
   g. Trespass is to infringe on the privacy, time, or attention of another. An example is discussing a patient's personal information with someone without the patient's consent.
   h. Defamation by computer. Email correspondence and other written documents are discoverable in court; avoid making disparaging remarks in email communications.

6. **Negligence.** **Negligence** is a failure to exercise reasonable care to avoid injuring others. It is the failure to do something that a reasonable person would do under the same circumstances, or the doing of something a reasonable person would not do. Negligence is characterized by carelessness, inattentiveness, and neglectfulness rather than by a positive intent to cause injury.[2]
   a. Negligence differs from an intentional tort in that negligence does not require the deliberate intent to commit a wrongful action; instead, the wrongful action itself is sufficient to constitute negligence.
   b. Examples of negligence include accidentally spilling a caustic, tissue-damaging chemical (e.g., concentrated sodium hypochlorite) on a patient, failing to update the patient's health history resulting in the patient's health being jeopardized, and incorrect treatment of periodontal disease. Professional liability insurance typically covers only unintentional torts or negligence.
7. **Upcoding.** **Upcoding** refers to reporting a higher level of service than was actually performed to a third-payer payer. An example of upcoding would be if a provider performed an adult prophylaxis (D1110 code) procedure on a patient, but billed the patient's insurance company using the more expensive scaling and root planning code (D4341 code) (the fee for a quadrant of scaling and root planning is about two to three times the fee of a prophylaxis). Upcoding is a fraudulent medical billing practice where the intent is to provide false information which misrepresents the actual procedure performed by billing the insurance carrier a code with a higher fee. The clinician should be mindful that upcoding is unethical, dishonest, and constitutes as dental fraud and abuse. Consequently, dental boards have been known to levy serious disciplinary measures, including revocation of license, for clinicians who have been found guilty of upcoding.

Areas of Potential Liability

A court judges a practitioner's alleged negligence by basing its verdict on a concept known as the *reasonably prudent person or professional standard of care*, which is defined as the standard of care that is recognized as acceptable and appropriate by reasonably prudent similar providers. Using this concept, the court compares the alleged actions of the defendant to how other similar experts who act in good faith would perform their duties when in the same situation. For example, the reasonably prudent professional standard of care for periodontal charting is that every adult patient will have a six-point periodontal charting with all numbers recorded at least once per year. Even if the oral health care provider is unaware of the current standard of care, failure to perform the aforementioned periodontal assessment will not hold up in court and constitutes as a form of negligence on the part of the provider. The top 10 areas of potential liability for dental hygienists are summarized in **Box 32-1**.

Box 32-1 Top 10 Areas of Potential Liability for Dental Hygienists

1. Failure to ask and document whether the patient has taken his or her premedication.
2. Failure to detect and document oral cancer.
3. Failure to update and document the patient's medical history.
4. Failure to detect and thoroughly document the presence of periodontal disease.
5. Injuring a patient.
6. Failure to document treatment thoroughly in the patient chart or computerized record.
7. Failure to protect patient privacy or divulging confidential patient information.
8. Failure to inform the patient about treatment options and the consequences of nontreatment.
9. Practicing outside the legal scope of practice. All dental hygienists should be well informed about the state practice act and follow the rules and regulations explicitly.
10. Failure to provide care that meets the established standards of care.

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Section 2

Documentation of Periodontal Care

The dental chart is a medicolegal record that serves as the practitioner's first line of defense in a malpractice suit. When a patient decides to file a lawsuit, the dental chart becomes the single most important piece of information relative to the suit. Faulty records and poor documentation can be the most important reason for the loss of a lawsuit.[5] At the completion of each patient visit, the oral health care provider should compose a detailed and accurate note that includes a patient's updated medical history, history of chief complaint, dental diagnoses, overall treatment plan, and plan for next visit. Recommendations for thorough documentation are summarized in **Table 32-1** and **Box 32-2**.

Table 32-1 Documentation Guidelines

Action	Why It Is Recommended
Format: ■ Write on the proper form or computer document. ■ Write or print legibly in blue or black ink. ■ Use correct grammar, spelling, and standard dental terminology. ■ Date each entry correctly.	■ It is important to write or print legibly to avoid miscommunication. (Some lawyers infer sloppy care from sloppy entries or charting.) ■ The date that actions occur or observations are made is a legal account of care provided.
Content: ■ Only record care that you have given or observations that you have made. Do not make entries for another care provider. ■ Enter information in a complete, accurate, concise, and factual manner. Entries may include the: ■ Reason for today's appointment ■ Through documentation of medical and dental history ■ Patient's chief complaint ■ Symptoms reported by the patient ■ Findings from the clinical periodontal assessment ■ Treatment options and recommendations ■ Patient treatment options ■ All assessment, educational, and treatment services rendered ■ Items given to the patient, such as home care aids ■ Date or interval of next appointment ■ Remember that in a liability situation, care or recommendations not recorded was not provided.	■ By making an entry in a dental record, you accept legal responsibility for that entry. ■ Use only commonly accepted dental terminology and standard abbreviations and symbols. Do not create your own abbreviations. Using correct terminology and abbreviations will prevent others from having to second-guess your meaning. ■ Proper and conscientious recording protects the patient, your employer, and you.

Action	Why It Is Recommended
Accountability: ■ Check the patient's name on the dental record and on the form where you are recording. ■ Always sign your first initial, last name, and title to each entry. ■ All entries should be written on the lines. No entries should be made in the margins or below the last line on the page. No lines should be skipped. ■ Do not use dittos, erasures, or correcting fluids. A single line should be drawn through an incorrect entry and words "mistaken entry" or "error in charting" should be printed above or beside the entry and signed. The entry should then be rewritten correctly. ■ Identify each page of the record with the patient's name and chart identification number. ■ Recognize that a patient record (chart) is permanent.	■ By verifying the patient's identification information, you ensure that you are recording the person's information on the correct record. ■ By signing your entry, you indicate that you are the person who needs to be consulted if further clarification of the information is needed. Additionally, signing your entry indicates that you accept legal responsibility for what you have written. ■ All lines should be used so that there is no opportunity for anyone to add information after a lawsuit is initiated. Making entries in the margins or below the last line on a page can cause juries to wonder if the entry was made at a later date. ■ Striking through an error is the only legal way to indicate a change in the dental record. Erasing or using correction fluid could be seen as an attempt to hide or change existing information.
Timing: ■ Record information in a timely manner. ■ Document care as closely as possible to the time of rendering treatment. ■ Do not record care as given before you have provided the care.	■ If you wait until the end of the day to record, you may forget important information. ■ Something may occur that prevents you from providing the anticipated care (e.g., the patient may become ill halfway through the appointment; the patient may refuse a fluoride treatment). If you record care as "provided" in the dental record, but then do not actually complete this care, you will have committed fraud.
Confidentiality: ■ Clinicians using patient records are bound professionally and ethically to keep in strict confidence all information they learn by reading patient records.	■ Individuals have a moral and legal right to expect that the information contained in their patient dental record will be kept private.

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Box 32-2 Determining How Much to Document: The Amnesia Test

Imagine that you have amnesia and cannot remember any of the treatment that you have performed for any of your patients since you started to work in a periodontal practice 5 years ago! Would you be able to read any one of your patient charts and be able to:

- Remember every assessment, educational, and treatment procedure that the patient has undergone and why this treatment was necessary?
- Remember what additional treatment has been recommended and accepted by the patient and know why this treatment is recommended?

Two criteria should dictate how much to document:

1. Include sufficient information that would allow you or any other clinician to determine exactly which assessment, educational, and treatment procedures were performed at each appointment; why that treatment was necessary; and what treatment is next—based solely on your documentation.
2. Include sufficient information that meets all the record keeping requirements of your state board.

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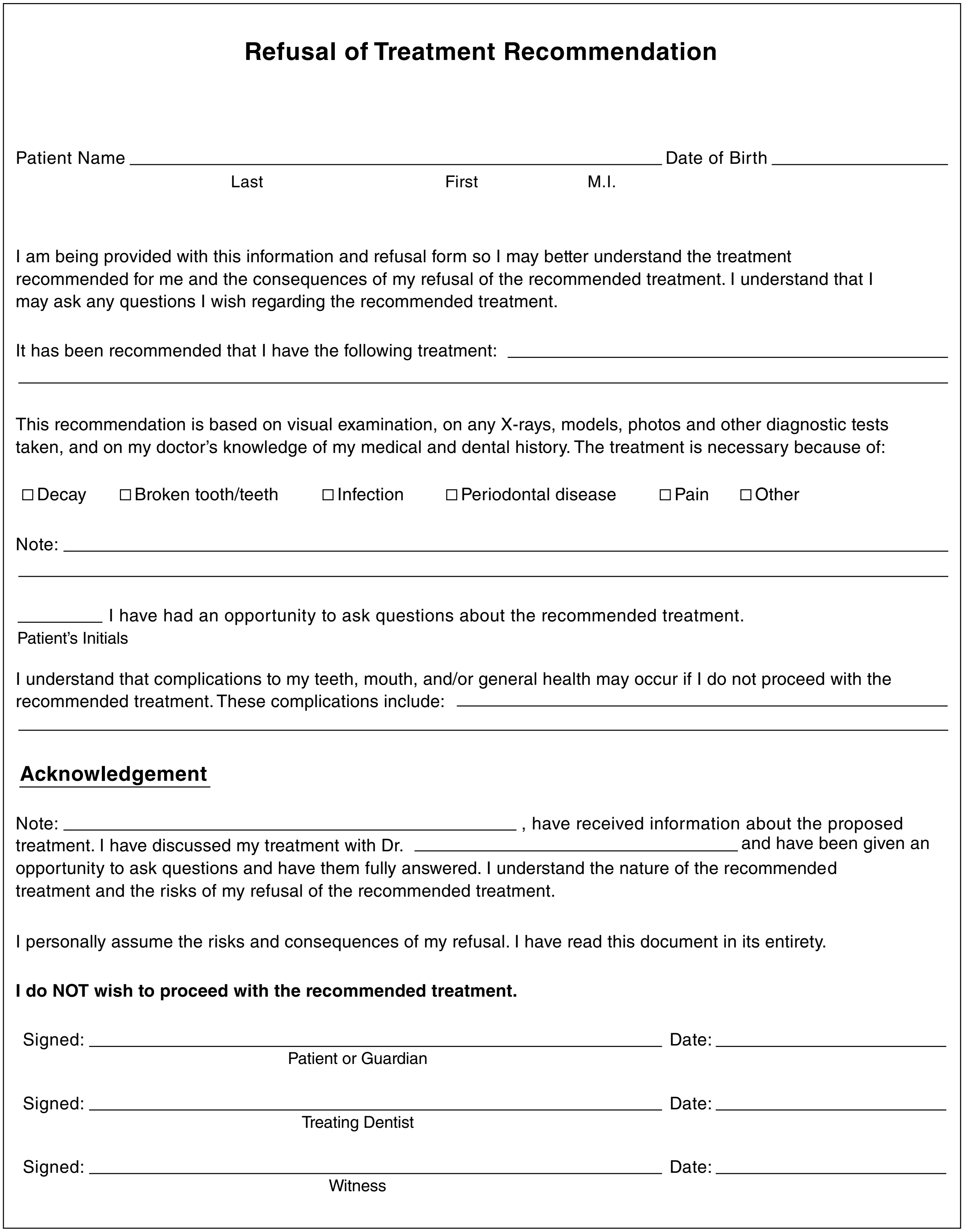
Principles for Thorough Documentation

1. **Recommendations for Thorough Documentation.** Tonner[6] recommends several principles that every dental professional should follow when documenting periodontal treatment in the patient chart or computerized record.
    a. **General Guidelines for Chart Entries**
        1. All entries should be complete and accurate using accepted dental terminology and abbreviations. Chart Entry-1 is an example of a complete chart entry.
        2. It is helpful to organize the services documented in sequential order so that no information is omitted.
        3. If handwritten, entries should be legible and in permanent ink.
        4. The health care provider making the entry should sign the entry with his or her first initial, last name, and title. Because many different people write in the patient chart, it is important that each entry be signed. If there are multiple dentists in the practice, the dentist that examines the hygienist's patient should be identified also.
        5. Thorough chart entries provide valuable information for the next clinician who treats this patient.
        6. The patient should be thoroughly interviewed regarding his or her medical status at each visit. Patients may not volunteer information when they are taking a new medicine or if there has been a change in their medical history. Nevertheless, the dental provider is legally and ethically obligated to thoroughly document any changes to the patient's medical status at each visit.

    b. **Treatment Options.** The health care provider should document all treatment options presented to the patient.
    c. **Appointment Schedule and Chart Entries**
        1. Chart entries should be consistent with the appointment schedule.
            a. With most dental software and computer scheduling, the patient's name must be on the schedule to make a chart entry.
            b. With manual appointment books, however, entries can be erased and changed.
        2. In the event of a lawsuit, doubt may be cast on the reliability of the office's records if the treatment dates in the chart do not match the appointment book entries.
        3. If the patient is being seen on an emergency basis, this circumstance should be recorded in the chart.

    d. **Cancellations and Missed Appointments.** All communication of any type relating to cancellations and missed appointments should be recorded in the patient chart. Infrequent periodontal maintenance appointments can lead to a recurrence and progression of periodontal disease. Chart Entry-2 and Chart Entry-3 provide examples of how to document missed and cancelled appointments.
    e. **Patient Noncompliance and Refusal of Treatment**
        1. Patient noncompliance with recommendations, such as (1) inadequate self-care, (2) continued smoking, (3) failure to regulate diabetes, or (4) failure to follow specific instructions, can lead to disease progression. Noncompliance should be noted in the chart (Chart Entry-4).
        2. Instances when a patient opts not to have recommended treatment or declines a referral to a specialist should be documented. In such situations, it is recommended that patients sign a "Refusal of Treatment Recommendation" document. An example of a refusal of recommend treatment form is shown in **Figure 32-1**. Further, when a patient is referred to a specialist, it is recommended that a copy of the referral letter be kept in the patient chart. Chart Entry-5 provides an example of documentation of inadequate self-care.



Refusal of Treatment Recommendation

Patient Name ______________________________ Date of Birth ______________
Last First M.I.

I am being provided with this information and refusal form so I may better understand the treatment recommended for me and the consequences of my refusal of the recommended treatment. I understand that I may ask any questions I wish regarding the recommended treatment.

It has been recommended that I have the following treatment: ______________________________

This recommendation is based on visual examination, on any X-rays, models, photos and other diagnostic tests taken, and on my doctor's knowledge of my medical and dental history. The treatment is necessary because of:

☐ Decay ☐ Broken tooth/teeth ☐ Infection ☐ Periodontal disease ☐ Pain ☐ Other

Note: ______________________________

__________ I have had an opportunity to ask questions about the recommended treatment.
Patient's Initials

I understand that complications to my teeth, mouth, and/or general health may occur if I do not proceed with the recommended treatment. These complications include: ______________________________

Acknowledgement

Note: ______________________________ , have received information about the proposed treatment. I have discussed my treatment with Dr. ______________________ and have been given an opportunity to ask questions and have them fully answered. I understand the nature of the recommended treatment and the risks of my refusal of the recommended treatment.

I personally assume the risks and consequences of my refusal. I have read this document in its entirety.

I do NOT wish to proceed with the recommended treatment.

Signed: ______________________________ Date: ______________
Patient or Guardian

Signed: ______________________________ Date: ______________
Treating Dentist

Signed: ______________________________ Date: ______________
Witness

Figure 32-1 Refusal of Recommended Treatment Form. Shown above is one example of a refusal of recommended treatment form.

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f. **Follow-up Telephone Calls.** Patients appreciate a follow-up telephone call from the dentist or hygienist following a long or difficult treatment procedure. For hygienists, a good rule of thumb is to call any patient that required anesthesia for periodontal instrumentation. Follow-up telephone calls should be documented (Chart Entry-6).

Date	Treatment Rendered
1/10/23	Reason for visit: 3-month periodontal maintenance. Medical history update: Patient now taking 1 aspirin a day per his physician's recommendation. Chief complaint: none. Oral cancer exam: normal. Periodontal probing: changes noted in charting. Plaque: light, calculus: light, bleeding areas noted on periodontal chart. Perio maintenance: perio instrumentation of all 4 quads; ultrasonic and hand instrumentation. Plaque removal by patient using toothbrush and interdental brush. Patient tolerated all procedures well. Patient education: reviewed use of tufted dental floss around distal surfaces of maxillary and mandibular molars. Tray fluoride application 1.23% APF gel for sensitivity. 4 bitewing radiographs. Next maintenance visit in 3 months. *R. Zimmer, RDH*

Chart Entry-1. Complete Chart Entry. This chart entry is an example of a thorough chart entry that documents all the events of the patient's appointment.

Date	Treatment Rendered
1/10/23	Patient missed maintenance appointment because of illness. *R. Zimmer, RDH*

Chart Entry-2. Missed Appointment. This chart entry is an example of documentation of a missed appointment due to illness.

Date	Treatment Rendered
1/10/23	Telephoned patient to confirm her 3-month maintenance appt. Patient cancelled and said that she would call to reschedule later. I reminded her of the importance of regular maintenance. *R. Zimmer, RDH*

Chart Entry-3. Cancelled Maintenance Appointment. This chart entry provides an example of the documentation for a cancelled periodontal maintenance appointment.

Date	Treatment Rendered
1/10/23	Discussed options for smoking cessation. Patient stated that "he is not interested in quitting smoking." *R. Zimmer, RDH*

Chart Entry-4. Patient Noncompliance. This chart entry provides an example of the documentation for patient noncompliance with recommendations.

Date	Treatment Rendered
1/10/23	Patient reports brushing twice daily but "does not have time to use an interdental brush." Showed patient signs of periodontal inflammation in the interdental areas. Explained benefits of interdental plaque removal and several alternatives for interdental self-care. Patient decided that he was not interested and stated that "he only wants to brush." *R. Zimmer, RDH*

Chart Entry-5. Inadequate Self-Care. This chart entry is an example of documentation of inadequate self-care by a patient.

Date	Treatment Rendered
1/10/23	Telephoned patient at home this evening to check on her. Patient reports that she "has no bleeding and rates her pain as a 2, on a scale of 1 to 10." Reminded her to use warm saltwater rinse before bedtime. *R. Zimmer, RDH*

Chart Entry-6. Follow-up Telephone Call. This chart entry is an example of documentation of a follow-up telephone call after a long or difficult treatment procedure.

Date	Treatment Rendered
1/10/23	Px, Ex

Chart Entry-7. Incomplete Chart Entry. Although this hygienist may have been quite thorough in delivering care, the chart does not reflect that.

Date	Treatment Rendered
1/10/23	Patient reports that his "gums no longer bleed during brushing." Tissue color, tone, and texture are much improved from 3 weeks ago. *R. Zimmer, RDH*

Chart Entry-8. Patient Comments. This chart entry is an example of how to include a patient's comments at a periodontal maintenance visit.

Pitfalls in Documentation

1. **Common Problems in Documentation.** Tonner outlines four common pitfalls in documentation.[6]
   a. **Making Entries in Haste.** Chart Entry-7 is an example of an inadequate chart entry. For example, in her haste to stay on schedule, the dental hygienist simply forgets to record that she did a periodontal charting and evaluation. Later, if the patient develops periodontal disease, he may accuse the dental practitioner of failure to diagnose. The dentist or hygienist may state to a jury that a periodontal evaluation is done on every patient. In the eyes of a jury, however, if a procedure is not recorded in the patient chart, it was not performed.
   b. **Skipping Lines Between Entries or Writing in Margins**
      1. **Keeping in the Lines or Skipping Lines**
         a. Chart entries should be written with small enough strokes to be contained within the space provided.
         b. No lines should be skipped on a treatment record form. All lines should be used so that there is no opportunity for anyone to add information after a lawsuit is initiated.
      2. **Writing in Margins or Below the Last Line.** All entries should be written on the lines, and no entries should be written in the margins or below the last line on the page. Doing so can cause juries to wonder if the entry was made at a later date.
   c. **Altering Chart Entries.** The single most common cause of punitive damages in a dental malpractice suit is altering the chart.
      1. Correction fluid should never be used to correct an entry. If an error is made, a single line should be drawn through the incorrect entry so that it can still be read, the words "charting error" or "mistaken entry" written above it, and the correct entry made on the next available line. The revised entry should be signed by the dental health care practitioner.
      2. Additional information should never be added to an entry from a previous appointment. Juries perceive such added entries to be fraudulent and deceptive.
      3. Forensic ink-dating analysis allows an expert to determine the date that ink was used on a particular document. Therefore, it is foolhardy to add things at a later date to a patient chart in an attempt to avoid or win a lawsuit.

   d. **Not Clearly Indicating Patient Comments.** Quotation marks should be used to indicate patient comments. This is especially important when making follow-up telephone calls after a difficult or invasive procedure. A sample chart entry is shown in Chart Entry-8.

Section 3

Computer-Based Electronic Health Records

The development and implementation of computer-based electronic health records (abbreviated as EHR) present both challenges and opportunities for oral health care providers in a clinical setting.[7,8]

Advantages of Computerized Patient Records

1. **Organization and Data Gathering**
   a. An electronic health record is a digital version of a patient chart that provides real-time data which is instantly available to authorized users on a secure EHR platform.
   b. Standardization of clinical data. All staff members use the same templates for gathering data and the same abbreviations.
   c. Enhanced legibility. Handwriting can often be illegible, which increases liability risk for the clinician.
   d. Easier and faster access to information. Information is only a few keystrokes away.
   e. Enhanced use of clinical images and radiographs.
   f. Provision of new ways to analyze clinical information. For example, digital radiography allows the clinician to view radiographs with digital tools designed to enhance the image and visualize bone levels around teeth.
   g. Potential for greater security of patient data. Paper records are vulnerable to fire, earthquake, and water damage from flooding. Computer-based patient records, in contrast, can be backed up offsite to preserve data. Note that computerized data is more secure only if it is continuously backed up to an offsite location separate from the dental office.
2. **Processing of Information**
   a. Patient information is accessible across the network simultaneously. For example, business assistants can read input from clinicians and be ready for patient check-out before the patient reaches the business office.
   b. Facilitates submission of insurance claims. Computerized information facilitates submission of dental insurance claim forms to insurance companies.
3. **Communication**
   a. Faster and better multidisciplinary interaction with specialists. Successful treatment and management of the patient's periodontal condition requires a team approach involving the primary care (general) dental team, the periodontal dental team, and often, other dental specialists or physicians.
   b. Continuous communication among health care providers is critical to the success of diagnosis, treatment, and maintenance. Ongoing communication is needed because it is common for the patient to be treated in phases, going back and forth between the primary care dental practice and the periodontal practice. A computer-based patient record can greatly ease communication among dental health care providers.

Cautions Regarding Computerized Records

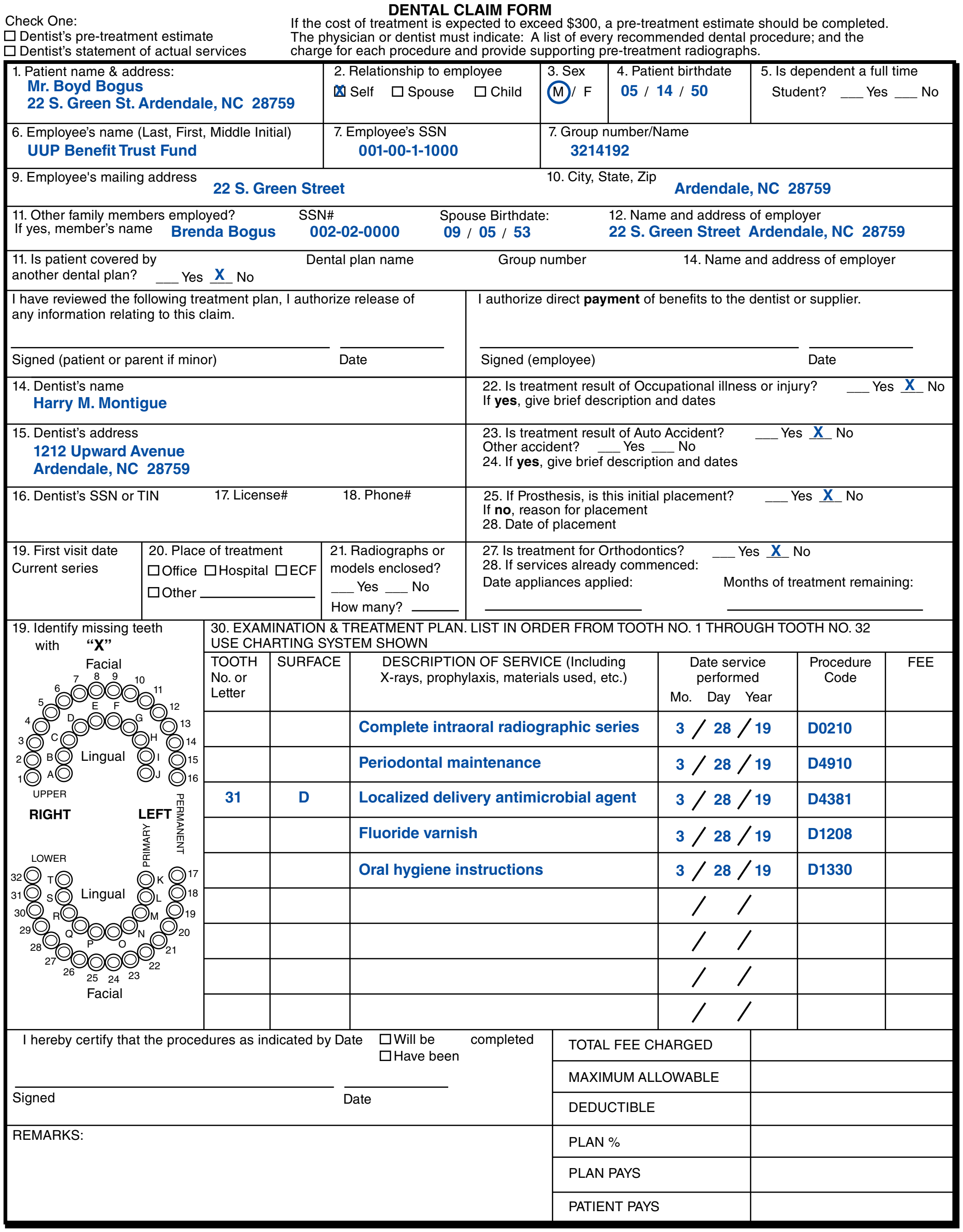
Even with advancing technologies, practitioners and staff must realize that the transition to computer-based patient records is not seamless, totally safe, or problem-free.

1. **Data Backup.** Anyone who has ever used a computer has experienced computers crashing, freezing-up, and/or frequently losing data. Hence, to avoid such situations, computer-based patient records must be backed up offsite to preserve data. Data should be continuously backed up to a secure offsite location.
2. **State Regulations.** In some states, computerized records may not eliminate the need to keep paper records due to legal requirements in those states. In such cases, the dental office may need to maintain patient data on paper and in computerized versions.

Section 4

Procedural Codes for Periodontal Treatment

This section highlights the importance of understanding various alphanumeric codes for dental procedures that cover oral health and dentistry. Such coding systems are essential in delivering a standardized and consistent way to document dental procedures. Coding systems are also used to convey important billing information to third party insurers. Claim submissions for dental care are submitted either electronically or by means of paper forms. An example of a completed dental insurance claim form is shown in **Figure 32-2**.


Facial
Lingual
UPPER
RIGHT
LEFT
PRIMARY
PERMANENT
LOWER
Lingual
Facial
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16
A B C D E F G H I J
17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 32
K L M N O P Q R S T

DENTAL CLAIM FORM

Check One:
☐ Dentist's pre-treatment estimate
☐ Dentist's statement of actual services

If the cost of treatment is expected to exceed $300, a pre-treatment estimate should be completed. The physician or dentist must indicate: A list of every recommended dental procedure; and the charge for each procedure and provide supporting pre-treatment radiographs.

1. Patient name & address: Mr. Boyd Bogus
22 S. Green St. Ardendale, NC 28759

2. Relationship to employee ☒ Self ☐ Spouse ☐ Child

3. Sex [✓ M] / F

4. Patient birthdate 05 / 14 / 50

5. Is dependent a full time Student? ___ Yes ___ No

6. Employee's name (Last, First, Middle Initial) UUP Benefit Trust Fund

7. Employee's SSN 001-00-1-1000

7. Group number/Name 3214192

9. Employee's mailing address 22 S. Green Street

10. City, State, Zip Ardendale, NC 28759

11. Other family members employed? If yes, member's name Brenda Bogus SSN# 002-02-0000 Spouse Birthdate: 09 / 05 / 53

12. Name and address of employer 22 S. Green Street Ardendale, NC 28759

11. Is patient covered by another dental plan? ___ Yes _X_ No Dental plan name Group number 14. Name and address of employer

I have reviewed the following treatment plan, I authorize release of any information relating to this claim.

Signed (patient or parent if minor) ____________ Date ______

I authorize direct **payment** of benefits to the dentist or supplier.

Signed (employee) ____________ Date ______

14. Dentist's name Harry M. Montigue

22. Is treatment result of Occupational illness or injury? ___ Yes _X_ No
If **yes**, give brief description and dates

15. Dentist's address 1212 Upward Avenue
Ardendale, NC 28759

23. Is treatment result of Auto Accident? ___ Yes _X_ No
Other accident? ___ Yes ___ No
24. If **yes**, give brief description and dates

16. Dentist's SSN or TIN 17. License# 18. Phone#

25. If Prosthesis, is this initial placement? ___ Yes _X_ No
If **no**, reason for placement
28. Date of placement

19. First visit date Current series

20. Place of treatment ☐ Office ☐ Hospital ☐ ECF ☐ Other ______

21. Radiographs or models enclosed? ___ Yes ___ No How many? ______

27. Is treatment for Orthodontics? ___ Yes _X_ No
28. If services already commenced:
Date appliances applied: ______ Months of treatment remaining: ______

19. Identify missing teeth with **"X"**

30. EXAMINATION & TREATMENT PLAN. LIST IN ORDER FROM TOOTH NO. 1 THROUGH TOOTH NO. 32 USE CHARTING SYSTEM SHOWN

TOOTH No. or Letter	SURFACE	DESCRIPTION OF SERVICE (Including X-rays, prophylaxis, materials used, etc.)	Date service performed Mo. Day Year	Procedure Code	FEE
		Complete intraoral radiographic series	3 / 28 / 19	D0210	
		Periodontal maintenance	3 / 28 / 19	D4910	
31	D	Localized delivery antimicrobial agent	3 / 28 / 19	D4381	
		Fluoride varnish	3 / 28 / 19	D1208	
		Oral hygiene instructions	3 / 28 / 19	D1330	
			/ /		
			/ /		
			/ /		
			/ /		

I hereby certify that the procedures as indicated by Date ☐ Will be ☐ Have been completed

Signed ____________ Date ______

REMARKS:

TOTAL FEE CHARGED	
MAXIMUM ALLOWABLE	
DEDUCTIBLE	
PLAN %	
PLAN PAYS	
PATIENT PAYS	

Figure 32-2 **Insurance Claim Form.** An example of a completed dental insurance claim form.

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Procedural Coding Overview

In the United States, the Code on Dental Procedures and Nomenclature, more commonly known as the CDT (Current Dental Terminology) Code, is a set of standardized alphanumeric codes for dental procedures that ensures consistency in documenting dental treatment. CDT codes are commonly used by practitioners to submit reimbursement claims to third-party payers.[9,10] Each alphanumeric code begins with the letter "D" (for dental) followed by four numbers. Further, each CDT code is organized into one of 12 sets of "categories of service," each with its own 5-digit alphanumeric codes, according to the discipline/specialty the individual code is related to. For example, the code for periodontal maintenance falls within the periodontics code series (D4000–D4999) and is designated by the CDT code D4910 (**Table 32-2**). CDT codes are very specific and should be reviewed carefully before specific dental treatment is coded.[10] Further, the clinician should stay abreast with any changes made to the CDT codes since the ADA conducts annual reviews, revisions, and additions to codes.

The CDT coding system is only applicable in the United States. Outside the United States, many other countries have devised their own unique coding systems to record services, communicate with the industry, and bill clients. For instance, Canada has developed their own 5-digit numeric codes (unlike the alphanumeric codes used in the United States) known as the Canadian Dental Procedure Codes. So, depending on which country the dental clinician practices, he/she will need to be accustomed to the specific coding system used for that particular country.

Table 32-2 Categories of Service

Categories of Service	Code Series	Examples of Codes Pertinent to the Practice of Dental Hygiene
Diagnostic	D0100–D0999	Periodic oral evaluation: D0120 Comprehensive oral evaluation: D0150 Comprehensive periodontal evaluation: D0180 Codes for radiographs
Preventive	D1000–D1999	Prophy, adult: D1110 Prophy, children: D1120 Fluoride varnish application: D1206
Restorative	D2000-D2999	N/A
Endodontics	D3000-D3999	N/A
Periodontics	D4000-D4999	Scaling and root planing four or more teeth per quadrant: D4341 Scaling and root planing one to three teeth per quadrant: D4342 Scaling in the presence of generalized moderate to severe gingival inflammation: D4346 Full-mouth debridement: D4355
Prosthodontics, removable	D5000–D5899	N/A
Maxillofacial prosthetics	D5900–D5999	N/A
Implant services	D6000–D6199	Implant maintenance procedures, including removal of prosthesis, cleansing of prosthesis, and abutments and reinsertion of prosthesis: D6080 Scaling and debridement in the presence of inflammation or mucositis of a single implant: D6081
Prosthodontics, fixed	D6200–D6999	N/A
Oral maxillofacial surgery	D7000–D7999	N/A
Orthodontics	D8000–D8999	N/A
Adjunctive general services	D9000–D9999	Application of desensitizing medicament: D9910

Data from American Dental Association. *CDT 2023: Current Dental Terminology*. American Dental Association; 2023:192.

Claim submissions for treatment rendered can be completed electronically or by means of paper forms. An example of a completed dental insurance claim form is shown in Figure 32-2.

Under the CDT system, all diagnostic procedures fall within the D0100–D0999 code series, whereas all preventive procedures (e.g., adult prophylaxis) are grouped within the D1000–D1999 code series. Therapeutic therapies fall under the D2000–D9999 code series. Each therapeutic code is grouped according to which discipline/specialty the specific code is related to.

Procedural Codes for Nonsurgical Periodontal Services

1. **Evolution of Dental Terminology**
    a. Members of the dental team should be aware that there is a continuous evolution of terminology used in dentistry and medicine.
    b. Traditionally in the dental literature, two terms have been used to describe the therapies employed to remove deposits from tooth surfaces. These terms are (1) *dental prophylaxis* and (2) *scaling and root planing*.
    c. Recently in the dental hygiene literature, increasing numbers of authors are using new terminology to describe periodontal instrumentation.
        1. The term *periodontal instrumentation* or *periodontal debridement* is suggested to replace the older terms *dental prophylaxis* and *scaling and root planing*.
        2. In dental hygiene literature, *periodontal instrumentation* is defined as the removal or disruption of plaque biofilm, its by-products, and biofilm-retentive calculus deposits from coronal surfaces, root surfaces, and within the pocket space, as indicated, for periodontal healing and repair.
2. **Procedural Codes for Insurance Reporting.** CDT continues to use the terms *prophylaxis* and *scaling and root planing* to describe periodontal instrumentation. Dental team members are required to apply the currently accepted procedural codes when filling out insurance forms and in communications with insurance companies or other third-party payers.
    a. **Examination Codes**
        1. **D0120: Periodic Oral Evaluation.** An evaluation performed on a patient of record to determine any changes in the patient's dental and medical health status since a previous comprehensive or periodic evaluation. This includes periodontal screening and may require interpretation of information acquired through additional diagnostic procedures.
        2. **D0150: Comprehensive Oral Evaluation**
        3. **D1354: Caries Arresting Agent.** This code is for the application of a caries arresting agent, such as silver diamine fluoride, on areas of exposed cementum.
        4. **D0180: Comprehensive Periodontal Evaluation—New or Established Patient.** This code is used for patients showing signs or symptoms of periodontal disease and for patients with risk factors such as smoking or diabetes. This examination code may be used when the hygienist performs a comprehensive periodontal evaluation including full-mouth, six-point probing and recording, charting of recession, furcations, tooth mobility, or tissue abnormalities once per year.
    b. **Currently Accepted Procedural Codes Pertaining to Periodontal Instrumentation**
        1. **D1110: Adult Prophylaxis (Four Quadrants)**
            a. **Definition.** Removal of supragingival and subgingival plaque, calculus, and stains from the tooth structures in the permanent and transitional dentition. It is primarily *preventive treatment* intended to control local irritating factors.
            b. **Indications**
                1. This code is indicated for non-periodontitis patients diagnosed with gingival health or non-periodontitis patients exhibiting signs of mild gingivitis.
                2. Supragingival and subgingival scaling on this type of patient usually can be completed in a single appointment.
                3. This code may also be used to describe and report the cleaning of complete dentures in edentulous patients.
        2. **D1120: Child Prophylaxis (Four Quadrants)**
            a. **Definition.** Removal of supragingival and subgingival plaque, calculus, and stains from the tooth structures in the primary and transitional dentition. Like the D1110 code, the D1120 code falls under the preventive category of service because it is intended to control local irritating factors.

   b. **Indications.** Same as outlined above for D1110. The only difference is that this code is applied when the prophylaxis is performed on a child who is aged 13 or younger.
3. **D4341: Periodontal Scaling and Root Planing—Four or More Teeth Per Quadrant**
   a. **Definition.** This procedure involves instrumentation of the crown and root surfaces to remove plaque and calculus from these surfaces. More important, unlike the D1110/D1120 codes, the D4341 is a *therapeutic code*, not a preventive one.
   b. **Indications.** A D4341 code is definitive treatment for patients exhibiting early stages of periodontitis or as a presurgical phase of treatment in more advanced cases.
4. **D4342: Periodontal Scaling and Root Planing—One to Three Teeth Per Quadrant**
   a. **Definition and Indications.** This code is essentially the same as the D4341 code, the difference being the number of teeth that are treated in a quadrant.
5. **D4910: Periodontal Maintenance**
   a. **Definition.** This code is a nonsurgical procedure that entails the removal of bacterial plaque and calculus from supragingival and subgingival regions, site-specific scaling and root planing where indicated, and polishing of the teeth.
   b. **Indications.** Unlike a dental prophylaxis (D1110/D1120), the D4910 code is reported for patients who currently exhibit *successfully stable periodontitis* following completion of definitive therapeutic periodontal therapy (e.g., nonsurgical scaling and root planing and/or surgical periodontal therapy). In other words, this is a *post-therapeutic code* that is performed to maintain the results of periodontal therapy. It is *not* a code to prevent disease like the D1110/D1120 codes.
   c. Although the D1110/D1120 codes and the D4910 code seem similar, they are, in fact, neither interchangeable nor should they be alternated in between recalls. Alternating codes is when a practitioner switches back and forth between using the D4910 code and the D1110 or D1120 code at each subsequent patient recall visit as a cost-saving measure for the patient (a prophylaxis is typically fully covered under a patient's insurance plan, whereas a periodontal maintenance may be covered partially or not at all.) Although alternating codes may seem patient-friendly, this constitutes as fraud and is judged as negligence on the part of the oral health care provider. As an example, if a patient has had a history of periodontitis that was previously treated (using the D4341/D4342 code or any periodontal surgical codes), then successfully managed (using the D4910 code in the past), and is presently in remission, then the appropriate code to report is the D4910 code, not the D1110/D1120 codes. This is because the patient who was previously diagnosed with periodontitis still has a reduced periodontium that needs to be maintained. Again, the saying "*once a periodontitis patient, always a periodontitis patient*" should be kept in mind because periodontitis is a chronic and long-term disease that is only kept in remission with frequent monitoring and periodic periodontal maintenance.
6. **D4355: Full Mouth Debridement to Enable Comprehensive Evaluation and Diagnosis.** This code should not be confused with the term *periodontal debridement* as used in dental hygiene literature. Full mouth debridement is the gross removal of plaque and calculus that interfere with the ability of the dentist to perform a comprehensive oral evaluation. Full mouth debridement refers to the removal of heavy supragingival calculus deposits *only* so that the clinic can perform the comprehensive evaluation and diagnosis. This is a preliminary procedure that will necessitate the need for additional periodontal instrumentation after the comprehensive evaluation and diagnosis is completed. Note that a D4355 code should not be completed on the same day that a D0150 code (comprehensive oral examination) or a D0180 code (comprehensive periodontal examination) is completed. This is because the gingival inflammation must subside before the oral health care provider can perform a more accurate assessment of the periodontium.
7. **D4921: Gingival Irrigation: Per Quadrant.** Irrigation of gingival pockets with medicinal agent, such as chlorhexidine or PVP-I. This code is not applicable when reporting use of mouth rinses or noninvasive chemical debridement.
8. **D5994: Periodontal Medicament Carrier with Peripheral Seal—Laboratory Processed.** A custom fabricated laboratory-processed carrier that covers the teeth and alveolar mucosa. Used as a vehicle to deliver prescribed medicaments for sustained contact with the gingiva, alveolar mucosa, and into the periodontal sulcus or pocket.

9. **D4346: Scaling in the Presence of Generalized Moderate or Severe Gingival Inflammation—Full Mouth, After Oral Evaluation**
    a. **Definition.** The removal of plaque, calculus, and stains from supra- and subgingival tooth surfaces when there is generalized moderate or severe gingival inflammation in the absence of periodontitis.
    b. **Indications**
        1. This code is indicated for gingivitis patients who have swollen, inflamed gingiva, generalized *suprabony* pockets, and moderate to severe bleeding on probing.
        2. The code should not be reported in conjunction with prophylaxis, scaling and root planing, or debridement procedures.
        3. Unlike a dental prophylaxis (D1110) which is preventive treatment, the D4346 procedure is therapeutic treatment.
10. **D0414: Laboratory Processing of Microbial Specimen to Include Culture and Sensitivity Studies, Preparation and Transmission of Written Report**
11. **D0600: Nonionizing Diagnostic Procedure Capable of Quantifying, Monitoring, and Recording Changes in Structure of Enamel, Dentin, and Cementum.** An example would be the use of a tool such as DIAGNOdent to aid in detection of caries.
12. **D6081: Scaling and Debridement in the Presence of Inflammation or Mucositis of a Single Implant, Including Cleaning of the Implant Surface, Without Flap Entry and Closure**
13. **D9910: Desensitization Medicaments.** The placement of medicament on the tooth to close dentinal tubules to decrease sensitivity.

c. **Codes for Radiographs.** The most common dental radiographs are:
    1. **D0210: Complete Intraoral Radiographic Series Including Bitewings**
    2. **D0220: Intraoral Periapical (First Film)**
    3. **D0230: Intraoral Periapical Film (Each Additional Film)**
    4. **D0240: Intraoral Occlusal Film**
    5. **D0250: Extraoral First Film, Such as a Cephalometric Film**
    6. **D0260: Extraoral Film (Each Additional Film)**
    7. **D0270: Single Bitewing Film**
    8. **D0272: Two Bitewing Films**
    9. **D0274: Four Bitewing Films**
    10. **D0330: Panoramic Film**
    11. **D0277: Vertical Bitewings—Seven to Eight Films**
    12. **D0350: Oral/Facial Images (Includes Traditional Photographs or Digital Images Obtained by Intraoral Cameras)**

d. **Codes for Topical Fluoride**
    1. **D1206: Topical Application of Fluoride Varnish**
    2. **D1208: Topical Application of Fluoride.** This code used to report prescription strength fluoride swishes, trays, isolates, or paint on fluorides, but not varnishes.

e. **Codes for Patient Counseling**
    1. **D1310: Nutritional Counseling for Control of Dental Disease.** Counseling on food selection and dietary habits as a part of treatment and control of periodontal disease and caries.
    2. **D1320: Tobacco Cessation Counseling for Control and Prevention of Oral Disease**
    3. **D1330: Oral Hygiene (Self-Care) Instructions.** Examples include tooth brushing technique, flossing, and the use of special oral hygiene aids.

Chapter Summary Statement

From a practical patient care standpoint and risk management perspective, the dental chart is an important medicolegal record that provides the *big picture* of the patient's medical/dental history and present-day condition(s), diagnoses, and treatment plan. All periodontal assessments, along with educational and treatment services, should be documented in the patient chart or electronic health record. If a patient files a malpractice lawsuit, the dental chart becomes the single most important piece of information related to the suit.

In the United States, CDT codes is a standardized system that achieves specificity and uniformity in accurately documenting dental treatment. Further, CDT codes are used to speed and simplify the reporting of dental treatments to third parties, such as private insurance companies or government-run health-insurance programs for qualified adults (such as Medicaid or Medicare). These codes are very specific and should be reviewed carefully before specific dental treatment is coded. Also, the clinician should keep abreast with the annual revisions made to the CDT codes.

Section 5

Focus on Patients

Clinical Patient Care

CASE 1

During a social gathering one evening, a dental hygienist tells her friend about an HIV-positive patient she had treated that day. As the news traveled down the grapevine and the patient learned that the hygienist had revealed his HIV status, he sued her. What specific charge could he bring against the hygienist? Would she be covered under the dentist/employer's malpractice coverage or her own personal malpractice coverage?

CASE 2

The dental hygienist performed an oral cancer screening on every patient, but she never wrote it in her progress notes. When a patient found out he had oral cancer, he sued his dentist and the dental hygienist. The patient had been seen for a prophylaxis and restorative care 6 months before his diagnosis of oral cancer, and the basis for his suit was that he felt the hygienist and dentist had been negligent in failing to detect the lesion. Why is it likely that the patient will win his suit against the dental practice?

CASE 3

During the informed consent process, the patient is informed of (1) his diagnosis; (2) purpose, description, benefits, and risks of the proposed treatment; (3) alternative treatment options; (4) prognosis of no treatment; and (5) costs. The patient asks questions and demonstrates that he understands all information presented during the discussion. Then the patient refuses any treatment. What, if anything, should the dental hygienist do?

Ethical Dilemma

Winnie RDH has been working as a dental hygienist for Dr. Mooney for the last year. It is her first job after graduation. She is very happy with her working arrangements, enjoys her coworkers and patients, and has been given increased office responsibilities, as well as a pay increase.

Winnie RDH has been instructed to review all the patients' chart entries—for all the clinicians in the office—at the end of each business day for accuracy and make corrections as needed. Dr. Mooney has authorized Winnie RDH, to write and sign all of his patient notes, to maximize his time with patient treatments.

Dr. Mooney has also instructed each of the four other hygienists in his employ, as well as Winnie RDH, to bill each cleaning as "quadrant periodontal instrumentation" as opposed to a prophylaxis, so every patient will receive the ultimate dental hygiene experience. He also requires that all patients receive localized delivery of antimicrobial agents, and that the respective hygienists enter the proper insurance code for that service.

Dr. Mooney sent Winnie RDH, to a practice management seminar, to help improve the management of the office. At the course, Winnie RDH learned that many of the above office practices were unethical. Winnie RDH wants to continue working for Dr. Mooney but is concerned about her liability.

1. Discuss Winnie's potential liability with Dr. Mooney's current office practices and documentation of the care provided by the hygienists in the office.
2. Are there ethical principles in conflict in this dilemma?

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CHAPTER 33

Future Directions for Management of Periodontal Patients

- Section 1 Contemporary and Evolving Diagnostic Technology
  - Computer-Linked Periodontal Probes
  - Ultrasonic Periodontal Probes
  - Digital Radiographs
  - Computed Tomographic Radiography
  - Other Technologies of Interest in the Future
- Section 2 Periodontal Disease/Systemic Disease Connections
  - Systemic Diseases with a Link to Periodontal Disease
  - Diabetes Mellitus in Periodontal Patients
- Section 3 Research in Dental Implantology
  - State of Dental Implantology
  - Research Questions Related to Maintenance of Dental Implants
- Section 4 Treatment Modalities in Periodontal Care
  - Lasers in Periodontal Care
  - Genetic Technology in Periodontal Care
  - Local Delivery Mechanisms in Periodontal Care
  - Host Modulation Therapies in Periodontal Care
  - Disease Risk Assessment in Periodontal Care
  - Advances Based on Stem Cell Biology

CLINICAL APPLICATION

While there are not many absolutes in periodontics, there is undoubtedly one: the simple fact that strategies for management of patients with periodontal diseases will continue to evolve and change over time. Thus, dental hygienists must be prepared to accept and readily implement evolving strategies to treat and manage periodontal diseases. This chapter explores a few relevant topics that point to the direction of ongoing and future dental research.

LEARNING OBJECTIVE

- Describe some strategies in the management of patients with periodontal diseases that are likely to evolve in the future.

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KEY TERMS

Computer-linked periodontal probe
Ultrasonic periodontal probe
Computed tomography
Medical lasers
Genetic testing
Stem cells
Undifferentiated (cells)
Dental stem cells

Section 1

Contemporary and Evolving Diagnostic Technology

1. **Computer-Linked Periodontal Probes**
    a. **Traditional Manual Probes.** For decades, traditional clinical assessment methods have been used to diagnose and monitor the periodontal condition and health of all patients. Many of these traditional clinical assessment methods involve the use of manual periodontal probes to measure both probing depths and clinical attachment levels.
        1. An experienced clinician can record probing depths fairly rapidly, and in many patients probing depths provide a reasonable assessment of periodontal health.
        2. Contrastingly, clinical attachment levels provide a more accurate assessment of the precise condition of the periodontium. However, clinical attachment levels are difficult to measure and record using manual periodontal probes.
        3. Accuracy with a traditional manual probe relies on skilled technique. With manual probing, accurate measurements are dependent on probe insertion force, probe tip position, and probe tip design.[1] Technical advances such as computer-linked and ultrasonic probes deliver pressure consistency and automate recording and documentation.
    b. **Computer-Linked Probing Systems.** **Computer-linked periodontal probes** use an optical handpiece that electronically reads and records measurements.[2–4]
        1. Automated probes use a constant gram force pressure of 15 g. Automated constant force probes provide objective measurements of pocket depth and attachment loss (**Figure 33-1**).
        2. As with manual probing, automated probing systems require clinician skill and experience to effectively use these systems.
        3. A systemic review of the literature looked at the reproducibility of manual probes versus automated probes in subjects with untreated periodontitis. Researchers found that both probes demonstrated similar reliability when used by a calibrated clinician.[5,6]
    c. **Ultrasonic Periodontal Probes.** **Ultrasonic periodontal probes** are also known as ultrasonographic periodontal probes or ultrasound periodontal probes. Conventional manual periodontal probes and computer-linked periodontal probes have the inherent disadvantages of causing unintentional patient discomfort and introducing a degree of variability in probing measurements. To resolve these issues, in the late 1990s, Hinders and colleagues introduced a "painless way to monitor gum disease" that does not involve probe tip insertion into the gingival sulcus.[7] Applying the same technology that NASA uses to inspect the structural integrity of rockets and that obstetricians use to monitor a developing fetus, ultrasonographic technology uses ultrasound to delineate objects or areas of different density within the body. Adapting this technology to periodontal applications, Hinders et al. proposed incorporating ultrasonic technology within a small intraoral handpiece as a noninvasive means to assess periodontal health. **Figure 33-2** shows an illustration of an ultrasonic periodontal probe.
        1. Compared to conventional periodontal probing, an ultrasonic periodontal probe promises reduced intra- and inter-examiner variability in probing measurements, improved ease of use, and enhanced patient comfort because the tip is not inserted in the sulcus.

2. The ultrasonic probe is a noninvasive ultrasound technique to detect, image, and map the upper boundary of the periodontal ligament (**Figure 33-3**).[8] Moreover, unlike conventional probing, the ultrasonic probe tip is placed at the gingival margin without entering the gingival sulcus, thereby eliminating the risk of added patient discomfort.
3. Like the transducer embedded within a magnetostrictive ultrasonic scaler, a transducer is embedded within the handpiece of an ultrasonic periodontal probe. When activated, this transducer emits a beam of ultrasound waves that exits through a hollow tip as a focused, narrow beam which then enters the gingival sulcus/pocket. (Note that the tip of the ultrasonic periodontal probe is not inserted into the sulcus. Rather, the tip is placed at the level of the gingival margin, then moved along the margin from line angle to line angle.) Once the beams enter the periodontal tissues, they are either absorbed, scattered, or reflected. The beams that are reflected echo back to the embedded transducer, which then "reads" the data to create a computerized two-dimensional graphical output. The clinician then moves the tip of the ultrasonic probe along the gingival margin, which corresponds to the measurements obtained by "walking the sulcus" with a conventional manual periodontal probe.
4. As the probe tip passes along the gingival margin, the ultrasonic probe "maps out" the depth measurements and tissue contour across the entire subgingival area. As such, more information is obtained as opposed to just collecting readings at six individual sites.
5. Advantages of the ultrasonic periodontal probe are that it is a more noninvasive means to monitoring periodontal health compared to conventional methods. Further, ultrasonic probing allows for more accurate and reliable assessments of the periodontal tissues, enhanced patient comfort, and computerized storage of data.
6. Disadvantages are that it requires operator training to apply this type of probe in a safe and effective manner and requires that the operator be able to interpret the results obtained. A second disadvantage is that this technology is substantially more expensive than a conventional manual probe.

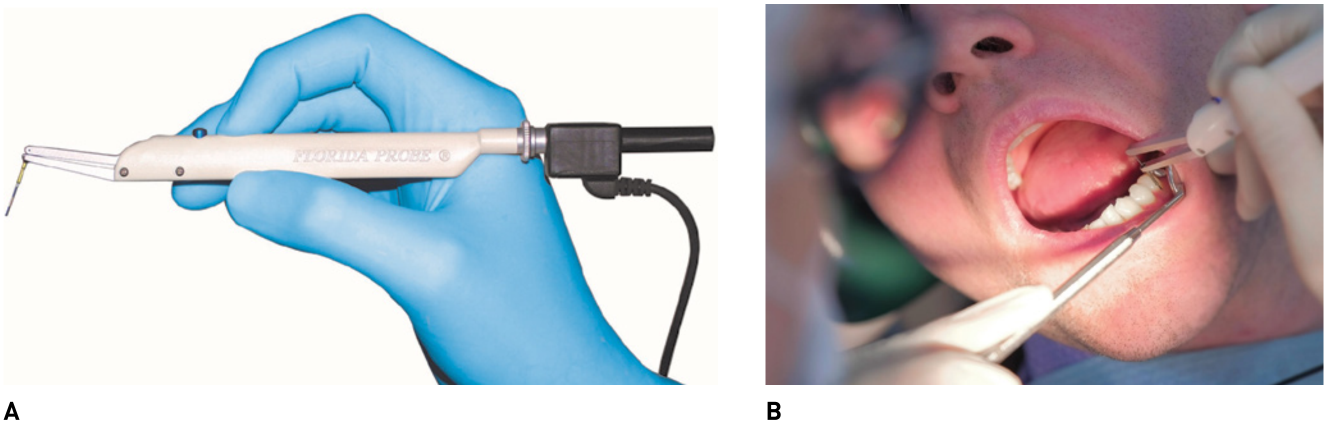

Figure 33-1 Computer-Linked Automated Probe. This is constant force probe that has been preset at 15 g of pressure. This constant force allows for objective and reproducible measurements of pocket depths, thereby reducing intra- and inter-examiner variability. Once the probe is in the sulcus, the practitioner taps on a foot pedal for the computer (attached to the computer-linked automated probe) to capture and register the measurements.

A: Courtesy of Florida Probe Corporation; **B:** © Vadim Zakharishchev/Shutterstock

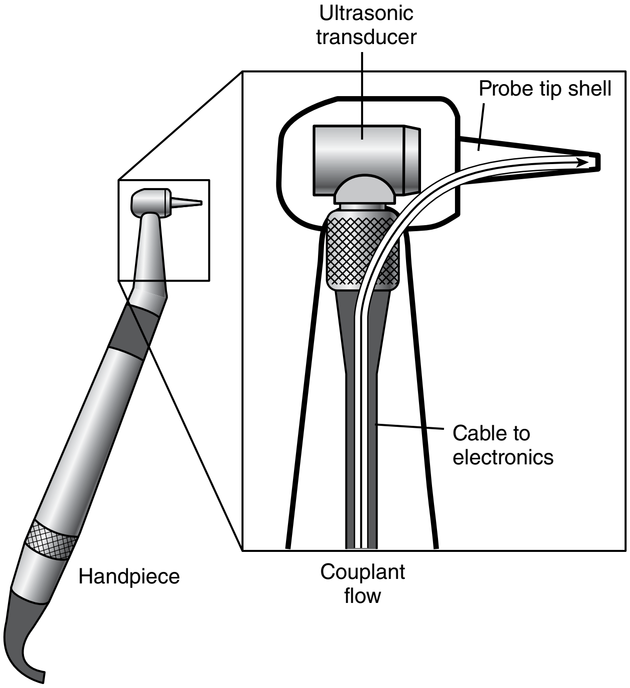
Ultrasonic transducer
Probe tip shell
Cable to electronics
Handpiece
Couplant flow

Figure 33-2 Illustration of an Ultrasonic Periodontal Probe. From its outward appearance, the ultrasonic periodontal probe does not look like a conventional periodontal manual probe. Rather, it appears similar to a conventional lightweight high-speed handpiece that a dentist uses to create a cavity preparation. However, unlike both the conventional periodontal probe and the conventional high-speed handpiece, the ultrasonic periodontal probe has an embedded transducer that converts electrical energy to electromagnetic waves. These electromagnetic waves are then emitted as a narrow, focused beam through the thin hollow tip. From there, the narrow, focused beam of electromagnetic pulses travels down into the periodontal ligament space. Once it hits a boundary between tissues (i.e., fluid and soft tissue), some of these pulses reflect back to the transducer, which is recorded by the transducer, and then relayed to a computer program that processes the information.

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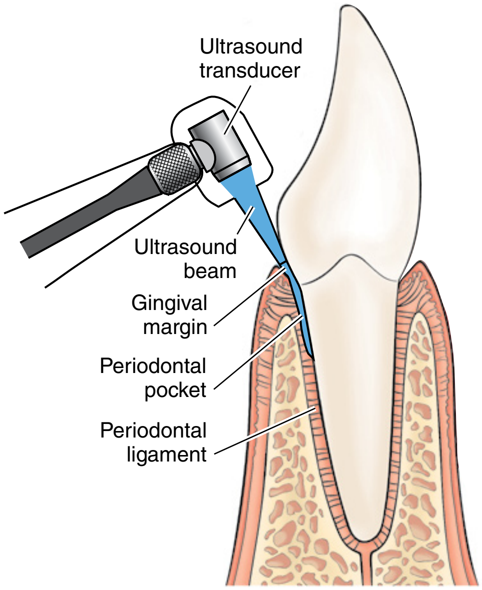
Ultrasound transducer
Ultrasound beam
Gingival margin
Periodontal pocket
Periodontal ligament

Figure 33-3 Ultrasonic Periodontal Probe. Illustration of an ultrasonic periodontal probe. The ultrasound transducer embedded within the probe emits a narrow beam of high-frequency ultrasound that is focused down the sulcus and enters the PDL space.

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2. **Digital Radiographs**
   a. **Film Radiography.** Although conventional (film) radiography is still widely used today, advances in diagnostic imaging technology are transforming the way that we take and interpret radiographic images. Digital radiography is a new type of diagnostic imaging technology that provides an enhanced image quality with better spatial resolution compared to conventional radiographs. Furthermore, unlike conventional radiographic techniques, digital radiography images are instantly available for distribution without the time and physical effort needed to either archive or retrieve film packets.[2,9–11]
   b. **Digital Radiography.** Digital (filmless) radiographic techniques have developed to the stage where they are now being used by most clinicians.
      1. Digital radiographic techniques allow members of the dental team to collect radiographic information using special sensors instead of printing the radiographic image on a film.
      2. These digital images are then stored on a computer that can be viewed on a computer screen or even printed when needed.
      3. Modern technology for viewing these images on computer screens has substantial advantages over the traditional use of radiographic film.
      4. Software for viewing these digitized images can eliminate distortion that is seen with traditional film; can allow for easy magnification of details; and can provide precise, anatomically correct measurements.
      5. The same software can also allow for enhancing aspects of a digitized image, providing members of the dental team with more details of the actual status of a tooth or of the periodontium.
      6. In addition, these digital images can be shared with other health care providers quite readily—as might be indicated during a patient referral or during consultation with a specialist.
3. **Computed Tomographic Radiography.** Another evolutionary diagnostic imaging modality is computed tomographic techniques.
   a. **Computed tomography** is a radiologic procedure using a machine called a scanner to examine a body site by taking a series of cross-sectional images one slice at a time in a full circle rotation (**Figure 33-4**).

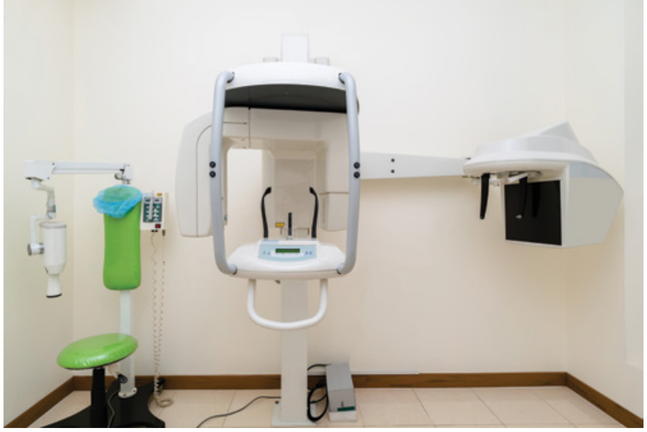

Figure 33-4 Computed Tomography. Computed tomography is a radiologic procedure using a machine called a scanner to examine a body site by taking a series of cross-sectional images one slice at a time in a full circle rotation. A computer then calculates and converts rates of absorption and density of the X-rays into a three-dimensional image (3-D) that can be viewed by the dental clinician.

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      1. Computed tomographic techniques provide clinicians with the ability to study minute details and precise dimensions of the jaws in a 3-D perspective on a computer screen.
      2. These details can be so precise that they can include a 3-D radiographic image of a thin slice made through the jaws at any specific location. For this reason, computed tomographic techniques are currently in use by many clinicians when planning for the placement of dental implants.

   b. Presently, the routine use of 3-D imaging to diagnose and treat periodontal disease is not warranted from a radiation exposure and cost perspective. However, in the future, if technological advancements in 3-D imaging are able to minimize radiation exposure and cost, 3-D imaging may become more widely accepted for routine use.[12]

4. **Other Technologies of Interest in the Future.** A variety of other technologies related to the diagnosis and treatment of patients with periodontal disease may indeed become more important as the results of further research become available. Examples of these other technologies include the following:
   a. Improved means of identification of the microbes populating periodontal pockets.
   b. Enhanced testing for genetic predispositions for developing periodontal diseases.
   c. Improved mechanisms for identifying the content of the gingival crevicular fluid as a means of understanding underlying periodontal disease processes, such as infrared spectral analysis.
   d. Development of techniques for using infrared spectroscopy to obtain diagnostic profiles of periodontal diseases.
   e. Increased utilization of noninvasive ultrasound imaging in the assessment of the status of the periodontium and other oral tissues.[13]

Section 2

Periodontal Disease/Systemic Disease Connections

Dental and medical researchers have been studying the connection between periodontitis and certain systemic diseases for some time. Research into this critical topic is ongoing and continues to offer insights into these connections.[14–22] Review of the dental literature will reveal many studies about how systemic conditions may be linked to periodontal diseases, but all of these areas will require further scientific study. For review, the complex bidirectional relationship between periodontal inflammation and systemic health is discussed in Chapters 14 and 15.

1. **Systemic Diseases with a Link to Periodontal Disease**
   a. Some examples of systemic diseases or conditions that may have a connection to periodontitis are atherosclerotic cardiovascular disease, adverse pregnancy outcomes, and diabetes mellitus.
   b. Here, we discuss the relationships between one specific condition—diabetes mellitus—and periodontal disease.
2. **Diabetes Mellitus in Periodontal Patients**
   a. **Need for Additional Research.** Our current understanding into the connection between diabetes and periodontal disease is still limited. Nevertheless, ongoing research contributions in this field will indeed impact the practice of dentistry.
      1. Research has demonstrated that patients with poorly controlled diabetes have an increased risk for periodontitis.[23–28]
      2. Because periodontitis is a type of infection, and because diabetes can lower the body's resistance to infections in general, it is not surprising that there is a connection between poorly controlled diabetes and periodontitis in some susceptible patients.
      3. In addition, research suggests that periodontal infection and the elimination of the periodontal infection through proper periodontal therapy have the potential to alter the body's control of blood sugar levels.
      4. It has even been suggested that thorough treatment of periodontitis in a diabetic patient may potentially lower the HbA1c levels and make it easier for a patient to manage his or her diabetic condition.[29]
   b. **Research Questions.** Many research questions need to be answered regarding the periodontitis–diabetes connection, but a few of those questions that can have a direct impact on the practice of dentistry are outlined below.
      1. Are the measures used to prevent or control periodontitis in the patient without diabetes mellitus adequate for the patient with diabetes mellitus?

    2. Since wound healing is altered in patients with diabetes, are there adjustments clinicians need to make when delivering dental hygiene therapy to maximize the potential for healing in these patients?
    3. What precise periodontal maintenance protocols are the most effective for patients with diabetes?
    4. When a dental clinician treats a patient with diabetes, what communication protocols can be most effective in ensuring that the patient's physician is aware of the patient's periodontal status so that adjustments in the therapy for diabetes can be made where needed?
  c. **Use of Intensive Therapies for Diabetes Mellitus.** In examining the periodontitis–diabetes connection, there is another line of inquiry that will also affect dental practice—the type of medical therapy used in diabetic patients.[29]
    1. In medicine, there have been dramatic improvements in the treatment regimens for patients with diabetes, and these regimens now frequently include intensive treatment with oral agents and with insulin.
    2. Unfortunately, some of these medical treatments have increased the risk for medical emergencies (such as hypoglycemia) during dental office treatment of a patient with diabetes.
    3. This medical trend in intensive therapies for patients with diabetes will continue.
    4. As physicians use more intensive therapies to manage patients with diabetes, all members of the dental team will need to have more knowledge about these therapies, about how to manage these patients in a dental setting, and about how to respond when a medical emergency arises.

Section 3

Research in Dental Implantology

1. **State of Dental Implantology.** In modern dentistry, dental implants are a viable option as one alternative for replacing most missing teeth. It should be noted that dental implants available today have a high success rate. Even though dental implantology has been intensively studied for several decades, there are still many unanswered questions related to this field, and research will continue. Because implants are susceptible to breakdown in the presence of inflammation, patient selection and the proper maintenance of the peri-implant tissues in a state of health are of paramount importance. The reader can refer to Chapter 8 to review the many widely accepted and evidence-based treatment protocols available to maintain dental implants.
2. **Research Questions Related to Maintenance of Dental Implants.** Much additional investigation is needed in dental implantology, and some examples of questions related to dental hygiene that are in need of further study are listed below. Answering these types of questions with appropriate scientific investigation is quite likely to have a substantial impact on clinical care delivered by the dental hygienist.
   a. What self-care measures can best prevent peri-implant infections?
   b. What are the most effective protocols for effective maintenance of implants?
   c. Should clinicians recommend the same techniques for minimizing the bacterial challenge to an implant that apply to a natural tooth?
   d. When treating dental implants patients, what types of instruments provide the greatest chance of maintaining periodontal health?

Section 4

Treatment Modalities in Periodontal Care

Treatment modalities for patients with periodontal diseases are constantly evolving. This section outlines some treatment modalities that can be expected to enhance more effective periodontal therapy as they evolve further.

1. **Lasers in Periodontal Care**
   a. Lasers have been widely used in many fields of medicine since the early 1960s. **Medical lasers** are medical devices that use precisely focused light sources to treat or remove tissues. Lasers produce a narrow beam of light with a single wavelength that can produce intense energy at precise locations.

    1. In dentistry, these intense light beams are passed down a narrow optical tubing and can be focused on a small area of tissue within the mouth and within the periodontium.
    2. Some laser beams are so intense that they can actually be used to remove oral soft tissue or to cut tissues in the mouth.
    3. Different types of lasers have been studied for use in dentistry, and each type has a somewhat different effect on soft tissue, enamel, dentin, pulp, and bone.
  - b. Lasers have been suggested for use in dentistry for a variety of dental applications. Some of these devices even have Food and Drug Administration (FDA) safety clearance for some intraoral soft tissue procedures.
    1. More study is needed, however, to clarify how these devices can be used appropriately in subgingival applications in patients with periodontal diseases, and some of these studies are in progress.[30,31]
    2. Some investigations have suggested a possible combined use of lasers as with conventional manual instruments to debride periodontally diseased root surfaces (**Figure 33-5**).
    3. Additional research will clarify appropriate uses for these devices in patients with periodontal diseases and may impact some of the therapy provided by dental hygienists for patients with periodontitis and for those with peri-implantitis.
    4. If further study of these devices confirms that patients with periodontal diseases do benefit from their use, lasers may one day be a routine part of the care of patients with periodontitis, peri-implantitis,[32] and perhaps even a part of the practice of dental hygiene.

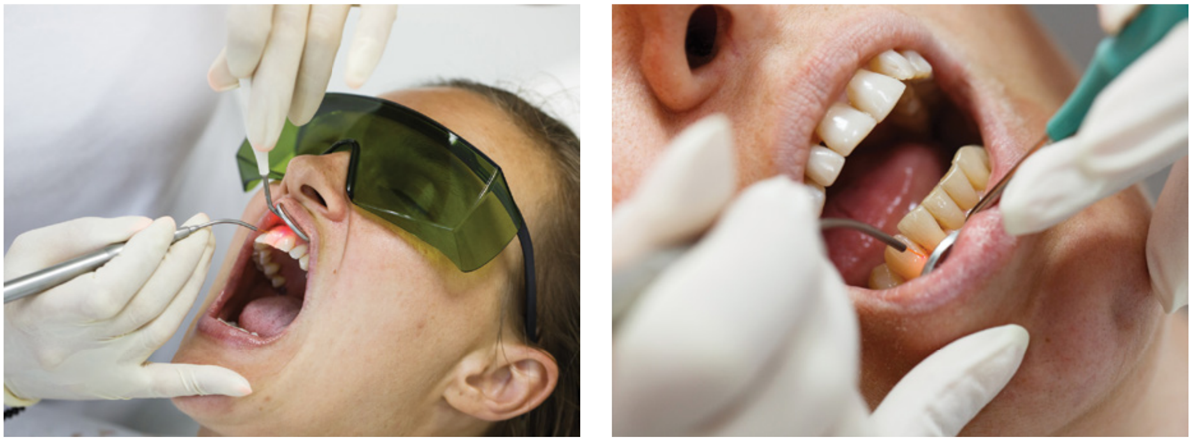

Figure 33-5 **Laser Being Used in Nonsurgical Periodontal Therapy.** A clinician using a modern diode dental laser for periodontal care. Patient is wearing protective glasses, preventing eyesight damage. Lasers may prove to be an effective instrument to have in the periodontal armamentarium because they have the potential to improve on efficiency and comfort for the patient.

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  - c. Currently, however, an American Academy of Periodontology statement on the efficiency of lasers in the nonsurgical treatment of inflammatory periodontal disease states that there is minimal evidence to support use of a laser for the purpose of subgingival periodontal instrumentation.[33] Additionally, for the treatment and management of peri-implantitis, a recent systemic review and meta-analysis concluded that laser therapy only produced identical reductions in probing depths, amount of radiographic bone fill, and clinical attachment level gains compared to other commonly used surface detoxification methods.[34]

2. **Genetic Technology in Periodontal Care.** **Genetic testing** is a type of medical test that identifies changes in chromosomes, genes, or proteins. The results of a genetic test can confirm or rule out a suspected genetic condition or help determine a person's chance of developing or passing on a genetic disorder.
  - a. Clinicians have known for a long time that there are many factors that can increase the risk of developing periodontitis.
    1. One factor that is known to increase the risk of developing periodontitis is failure to control bacterial plaque growth on the teeth, thereby increasing the bacterial challenge to the periodontium.

2. Scientific studies also indicate that certain genetic factors determine how an individual patient's host defenses actually react to an increased bacterial challenge.
3. Based upon the current research literature available, it now appears that a key factor in determining whether a patient develops periodontitis in response to the bacterial challenge is how the body reacts to that bacterial challenge.
4. One major determinant of how the body reacts to the bacterial challenge is genetics (or inherited characteristics).

b. Much more study into the genetic factors that increase the risk for periodontitis is needed, but it is already possible to use some types of genetic information to guide clinical decision-making in a small group of selected patients.

1. Genetic testing can identify patients carrying gene mutations for several rare syndromes that are often accompanied by a form of periodontal disease (**Figure 33-6**).

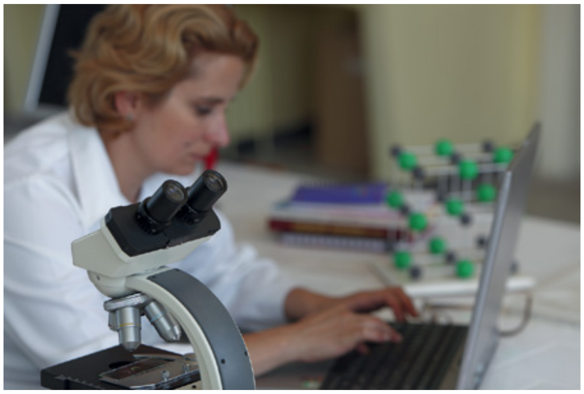

Figure 33-6 **Genetic Testing.** Scientific studies also indicate that certain genetic factors determine how an individual patient's host defenses actually react to an increased bacterial challenge. Much more study into the genetic factors that increase the risk for periodontitis is needed, but it is already possible to use some types of genetic information to guide clinical decision-making in a small group of selected patients.

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2. In addition to identifying patients with rare syndromes, a commercially available genetic susceptibility test is available for more severe forms of periodontitis.
3. In this test, specific gene polymorphisms (forms) that have been associated with the development of periodontitis can be detected.
4. Ongoing scientific investigations will undoubtedly clarify how such genetic testing can be used in periodontitis patient management.

c. As more and more scientific information about identifying genetic control of host defenses becomes available, it is quite likely that this information will impact how we manage patients with periodontal diseases and will impact the practice of dental hygiene.

3. **Local Delivery Mechanisms in Periodontal Care**

a. Research has demonstrated that using local delivery mechanisms for antimicrobial chemicals in patients with periodontitis has a small but measurable impact upon clinical parameters, such as attachment levels.[35] Reported analyses of the long-term effects of chemotherapeutic agents usually do not extend beyond a few months to a year. Nonsurgical periodontal instrumentation remains the gold standard for the treatment of inflammatory periodontitis.[31]

- b. Several areas of research investigation are needed related to these local delivery mechanisms, and some examples of research questions about this topic that need to be answered are listed below.
    1. Can future local delivery mechanisms be designed that have a greater clinical impact than those currently available for clinical use?
    2. What specific local delivery treatment protocols should be followed to produce the most benefit for individual patients?
    3. Are there additional antimicrobial agents that can be delivered safely using the local delivery concept?
    4. Can other therapy provided by the dental hygienist be enhanced by using some of these local delivery mechanisms?
- c. Research into the modification of polymers, manufacturing technologies, and carrier systems will undoubtedly lead to vastly improved drug delivery systems that may have an impact on periodontal therapy strategies.
- d. Research into the use of local delivery mechanisms for antimicrobial agents continues. It is probable that as this modality improves in clinical effectiveness, using local delivery mechanisms may become more and more useful in the care of the periodontal patient by the dental hygienist.

4. **Host Modulation Therapies in Periodontal Care**
    - a. The host immunoinflammatory response has a dual role in the body. On the one hand, it plays a protective role in defending the host from pathogens, maintaining homeostasis, and preserving the structural integrity of tissues and organs. On the other hand, in certain susceptible individuals, the extent and effects of the immunoinflammatory response can overcome the host's ability to recover from the harmful effects of inflammation. This is seen in periodontitis, where in certain susceptible individuals, it is their *immunoinflammatory response* that destroys the attachment apparatus, not the dental biofilm per se. To mitigate the host immunoinflammatory response, several host modulating agents are currently being investigated as potential medications to manage periodontitis. Chapter 29 provides a general overview of the recent advances made in host modulators.
        1. A variety of host modulation therapies have been investigated that could be used as adjunctive (supplemental) treatment in patients with periodontitis.[36–38]
        2. Host modulation therapies usually involve using medications that can alter biochemical pathways in a manner that will (1) slow attachment loss, (2) slow alveolar bone loss, or (3) decrease inflammation.
    - b. Investigations into possible host modulation therapies have already resulted in one commercially available medication (subantimicrobial dose doxycycline hyclate) that can be used as adjunctive treatment in patients with chronic periodontitis.
        1. This medication can be used to inhibit the actions of the enzyme collagenase (MMP-8), an enzyme involved in the destruction of collagen.
        2. Collagen is one of the components of many of the structures that make up the periodontium. Thus, lowering the activity of collagenase can slow the progress of periodontitis.
        3. Investigations are ongoing into a number of other possible host modulation therapies that include studies into (1) modulation of cytokines (chemicals involved in periodontitis that can result in increased periodontal disease progression), (2) reduction of prostaglandins (chemicals that enhance inflammation in the gingiva and in the periodontium), and (3) slowing alveolar bone loss with chemical agents.
    - c. In addition, an interesting future direction for research will include local delivery systems that can deliver varied concentrations of host modulating agents over a sustained period of time. Variable concentrations could be used to achieve the maximum therapeutic effects when planning individualized nonsurgical treatment for patients with periodontal disease.
    - d. As further scientific investigations improve our understanding of host modulation therapies, there are likely to be a variety of new therapeutic options for members of the dental team to use in patient management.
5. **Disease Risk Assessment in Periodontal Care**
    - a. Recently, there has been increased interest in identifying clinical tools that can be used to quantify a patient's risk for developing periodontitis.[39–41]

1. Traditionally, clinicians have assessed the risk of developing periodontitis subjectively, but studies have shown that subjective risk assessment is surprisingly variable even among clinicians who are experts.[36]
2. Objective periodontal disease risk assessment tools would be quite useful to members of the dental team if they provided a method of risk assessment that could accurately predict which patients are most likely to develop periodontitis.
3. Examples of risk factors that have been suggested to be predictive of periodontal disease activity are listed in **Box 33-1**.

Box 33-1 Examples of Factors That May Help Predict Periodontal Disease Activity

- Smoking
- Poorly controlled diabetes
- Poor patient self-care
- Severity of alveolar bone loss
- Positive family history
- Presence of pocket depths >6 mm
- Age
- Gender
- Gingival bleeding/bleeding on probing
- Number of missing teeth
- Specific periodontal pathogens

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4. Using risk assessment tools to identify the patients with the highest risk for developing periodontitis would allow members of the dental team to provide more aggressive treatment for those patients.
5. In addition, these tools might identify which periodontal patients should be referred to a specialist early in their treatment and which patients can best be managed in a general dental setting.

b. Studies show that some of these risk assessment tools are reasonable predictors of alveolar bone loss and loss of periodontally affected teeth.
   1. It is likely that some of these tools for quantifying a patient's risk will soon be in widespread use in dental offices.
   2. Guidelines from the American Academy of Periodontology indicate that periodontal disease risk assessment should be part of every comprehensive dental and periodontal evaluation.
   3. The American Academy of Periodontology has even developed a simplified form of risk assessment for use by patients. This web-based patient self-assessment can be viewed at www.perio.org.
   4. These risk assessment tools would be useful to the dental hygienist and the dentist in planning therapy and in identifying patients in need of immediate referral.

6. **Advances Based on Stem Cell Biology**
   a. **Introduction to Stem Cell Biology.** Stem cell biology is an emerging field of medical research that can have a profound effect on medical therapy available for certain systemic diseases and that may have utility in regeneration of periodontal tissues in the future.[42–47]
      1. **Stem cells** are the "master cells" of the human body that have the ability to develop into any one of the body's more than 200 cell types.
      2. Stem cells are unspecialized (**undifferentiated**) cells. When a stem cell divides, each new cell has the potential either to remain a stem cell or become another type of cell with a more specialized function, such as a muscle cell, a red blood cell, or a brain cell. **Figure 33-7** depicts the process of cellular differentiation.

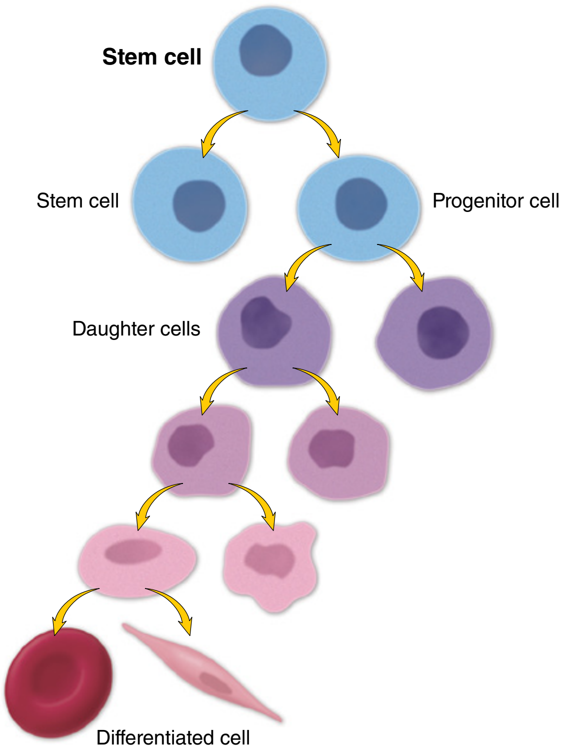

Figure 33-7 Cellular Differentiation. Cellular differentiation is the process by which cells become more specialized. Undifferentiated stem cells divide into like stem cells or parent (progenitor) cells, which continue to divide and further differentiate into highly specialized and functional cells.

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3. Stem cells retain the ability to divide throughout life and give rise to cells that can become highly specialized and take the place of cells that die or are lost. Stem cells contribute to the body's ability to renew and repair its tissues.

b. **Dental Stem Cell Research**
   1. Human stem cells have already been isolated from the periodontal ligament, dental pulp tissue, exfoliated deciduous teeth, dental papillae, and dental follicles.
   2. **Dental stem cells** apparently can differentiate into specific components of the periodontium (such as periodontal ligament and cementum).
   3. Given their unique regenerative abilities, stem cells offer new potentials for treating diseases such as diabetes, and to influence periodontal treatment strategies. However, much work remains to be done in the laboratory and the clinic to understand how to use these cells for cell-based therapies to treat disease, which is also referred to as regenerative or reparative medicine.

Chapter Summary Statement

All members of the dental team should anticipate many changes that will take place as advancements in research and innovation occurs. This chapter presented a brief overview of a few of the possibilities for future directions in the management of patients with periodontal diseases.

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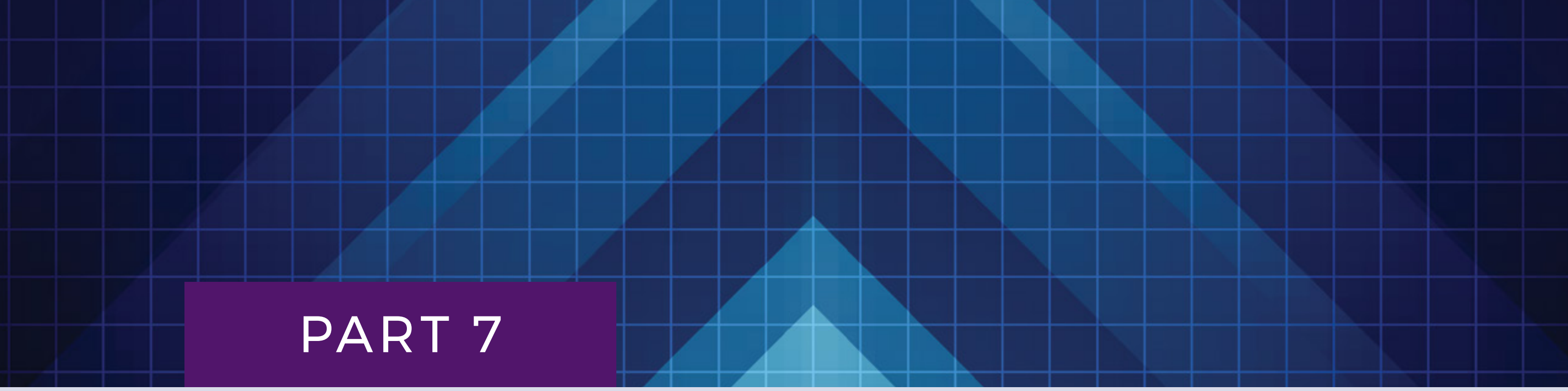

PART 7

Comprehensive Patient Cases

CHAPTER 34 Comprehensive Patient Cases 771

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CHAPTER 34

Comprehensive Patient Cases

- Fictitious Patient Case 1—Mr. Karn
- Fictitious Patient Case 2—Mr. Wilton
- Fictitious Patient Case 3—Ms. Sandsky
- Fictitious Patient Case 4—Mr. Verosky
- Fictitious Patient Case 5—Mr. Tomlinson

LEARNING OBJECTIVE

- Apply the content from the chapters in this book to answer the decision-making questions for the hypothetical case scenarios presented in this chapter.

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Fictitious Patient Case 1—Mr. Karn

Figure 34-1 Mr. Karn

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Patient Profile

Mr. Karn is a 47-year-old high school administrator who has recently moved to your city. He came to the dental office because he would like to know if it is possible to replace his missing upper right first molar tooth with a dental implant.

During Mr. Karn's first office visit, he informs you that he has been too busy lately to get a dental check-up and that he has not seen a dentist for quite a few years. Mr. Karn states that he brushes his teeth twice daily when he has time and that he does not floss regularly even though he knows that he should. He also uses an over-the-counter mouth rinse occasionally.

Patient Health History

- On the day of his first visit to your dental office, Mr. Karn's blood pressure is 130/80 mm Hg and his pulse is 62 beats/min.
- A review of Mr. Karn's health history reveals that he takes two medications: Zocor® (simvastatin) and Procardia® (nifedipine).
- Mr. Karn also states that he smokes between one half and one pack of cigarettes each day.
- Mr. Karn's dental history reveals that the maxillary right first molar was extracted over 6 years due to non-restorability reasons.

Clinical Photographs for Mr. Karn

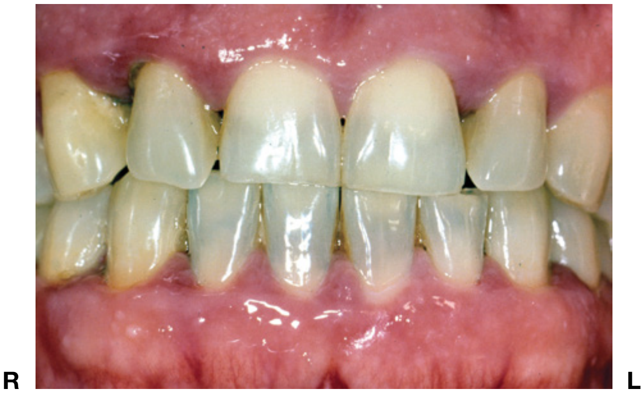

Figure 34-2A **Anterior Teeth, Facial View.**

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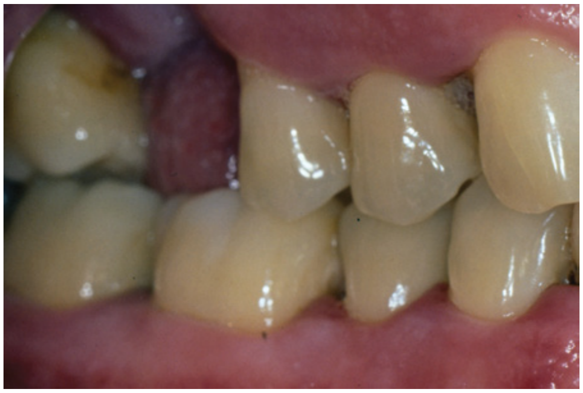

Figure 34-2B **Right Side, Facial View.**

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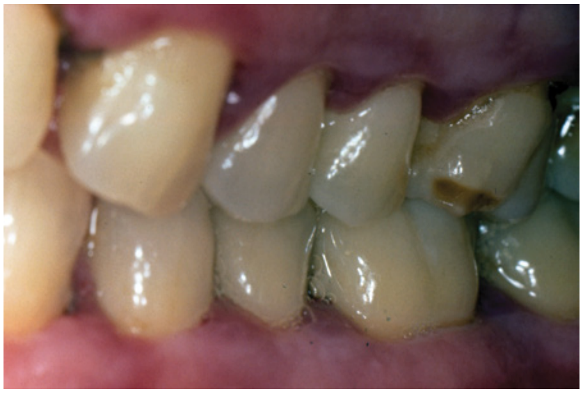

Figure 34-2C **Left Side, Facial View.**

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Clinical Photographs for Mr. Karn

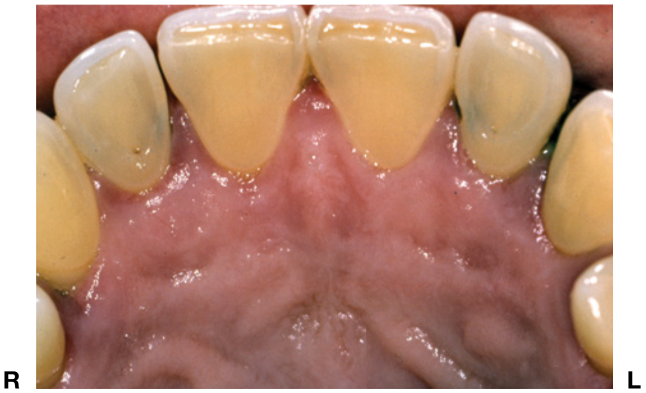

Figure 34-3A Maxillary Anterior, Lingual View.

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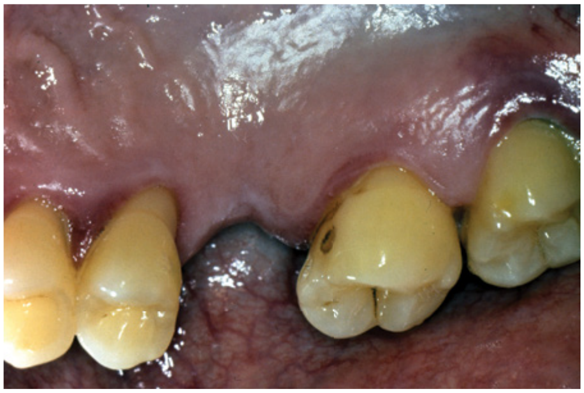

Figure 34-3B Maxillary Right, Lingual View.

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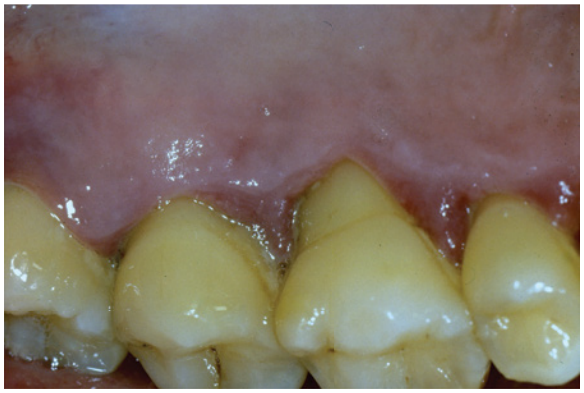

Figure 34-3C Maxillary Left, Lingual View.

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Clinical Photographs for Mr. Karn

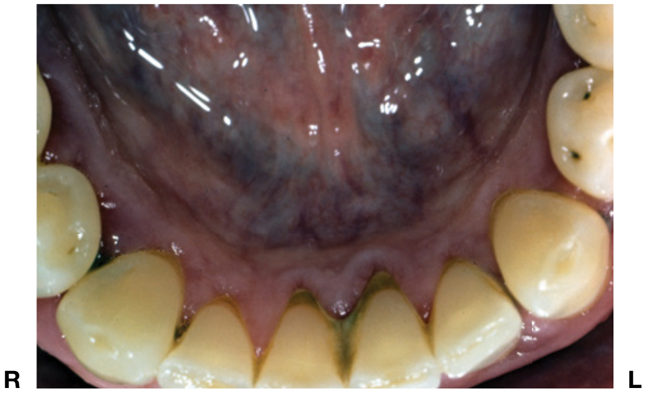

Figure 34-4A Mandibular Anterior, Lingual View.

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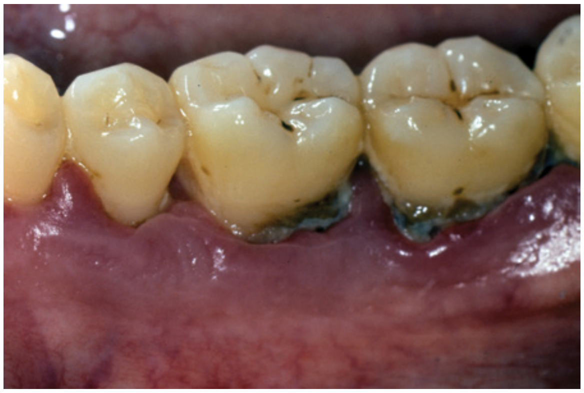

Figure 34-4B Mandibular Right, Lingual View.

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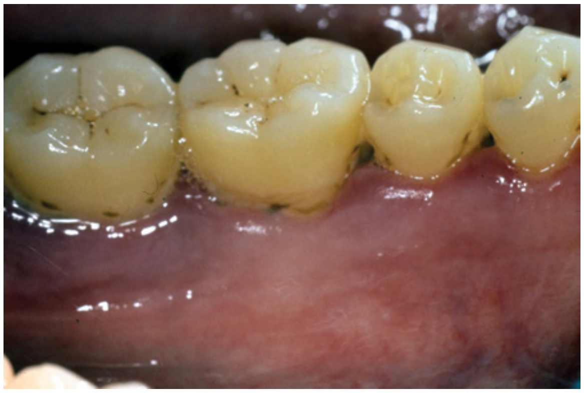

Figure 34-4C Mandibular Left, Lingual View.

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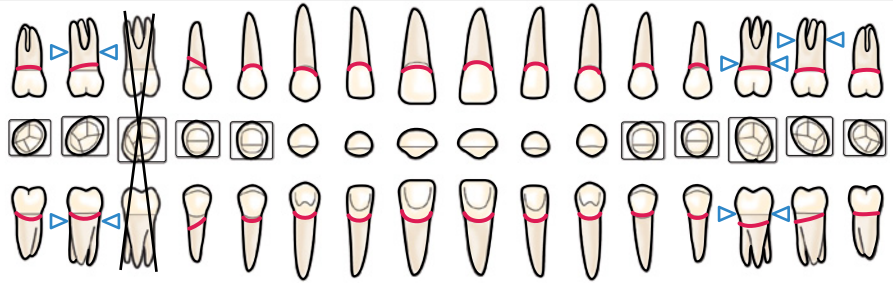
CASE #1

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	**Maxilla**
					I	I						I			I	Mobility (I, II, III)
+	+		+	+	+	+			+	+	+	+	+	+	+	Bleeding/Purulence (+)
646	647		535	536	415	322	322	334	425	435	536	626	638	846	746	Attachment Level (CEJ to BP)
646	635		325	536	525	435	433	334	425	435	536	626	638	846	746	Probing Depth (FGM to BP)



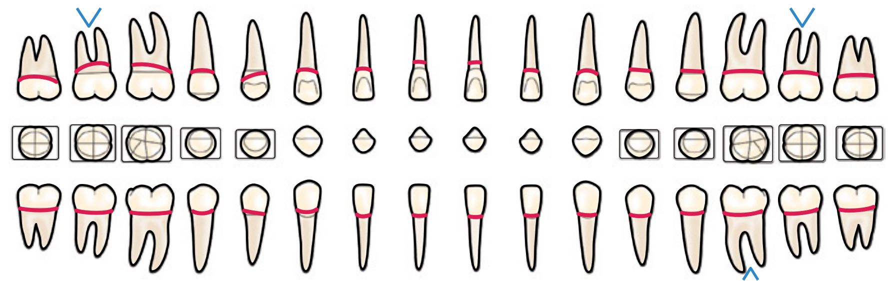
Facial

Palatal

+	+		+	+	+	+	+	+	+	+	+	+	+	+	+	Bleeding/Purulence (+)
646	646		546	526	536	425	443	324	424	525	535	626	859	937	736	Attachment Level (CEJ to BP)
636	525		335	526	536	425	443	324	424	525	535	626	627	827	736	Probing Depth (FGM to BP)
••••	•••		••	•••	•••	•••	•	••	•	•••	••	•••	••••	•••	•••	^F_PPlaque
	✓		✓	✓				✓	✓				✓	✓	✓	Supragingival Calculus
✓	✓		✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	Subgingival Calculus
4					3						4					PSR Code

Right *Left*

32	31	30	29	28	27	26	25	24	23	22	21	20	19	18	17	**Mandible**
			I				I	I								Mobility (I, II, III)
+		+	+	+	+	+		+		+	+	+		+	+	Bleeding/Purulence (+)
546	746	736	635	435	534	324	534	434	324	324	434	435	536	746	635	Attachment Level (CEJ to BP)
546	736	626	635	535	534	324	423	323	324	324	434	435	536	746	635	Probing Depth (FGM to BP)



Lingual

Facial

+	+	+	+	+		+	+	+		+		+		+	+	Bleeding/Purulence (+)
546	736	625	535	635	534	324	423	323	324	324	434	435	526	736	625	Attachment Level (CEJ to BP)
546	736	625	535	635	534	324	423	323	324	324	434	435	526	736	625	Probing Depth (FGM to BP)
••••	••••	••••	•••	•••	••••	•••	•••	•••	••••	••••	••	•••	••••	•••	•••	^L_FPlaque
					✓	✓	✓	✓	✓			✓	✓			Supragingival Calculus
✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	Subgingival Calculus
4					3						4					PSR Code

Figure 34-5 **Mr. Karn's Periodontal Chart.**

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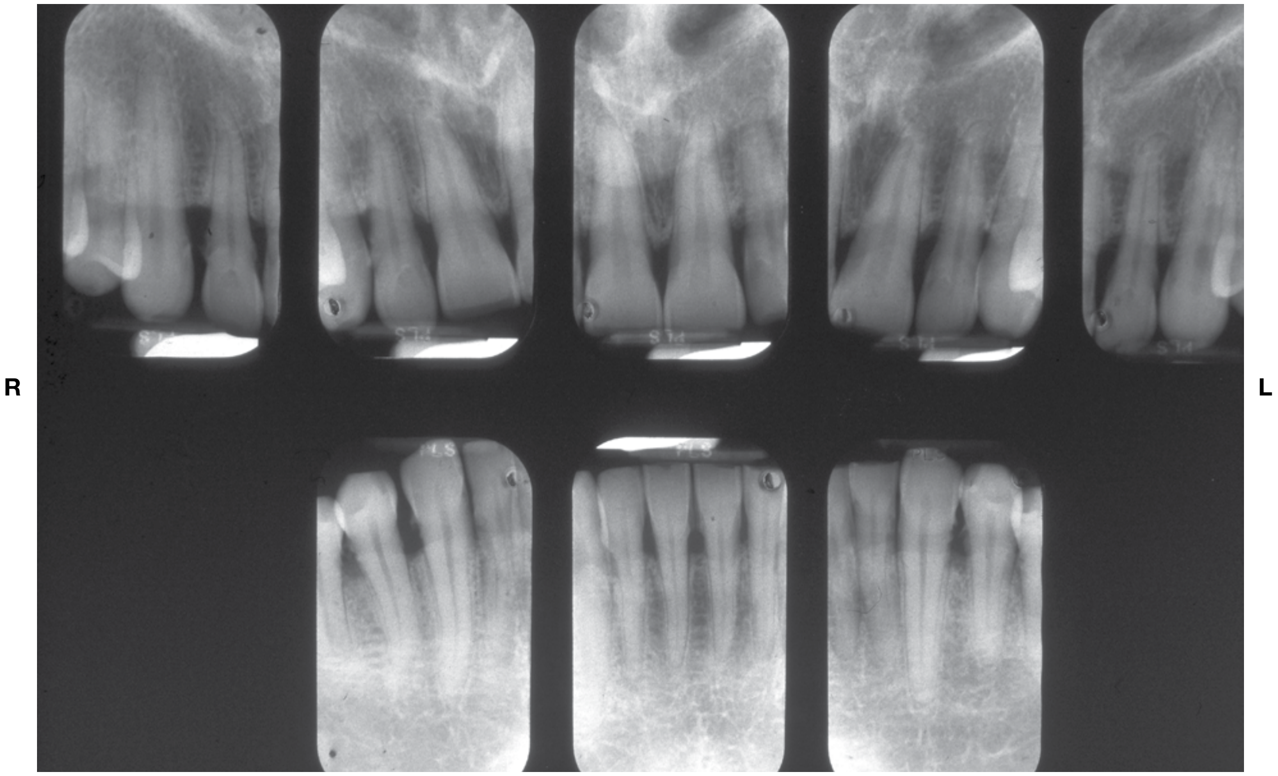

Figure 34-6A **Radiographs: Anterior Teeth.**

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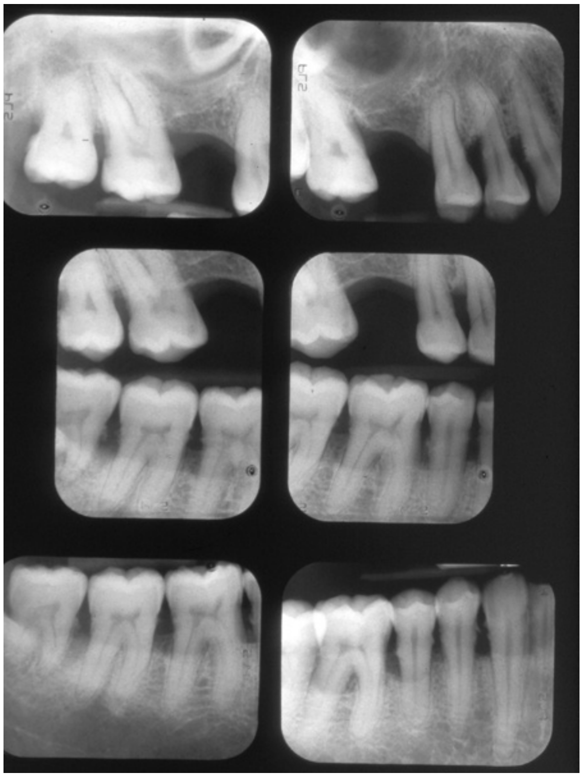
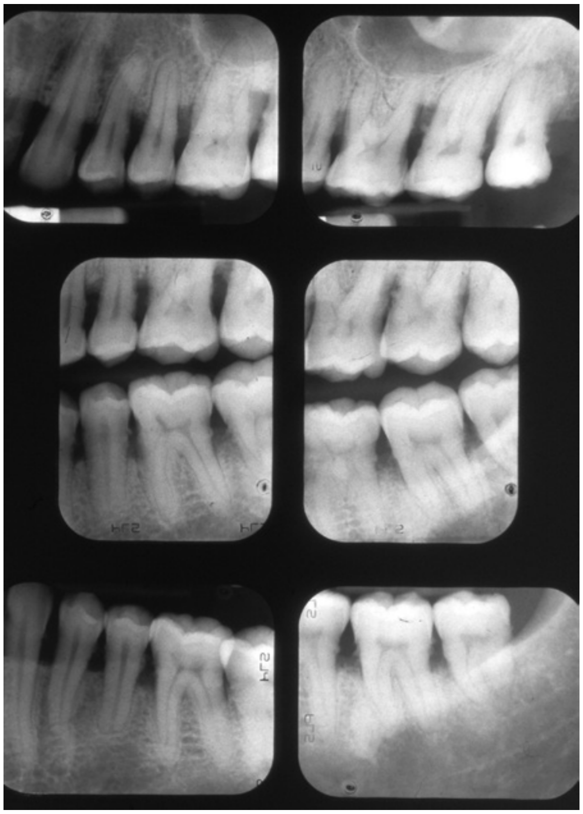


Figure 34-6B Radiographs: Right Posterior Teeth.

© Wolters Kluwer

Figure 34-6C Radiographs: Left Posterior Teeth.

© Wolters Kluwer



Decision-Making Questions for Case 1: Mr. Karn

1. What should Mr. Karn be told about the possibility of replacing his maxillary right first molar tooth with a dental implant? Note that this question was what prompted Mr. Karn to make an appointment in your dental office.
2. What factors in Mr. Karn's profile indicate that achieving an acceptable level of patient self-care may be a problem for the dental team?
3. What factors revealed in Mr. Karn's health history will be critical for the dental team to consider during treatment?
4. What signs of gingival inflammation are evident in Mr. Karn's clinical photographs?
5. What etiologic risk factors for periodontal diseases are evident in Mr. Karn's clinical photographs?
6. How might the presence of the furcation involvements found during Mr. Karn's periodontal evaluation affect his periodontal treatment?
7. Does Mr. Karn's periodontal evaluation indicate that he has attachment loss present on some teeth?
8. What etiologic factors for gingival and periodontal diseases are evident in Mr. Karn's dental radiographs?
9. What specific findings on Mr. Karn's radiographs indicate that alveolar bone loss is present?
10. How would you characterize Mr. Karn's periodontal condition? Do you think that he has gingivitis, periodontitis, neither, or both? What clinical or radiographic findings did you use to reach your conclusion?
11. Develop a step-by-step nonsurgical treatment plan for Mr. Karn.
12. What information should your team give Mr. Karn about his periodontal condition?
13. What should Mr. Karn be told about the possible need for periodontal surgery later in the treatment?
14. What should Mr. Karn be told about the need for continuing treatment such as periodontal maintenance?

Fictitious Patient Case 2—Mr. Wilton

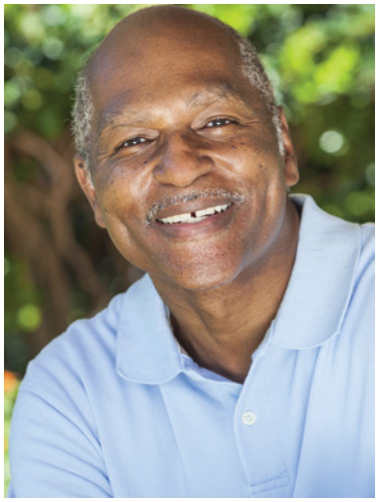


Figure 34-7 Mr. Wilton

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Patient Profile

Mr. Wilton is a 52-year-old manager of a local gardening store who has come to your dental office for an initial visit. During his patient interview, Mr. Wilton informs you that he made this appointment at his wife's insistence. He states that his wife wants to know if there is anything that can be done about his bad breath. Mr. Wilton informs you that he cannot seem to get his bad breath under control using mouth rinses.

Patient Health History

- At the time of his initial visit, Mr. Wilton's blood pressure is 164/100 mm Hg and his pulse rate is 74 beats/minute.
- Mr. Wilton informs you that he is taking amoxicillin prescribed by his physician for an ear infection.
- Mr. Wilton tells you that he had high blood pressure once and that he did take a prescribed medication a few years ago for that condition. He tells you that he was feeling just fine so he stopped taking the prescribed blood pressure medication.
- Mr. Wilton has a history of extractions due to periodontitis reasons.

Clinical Photographs for Mr. Wilton

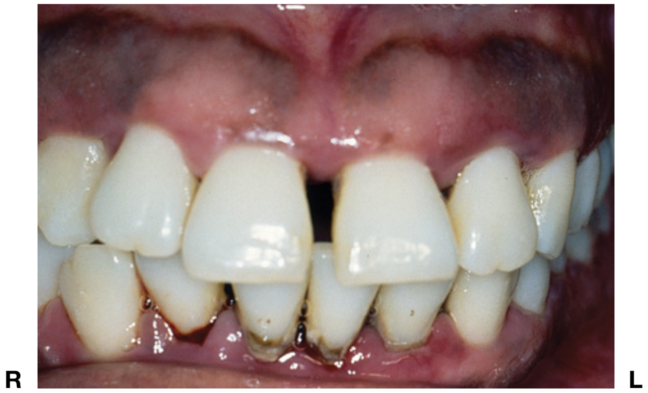

Figure 34-8A **Anterior Teeth, Facial View.**

© Wolters Kluwer

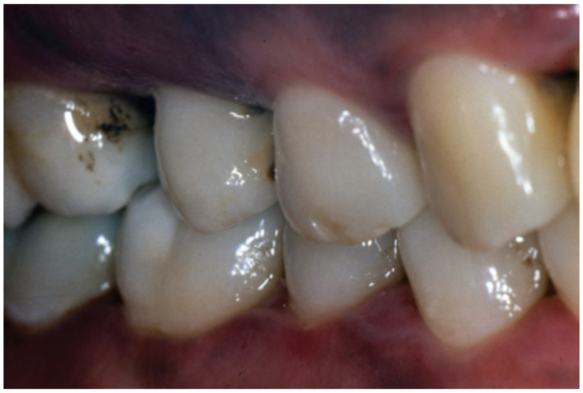

Figure 34-8B **Right Side, Facial View.**

© Wolters Kluwer

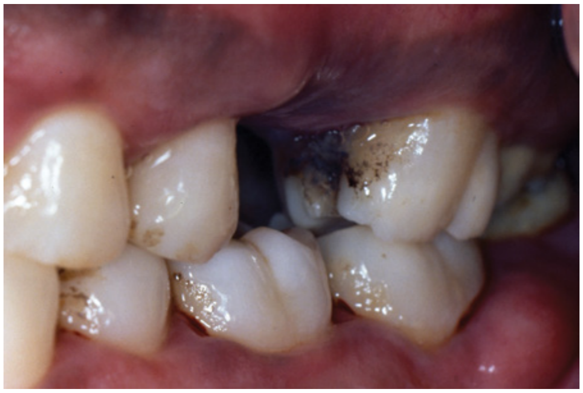

Figure 34-8C **Left Side, Facial View.**

© Wolters Kluwer

Clinical Photographs for Mr. Wilton

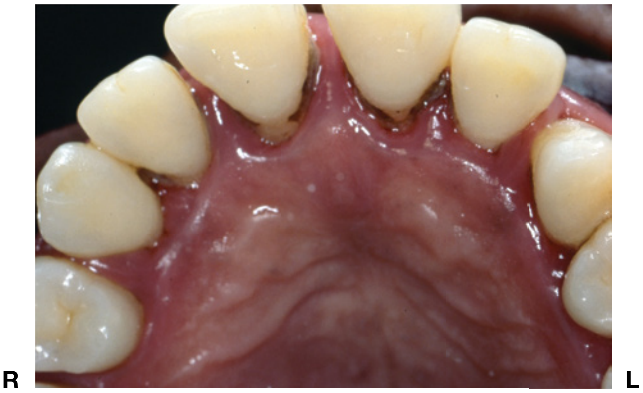

Figure 34-9A **Maxillary Anteriors, Lingual View.**

© Wolters Kluwer

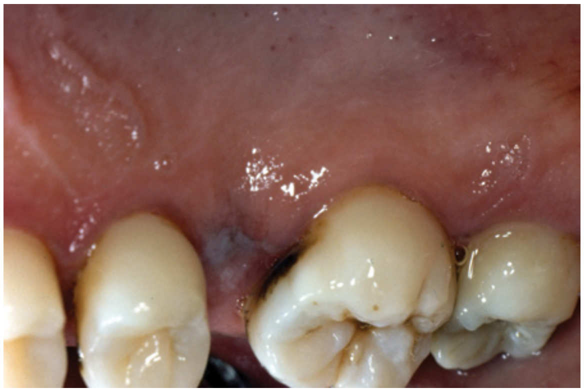

Figure 34-9B **Maxillary Right, Lingual View.**

© Wolters Kluwer

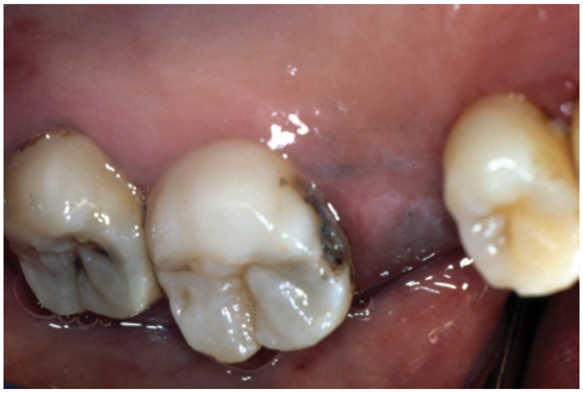

Figure 34-9C **Maxillary Left, Lingual View.**

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Clinical Photographs for Mr. Wilton

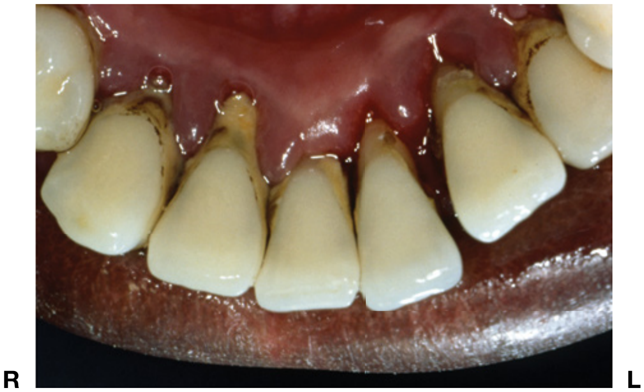

Figure 34-10A **Mandibular Anteriors, Lingual View.**

© Wolters Kluwer

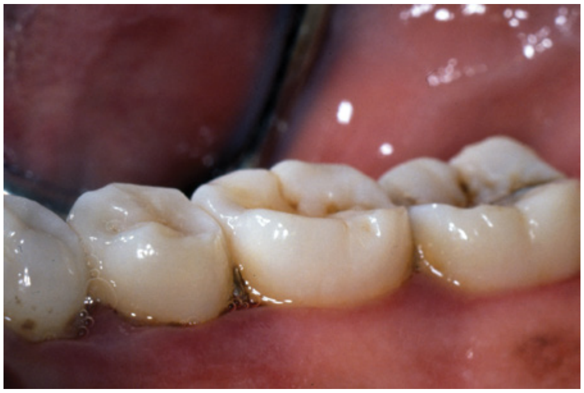

Figure 34-10B **Mandibular Right, Lingual View.**

© Wolters Kluwer

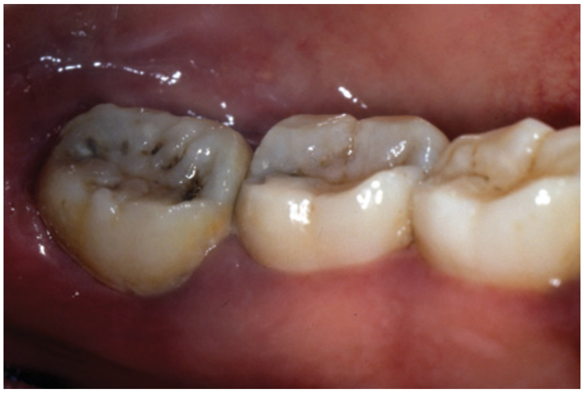

Figure 34-10C **Mandibular Left, Lingual View.**

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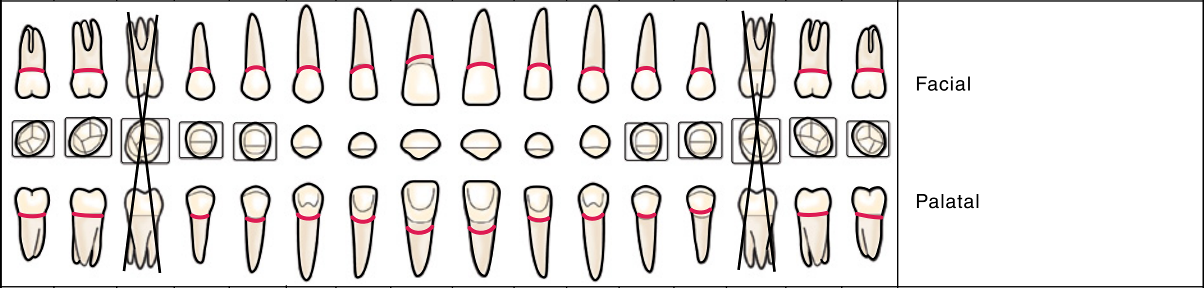
CASE #2

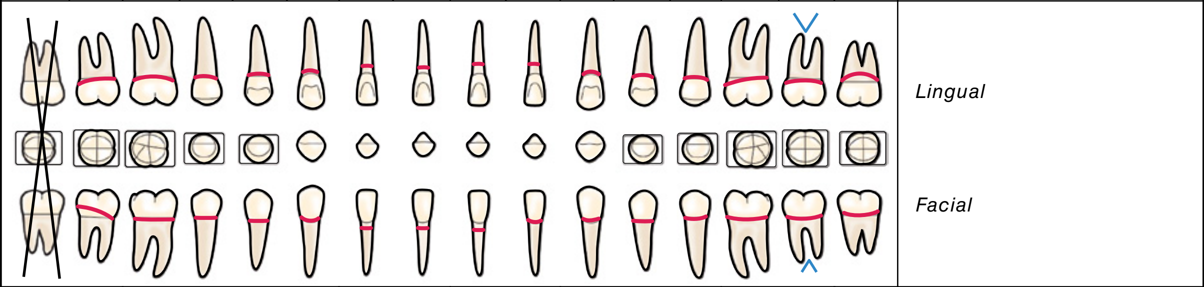
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	**Maxilla**
			I			I	I	II	I		I					Mobility (I, II, III)
+	+			+		+	+	+		+	+	+		+	+	Bleeding/Purulence (+)
635	634		338	535	537	626	736	537	625	536	725	524		435	535	Attachment Level (CEJ to BP)
635	634		338	535	537	626	625	537	625	536	725	524		435	535	Probing Depth (FGM to BP)



+	+		+	+	+		+	+		+	+	+		+		Bleeding/Purulence (+)
535	634		438	534	636	536	646	746	535	536	726	423		426	535	Attachment Level (CEJ to BP)
535	634		438	534	636	536	535	635	535	536	726	534		426	535	Probing Depth (FGM to BP)
⁘	⁘	✕	⁖	⁘	⁘	⁖	⁖	⁖	⁘	⁖	⁖	⁘	✕	⁘	⁘	F/P Plaque
	✓				✓	✓	✓	✓	✓	✓		✓		✓	✓	Supragingival Calculus
✓	✓		✓	✓	✓	✓	✓	✓	✓	✓	✓	✓		✓	✓	Subgingival Calculus
4						4						4				PSR Code

Right *Left*

32	31	30	29	28	27	26	25	24	23	22	21	20	19	18	17	**Mandible**
			I			II	II	II	II							Mobility (I, II, III)
	+	+	+		+	+	+	+	+	+	+	+	+		+	Bleeding/Purulence (+)
	435	536	545	524	535	746	656	647	748	635	524	535	535	636	645	Attachment Level (CEJ to BP)
	535	536	545	524	535	635	545	536	637	635	524	535	635	636	635	Probing Depth (FGM to BP)



	+	+		+	+	+	+	+	+	+	+		+	+	+	Bleeding/Purulence (+)
	435	526	535	424	535	735	646	636	536	524	525	425	525	526	625	Attachment Level (CEJ to BP)
	535	526	535	424	535	624	535	525	536	524	525	425	525	526	625	Probing Depth (FGM to BP)
✕	⁘	⁘	⁘	⁘	⁘	⁘	⁘	⁘	⁘	⁘	⁘	⁘	⁘	⁘	⁘	L/F Plaque
					✓	✓	✓	✓	✓	✓					✓	Supragingival Calculus
	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	Subgingival Calculus
4					4						4					PSR Code

Figure 34-11 **Mr. Wilton's Periodontal Chart.**

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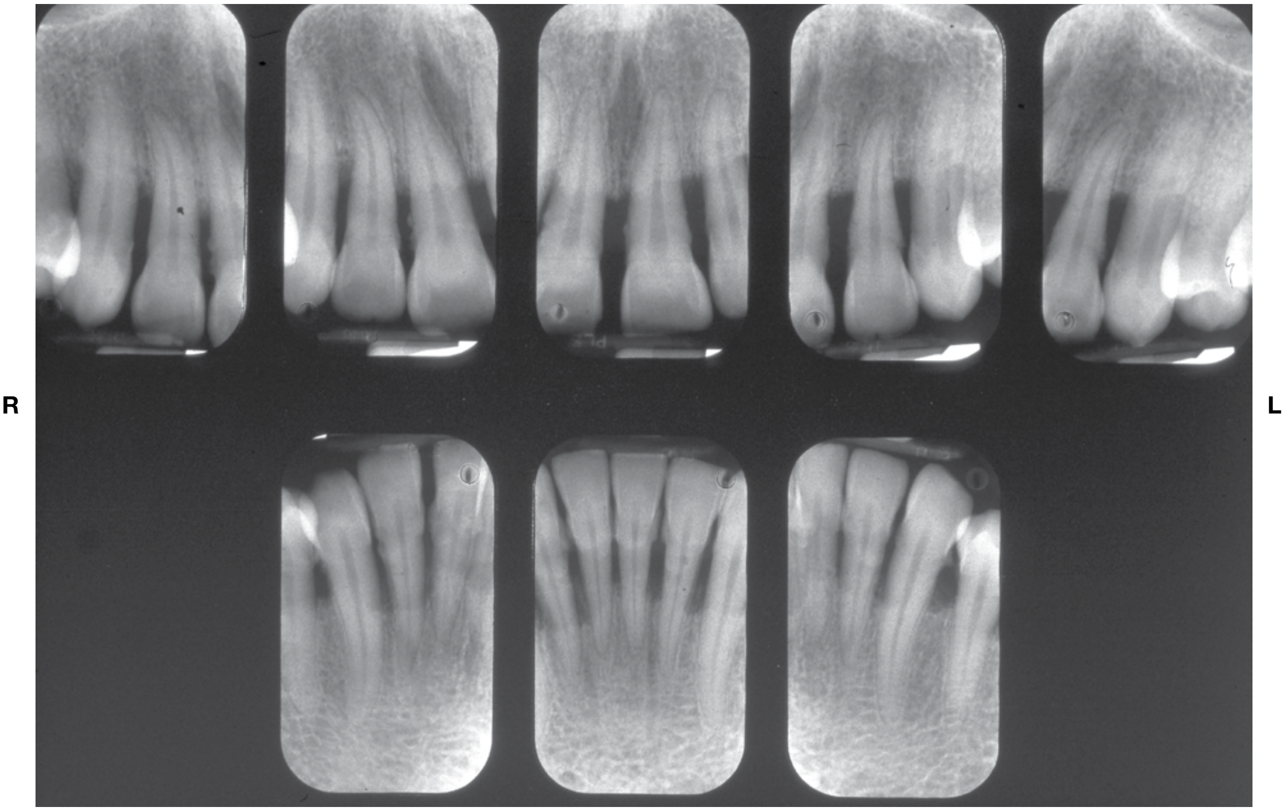

Figure 34-12A Radiographs: Anterior Teeth.

© Wolters Kluwer

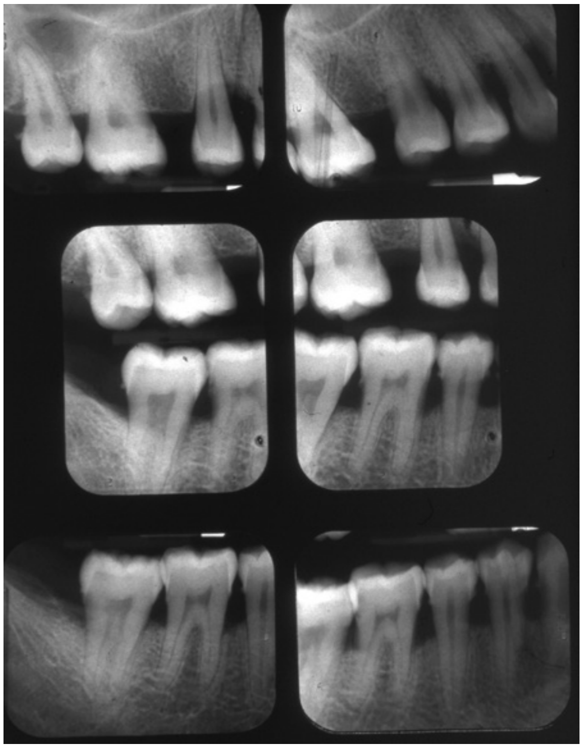

Figure 34-12B **Radiographs: Right Posterior Teeth.**

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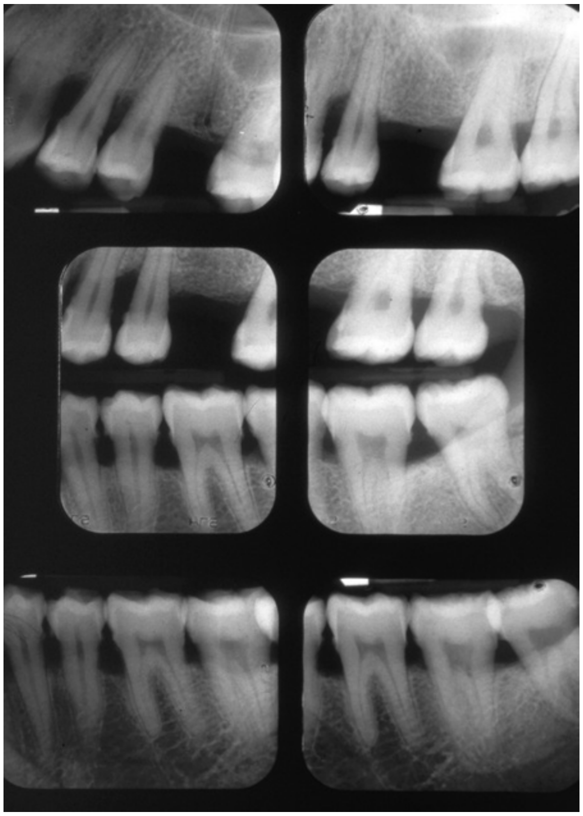
Figure 34-12C **Radiographs: Left Posterior Teeth.**

© Wolters Kluwer



Decision-Making Questions for Case 2: Mr. Wilton

1. What information should your team give Mr. Wilton regarding his bad breath? Note that this was the complaint that prompted Mr. Wilton to make an appointment in your office.
2. What factors in Mr. Wilton's profile indicate that achieving an acceptable level of patient self-care may be a problem for the dental team?
3. What factors revealed in Mr. Wilton's health history will be critical for the dental team to consider during periodontal evaluation or treatment?
4. What signs of gingival inflammation are evident in Mr. Wilton's clinical photographs?
5. What etiologic risk factors for gingival and periodontal diseases are evident in Mr. Wilton's clinical photographs?
6. Does Mr. Wilton's periodontal evaluation indicate that he has attachment loss present on some teeth?
7. In response to your questions, Mr. Wilton informs you that the spaces between his front teeth were not there a few years ago. What do you think may be causing these spaces between his teeth to appear?
8. What etiologic factors for periodontal diseases are evident in Mr. Wilton's dental radiographs?
9. On Mr. Wilton's radiographs, what specific findings indicate that he has alveolar bone loss present?
10. How would you characterize Mr. Wilton's periodontal condition? Do you think that he has gingivitis, periodontitis, neither, or both? What clinical or radiographic findings did you use to reach your conclusion?
11. Develop a step-by-step nonsurgical periodontal treatment plan for Mr. Wilton.
12. What information should your team give Mr. Wilton about his periodontal condition?
13. What should Mr. Wilton be told about the possible need for periodontal surgery later in the treatment?
14. What should Mr. Wilton be told about the need for continuing treatment such as periodontal maintenance?
15. What should your team tell Mr. Wilton if he refuses your team's recommendations for periodontal therapy?

Fictitious Patient Case 3–Ms. Sandsky

Figure 34-13. Mrs. Sandsky

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Patient Profile

Ms. Sandsky is a 42-year-old department store manager who has come to your dental office to get her dental condition in order. She states that she has neglected her dental care because she has been taking care of the dental needs of her children for many years, but now she is ready to take care of herself.

She informs your dental team that some years ago she was told that she had gum disease but elected not to receive any care for that condition at the time. She states that she brushes and flosses daily now in hopes that these actions can help her keep her teeth.

Patient Health History

- At the time of Ms. Sandsky's initial dental visit, her blood pressure measures 130/83 mm Hg and her pulse is 75 beats/min.
- She explains that she has problems with gastroesophageal reflux disease and elevated cholesterol.
- She reports that currently she is taking Prilosec® (omeprazole) and Lipitor® (atorvastatin) prescribed by her physician and that she is also taking multivitamin tablets because she thinks she needs them.
- Ms. Sandsky informs you that she smoked 1 pack of cigarettes daily for about 8 years when she was younger, but that she quit smoking 10 years ago and has not smoked since that time.
- Ms. Sandsky's dental history reveals a history of extractions of more than 5 teeth extracted due to periodontitis.

Clinical Photographs for Ms. Sandsky

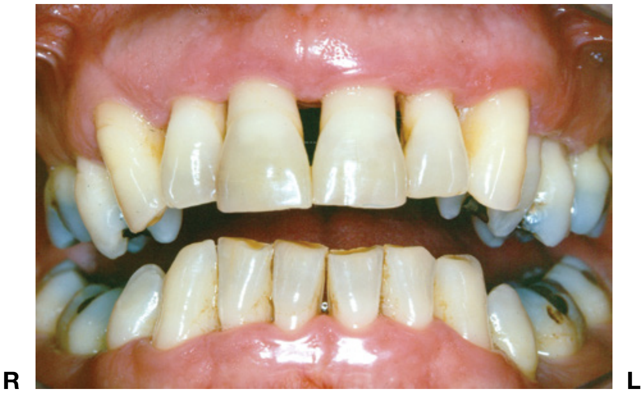

Figure 34-14A **Anterior Teeth, Facial View.**

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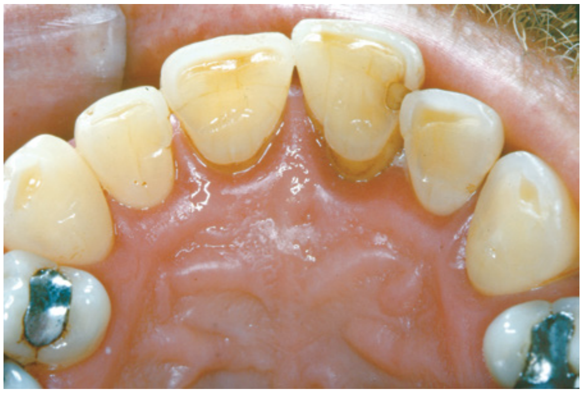


Figure 34-14B **Maxillary Anterior Teeth, Lingual View.**

© Wolters Kluwer

Clinical Photographs for Ms. Sandsky

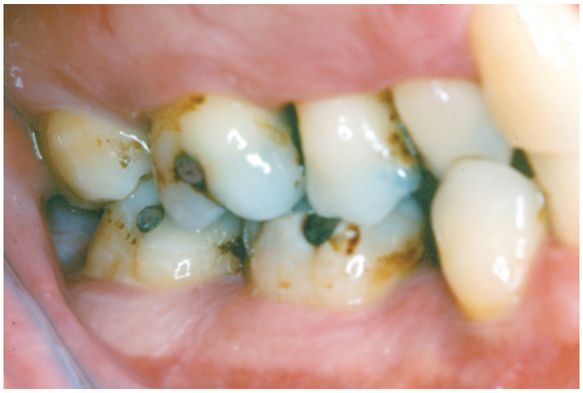


Figure 34-14C Right Side, Facial View.

© Wolters Kluwer

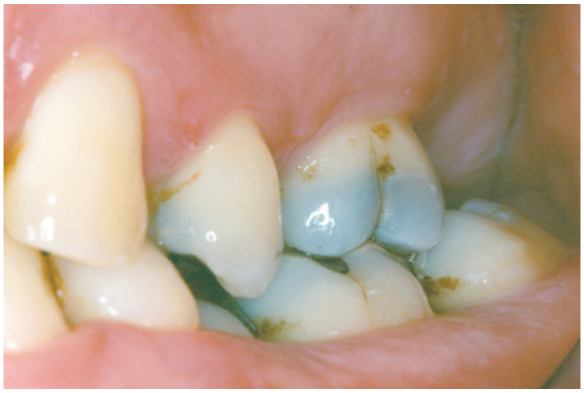
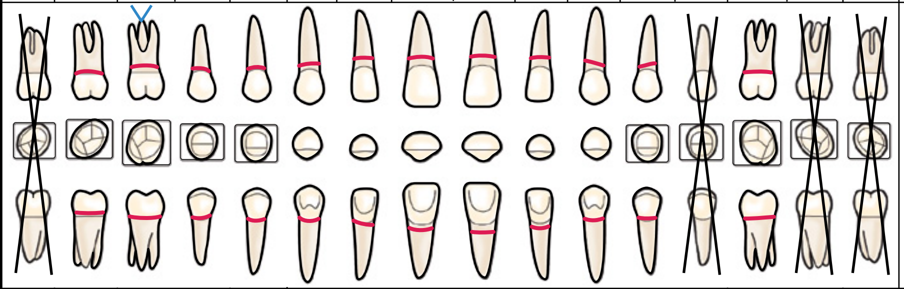


Figure 34-14D Left Side, Facial View.

© Wolters Kluwer

CASE #3

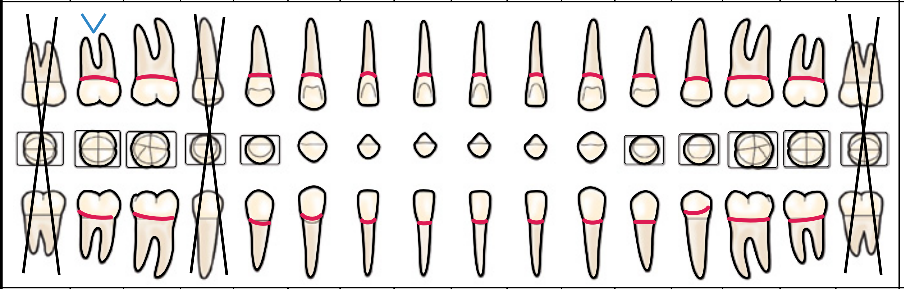
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	**Maxilla**
							I	I			I					Mobility (I, II, III)
	+	+	+	+		+	+	+		+	+		+			Bleeding/Purulence (+)
	646	544	535	545	646	656	656	656	756	655	646		545			Attachment Level (CEJ to BP)
	646	545	535	545	535	545	545	545	645	545	635		545			Probing Depth (FGM to BP)



	+	+	+	+	+	+	+	+	+	+	+		+			Bleeding/Purulence (+)
	645	545	543	545	545	645	656	656	666	545	535		545			Attachment Level (CEJ to BP)
	645	545	545	545	545	645	545	545	555	545	535		545			Probing Depth (FGM to BP)
																$^{F}_{P}$Plaque
	✓	✓	✓	✓		✓			✓	✓			✓			Supragingival Calculus
	✓	✓	✓	✓	✓	✓	✓	✓		✓	✓		✓			Subgingival Calculus
																PSR Code

Right *Left*

32	31	30	29	28	27	26	25	24	23	22	21	20	19	18	17	**Mandible**
																Mobility (I, II, III)
	+	+	+	+	+	+	+	+	+	+	+	+	+	+		Bleeding/Purulence (+)
	455	545		545	545	544	444	544	545	543	543	434	545	545		Attachment Level (CEJ to BP)
	455	545		545	545	544	444	544	545	545	434	434	545	545		Probing Depth (FGM to BP)



											I					Bleeding/Purulence (+)
	355	545		545	544	444	444	445	545	545	535	525	545	545		Attachment Level (CEJ to BP)
	455	545		544	544	444	444	445	545	545	535	646	545	545		Probing Depth (FGM to BP)
																$^{L}_{F}$Plaque
	✓	✓		✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓		Supragingival Calculus
	✓	✓		✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓		Subgingival Calculus
																PSR Code

Figure 34-15 **Ms. Sandsky's Periodontal Chart.**

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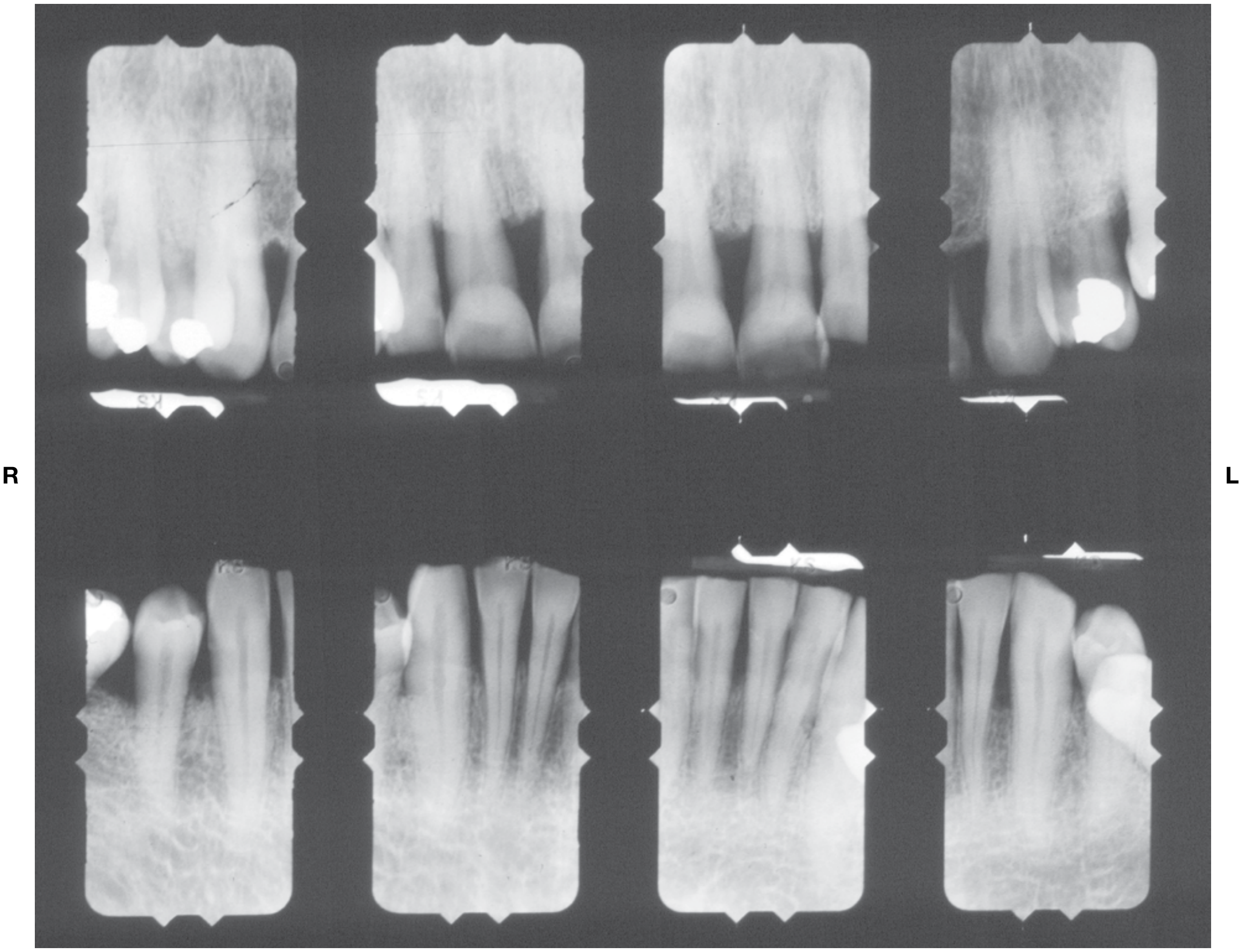

Figure 34-16A **Radiographs: Anterior Teeth.**

© Wolters Kluwer

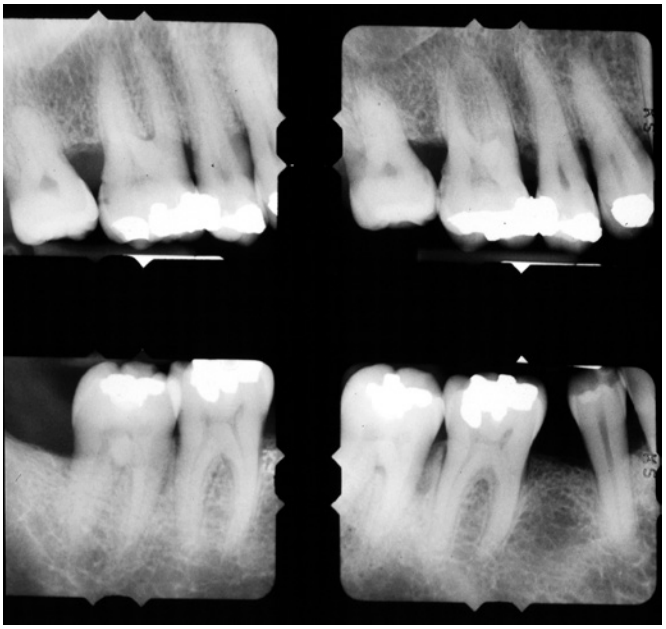

Figure 34-16B **Radiographs: Right Posterior Teeth.**

© Wolters Kluwer

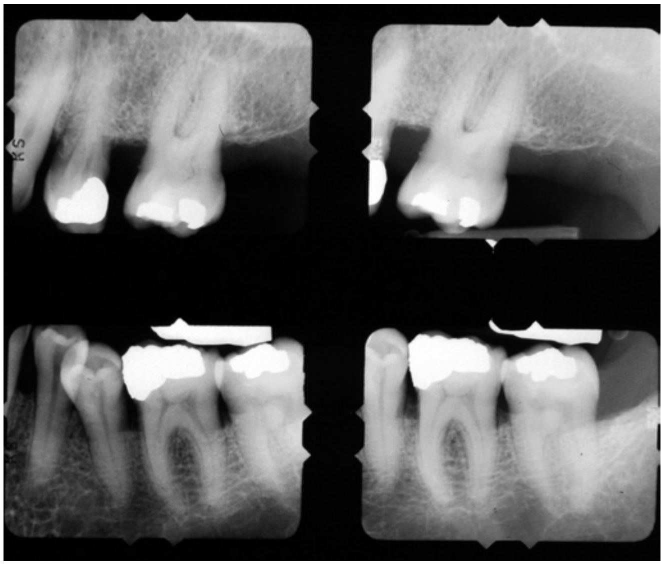

Figure 34-16C **Radiographs: Left Posterior Teeth.**

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Decision-Making Questions for Case 3: Ms. Sandsky

1. What factors in Ms. Sandsky's profile indicate that achieving an acceptable level of patient self-care may not be as difficult for her as it is for many patients?
2. Will the medications being taken by Ms. Sandsky require any special precautions during treatment by the members of the dental team?
3. How might the history of smoking relate to Ms. Sandsky's past and current risk for periodontal disease?
4. What signs of gingival inflammation are evident in Ms. Sandsky's clinical photographs?
5. What etiologic risk factors for gingival and periodontal diseases are evident in Ms. Sandsky's clinical photographs?
6. Does Ms. Sandsky's periodontal evaluation indicate that she has attachment loss present on some teeth?
7. In response to your questions, Ms. Sandsky informs you that the open triangular space between her upper front teeth was not there a few years ago. What do you think may be causing this space between her teeth to appear?
8. What etiologic factors for gingival and periodontal diseases are evident in Ms. Sandsky's dental radiographs?
9. How would you characterize Ms. Sandsky's periodontal condition? Do you think that she has gingivitis, periodontitis, neither or both? What clinical or radiographic findings did you use to reach your conclusion?
10. Develop a step-by-step nonsurgical treatment plan for Ms. Sandsky.
11. What information should your team give Ms. Sandsky about her periodontal condition?
12. What should Ms. Sandsky be told about the possible need for periodontal surgery later in the treatment?
13. What should Ms. Sandsky be told about the need for continuing treatment such as periodontal maintenance?

Fictitious Patient Case 4—Mr. Verosky

Figure 34-17 Mr. Verosky

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Patient Profile

Mr. Verosky is a 62-year-old male who was recently elected to a local city council. Since being elected, he has become very interested in getting his oral health up to par.

During his initial visit, Mr. Verosky explains that he has always had spaces between his front teeth and is not really worried about that. He tells you that his major concern is that he has been told he has periodontal disease, and he does not want to lose his teeth.

Patient Health History

- On the day of his first visit to your dental office Mr. Verosky's blood pressure is 142/90 mm Hg and his pulse is 66 beats/min.
- Review of Mr. Verosky's health history reveals that he is supposed to be taking two medications: losartan/hydrochlorothiazide tablets and low-dose aspirin, but he readily admits that he frequently "forgets" to take his medications.
- Mr. Verosky also states that he smoked cigarettes prior to age 40, but that he quit smoking during his early 40s.

Clinical Photographs for Mr. Verosky

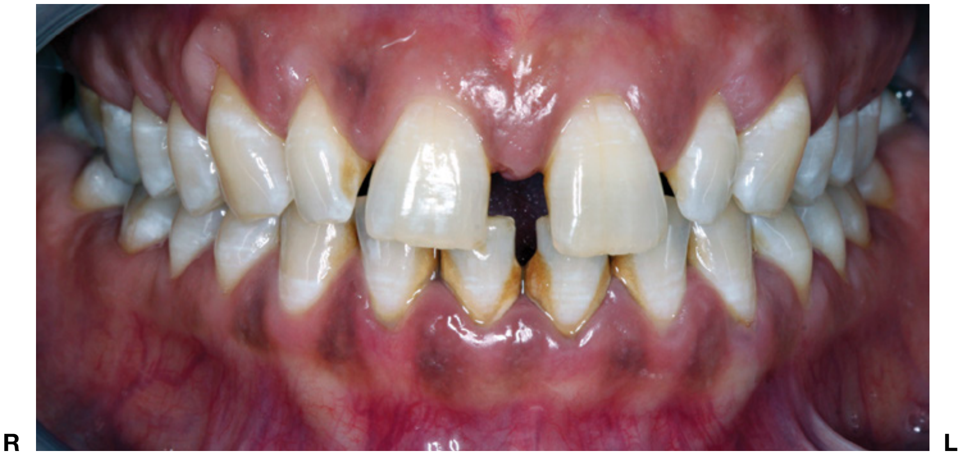

Figure 34-18A **Anterior Teeth, Facial View.**

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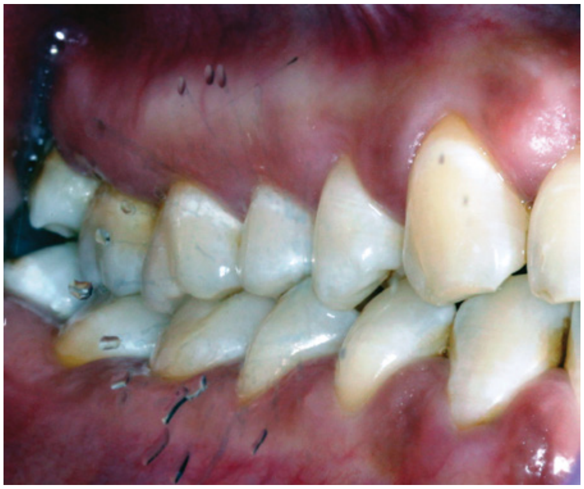

Figure 34-18B **Right Side, Facial View.**

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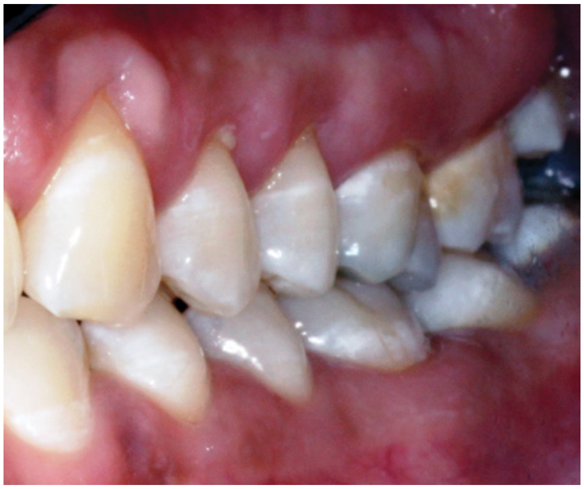

Figure 34-18C **Left Side, Facial View.**

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Clinical Photographs for Mr. Verosky

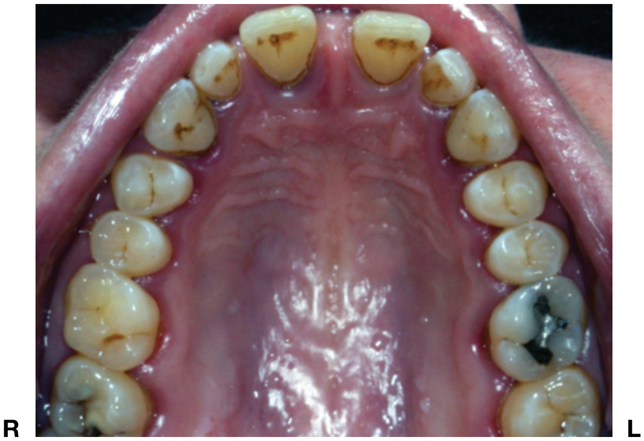

Figure 34-19A **Maxillary Arch, Lingual View.**

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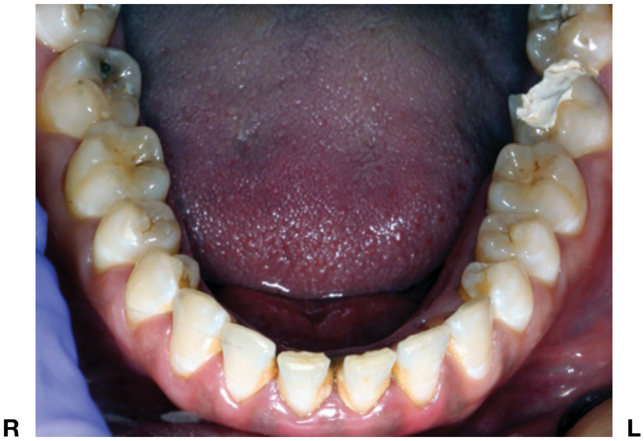

Figure 34-19B **Mandibular Arch.**

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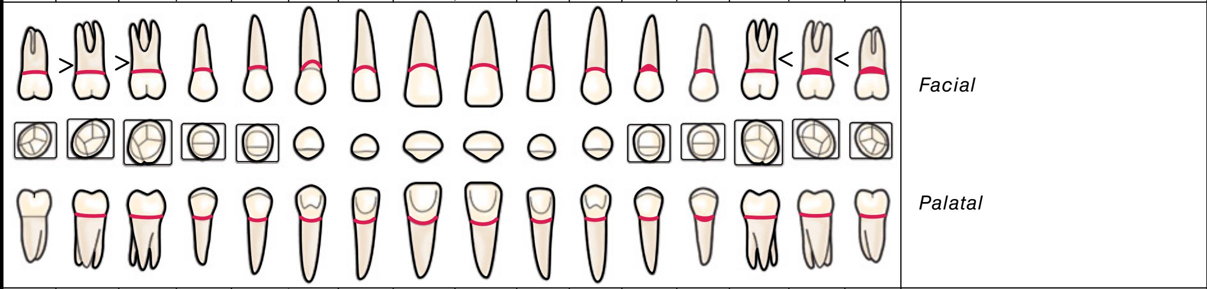
CASE #4

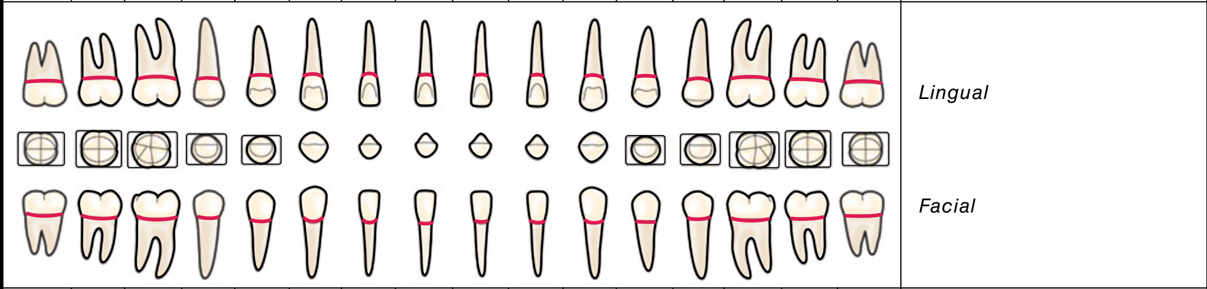
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	**Maxilla**
		I											I			Mobility (I, II, III)
+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	Bleeding/Purulence (+)
336	867	646	241	342	151	242	111	111	233	232	234	313	425	447	544	Attachment Level (CEJ to BP)
558	756	535	322	423	222	323	313	313	324	323	325	424	425	446	655	Probing Depth (FGM to BP)



+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	Bleeding/Purulence (+)
233	665	676	212	222	444	222	223	323	222	222	223	312	555	558	410	Attachment Level (CEJ to BP)
555	654	545	323	333	555	333	334	434	333	333	334	423	424	437	633	Probing Depth (FGM to BP)
																F/P Plaque
✓	✓	✓											✓	✓	✓	Supragingival Calculus
✓	✓	✓	✓	✓					✓		✓	✓	✓	✓	✓	Subgingival Calculus
4					3						4					PSR Code

Right *Left*

32	31	30	29	28	27	26	25	24	23	22	21	20	19	18	17	**Mandible**
I															I	Mobility (I, II, III)
+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	Bleeding/Purulence (+)
336	444	433	333	222	222	222	221	122	331	121	232	244	443	534	434	Attachment Level (CEJ to BP)
547	555	544	434	323	323	323	322	223	422	222	333	345	544	545	655	Probing Depth (FGM to BP)



+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	Bleeding/Purulence (+)
344	444	432	222	222	222	232	222	232	222	222	232	232	233	435	423	Attachment Level (CEJ to BP)
555	525	523	323	323	323	333	323	333	323	323	323	323	324	526	533	Probing Depth (FGM to BP)
																L/F Plaque
					✓	✓	✓	✓	✓	✓						Supragingival Calculus
✓	✓	✓	✓		✓	✓	✓	✓	✓	✓	✓		✓	✓	✓	Subgingival Calculus
3					3						4					PSR Code

Figure 34-20 **Mr. Verosky's Periodontal Chart.**

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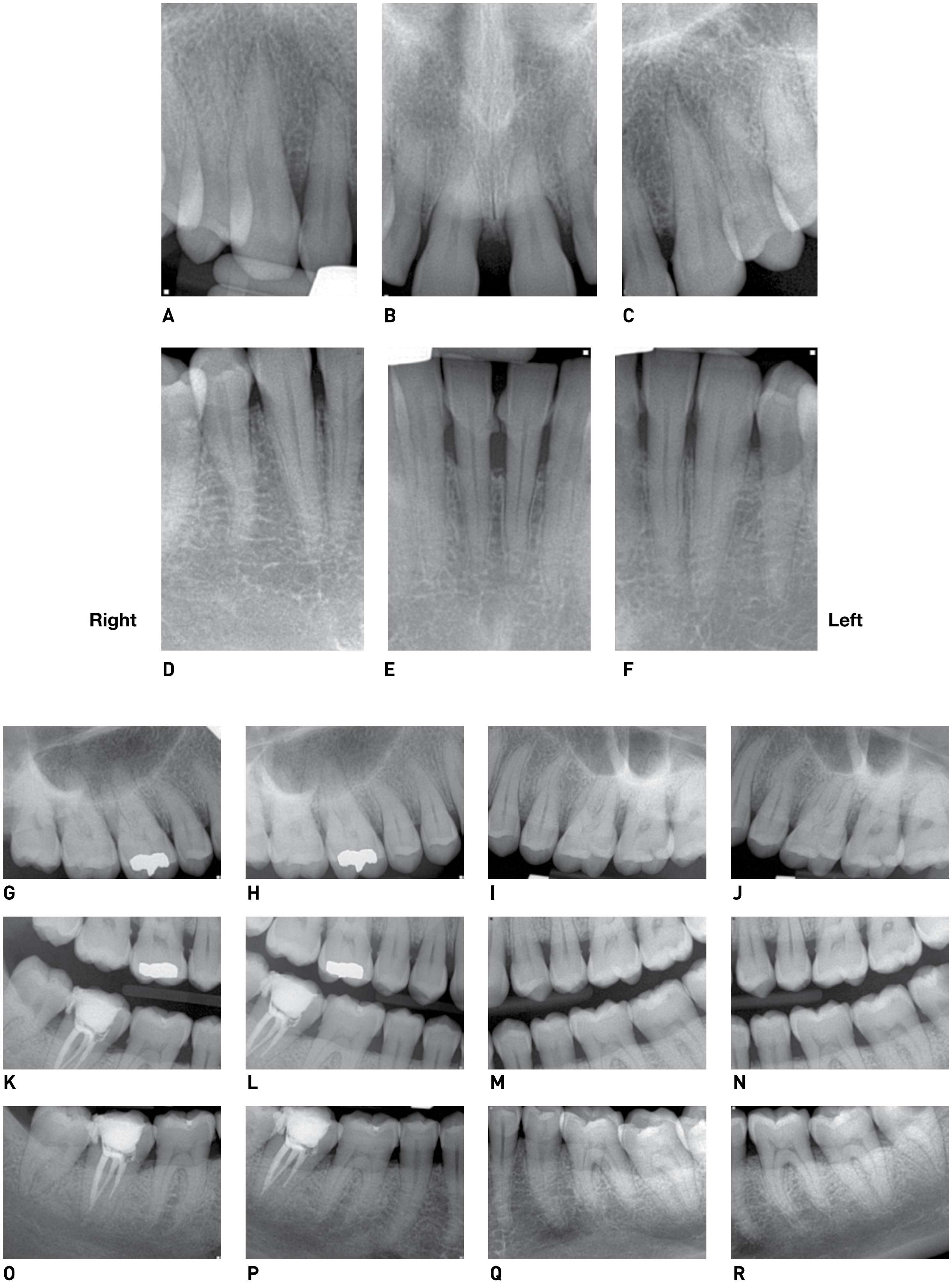
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Figure 34-21A–R Radiographs for Mr. Verosky.

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Decision-Making Questions for Case 4: Mr. Verosky

1. What should Mr. Verosky be told about the spaces between his anterior teeth?
2. What factors in Mr. Verosky's health history will be critical for the dental team to consider during treatment?
3. What signs of inflammation are evident on Mr. Verosky's clinical photographs?
4. What etiologic risk factors for gingival and periodontal diseases are evident on Mr. Verosky's clinical evaluation?
5. Does Mr. Verosky's periodontal evaluation indicate that he has attachment loss on some of his teeth? How did you arrive at your conclusion?
6. What etiologic risk factors for gingival and periodontal diseases are evident from Mr. Verosky's radiographs?
7. On Mr. Verosky's radiographs, what specific findings indicate that the alveolar bone level is normal or abnormal?
8. How should you characterize Mr. Verosky's periodontal condition? Do you think that he has gingivitis, periodontitis, neither, or both? What clinical or radiographic findings did you use to reach your conclusion?
9. Develop a step-by-step nonsurgical treatment plan for for Mr. Verosky.
10. What information should your team give Mr. Verosky about his periodontal condition?
11. What should Mr. Verosky be told about the possible need for periodontal surgery later in his treatment?
12. What should Mr. Verosky be told about the need for continuing treatment such as periodontal maintenance?
13. Mr. Verosky has a temporary restoration in a lower molar tooth. What should he be told about this restoration?

Fictitious Patient Case 5—Mr. Tomlinson

Figure 34-22. Mr. Tomlinson

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Patient Profile

Mr. Tomlinson is a 48-year-old patient who is new to your dental office. He works as a computer technician in a large local firm. This is his first job following an extended tour of military duty.

During his initial visit, Mr. Tomlinson explains that he has no dental problems, but he decided to make an appointment for a "cleaning" because he has a new dental insurance plan. He tells you that everyone in his family has always had pretty good teeth, so he is certain that you will not find anything wrong with his teeth or with his old fillings.

Patient Health History

- On the day of his first visit to your dental office, Mr. Tomlinson's blood pressure is 132/82 mm Hg and his pulse is 64 beats/min.
- Review of Mr. Tomlinson's health history reveals he is currently on Fosamax® (alendronate) to manage his osteoporosis and Flonase®, a fluticasone propionate nasal spray for seasonal allergies.
- He states that he is currently under treatment for posttraumatic stress disorder (PTSD) that has resulted from his multiple military deployments. Currently, he is on Zoloft® (which is a brand name for the drug sertraline) and Prozac® (which is a brand name for the drug fluoxetine) to manage his PTSD.
- He cannot recall the reasons for his past extractions.

Clinical Photographs for Mr. Tomlinson

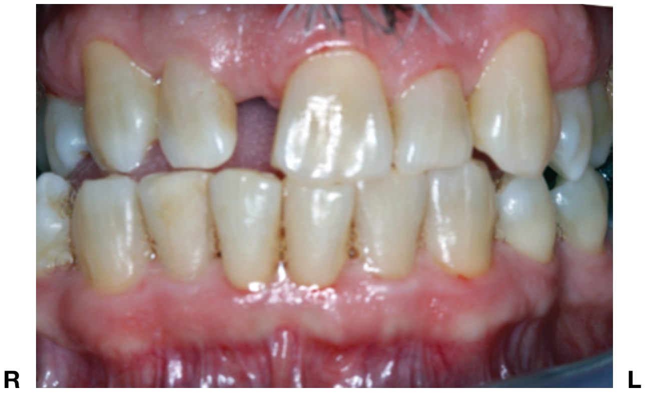

Figure 34-23A **Anterior Teeth, Facial View.**

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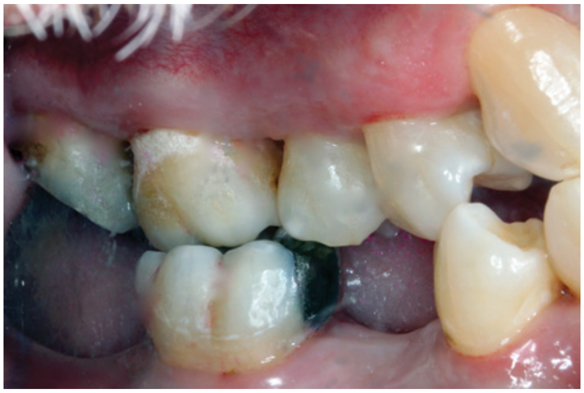

Figure 34-23B **Right Side, Facial View.**

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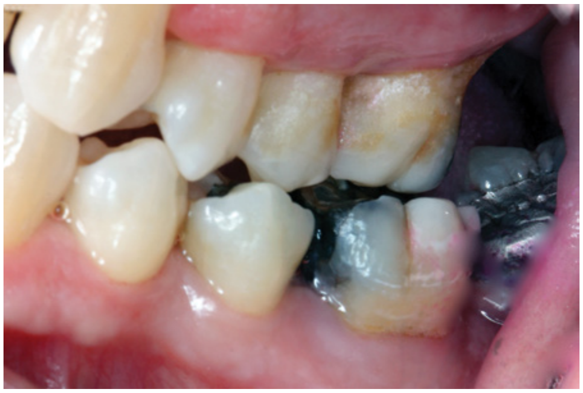

Figure 34-23C **Left Side, Facial View.**

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Clinical Photographs for Mr. Tomlinson

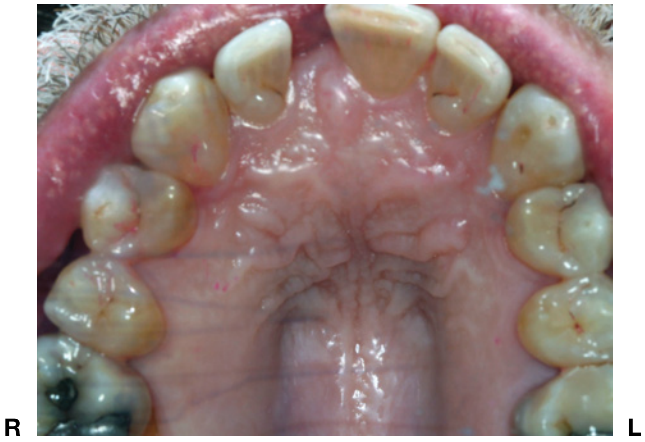

Figure 34-24A **Maxillary Arch, Lingual View.**

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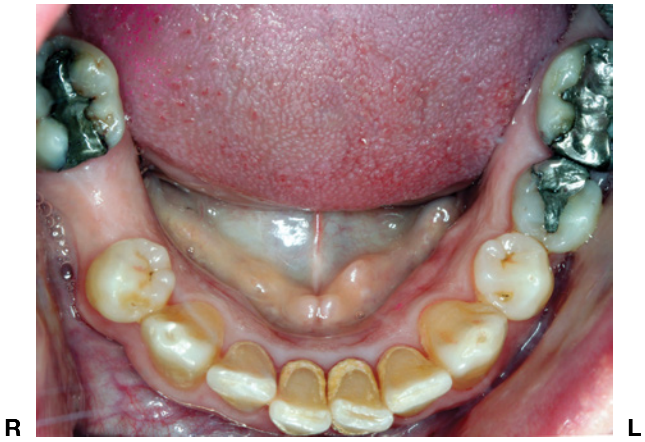

Figure 34-24B **Mandibular Arch.**

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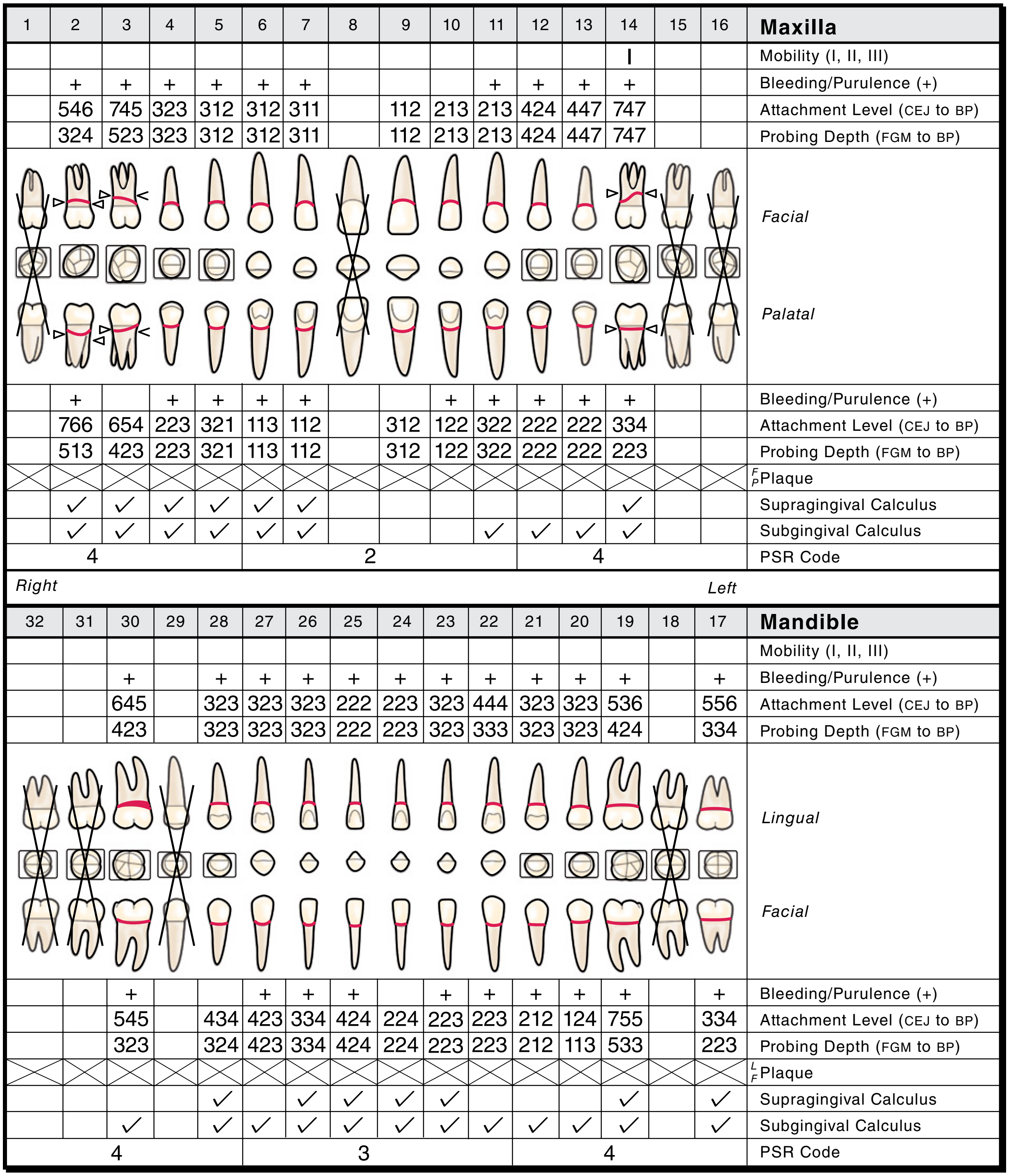
CASE #5



1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	**Maxilla**
													I			Mobility (I, II, III)
	+	+	+	+	+	+				+	+	+	+			Bleeding/Purulence (+)
	546	745	323	312	312	311		112	213	213	424	447	747			Attachment Level (CEJ to BP)
	324	523	323	312	312	311		112	213	213	424	447	747			Probing Depth (FGM to BP)
																Facial
																Palatal
	+		+	+	+	+			+	+	+	+	+			Bleeding/Purulence (+)
	766	654	223	321	113	112		312	122	322	222	222	334			Attachment Level (CEJ to BP)
	513	423	223	321	113	112		312	122	322	222	222	223			Probing Depth (FGM to BP)
X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	$^{F}_{P}$Plaque
	✓	✓	✓	✓	✓	✓							✓			Supragingival Calculus
	✓	✓	✓	✓	✓	✓				✓	✓	✓	✓			Subgingival Calculus
4					2						4					PSR Code

Right *Left*

32	31	30	29	28	27	26	25	24	23	22	21	20	19	18	17	**Mandible**
																Mobility (I, II, III)
		+		+	+	+	+	+	+	+	+	+	+		+	Bleeding/Purulence (+)
		645		323	323	323	222	223	323	444	323	323	536		556	Attachment Level (CEJ to BP)
		423		323	323	323	222	223	323	333	323	323	424		334	Probing Depth (FGM to BP)
																Lingual
																Facial
		+			+	+	+		+	+	+	+	+		+	Bleeding/Purulence (+)
		545		434	423	334	424	224	223	223	212	124	755		334	Attachment Level (CEJ to BP)
		323		324	423	334	424	224	223	223	212	113	533		223	Probing Depth (FGM to BP)
X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	$^{L}_{F}$Plaque
				✓		✓	✓	✓	✓				✓		✓	Supragingival Calculus
		✓		✓	✓	✓	✓	✓	✓	✓	✓	✓	✓		✓	Subgingival Calculus
4					3						4					PSR Code

Figure 34-25 **Mr. Tomlinson's Periodontal Chart.**

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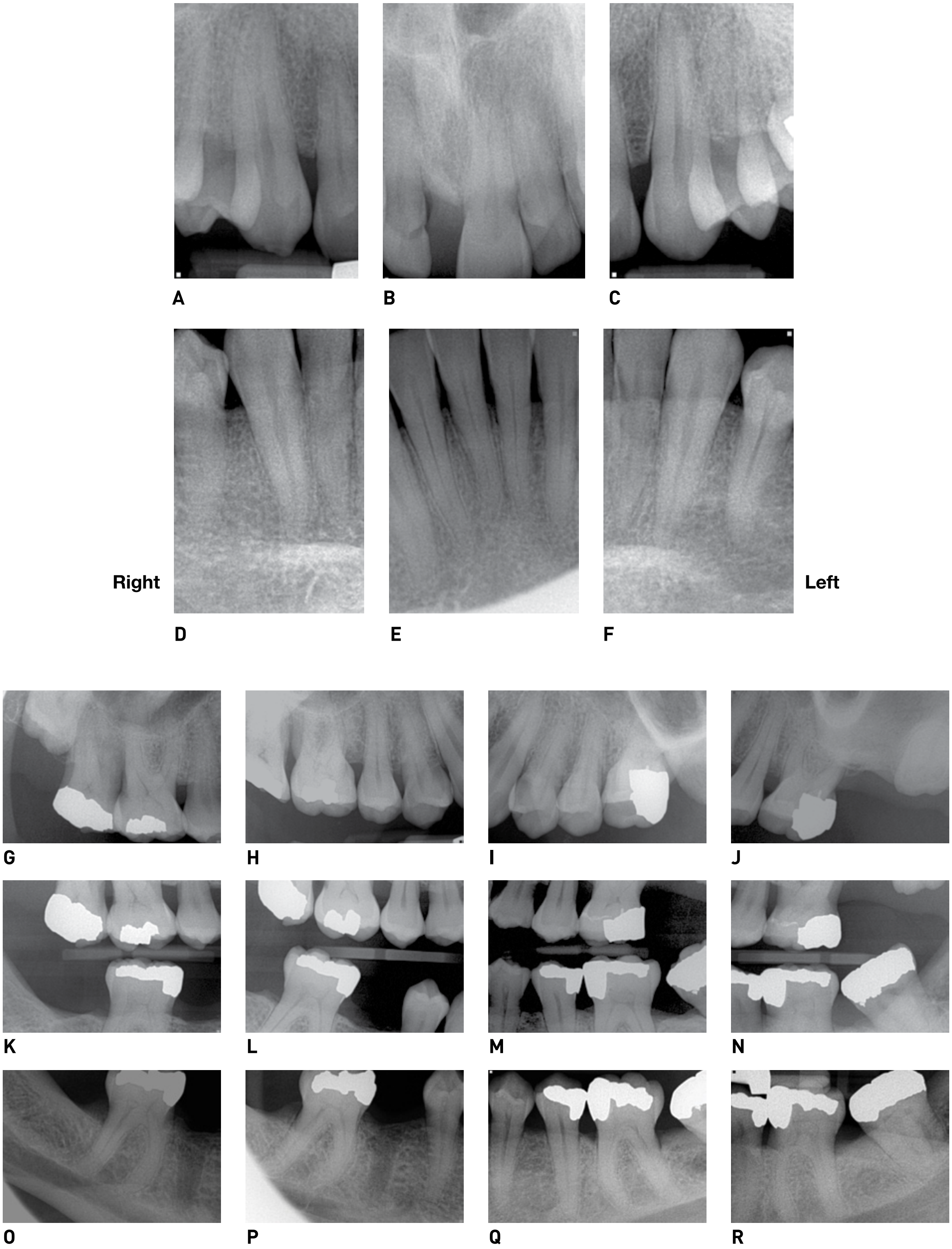
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Figure 34-26A–R Radiographs for Mr. Tomlinson.

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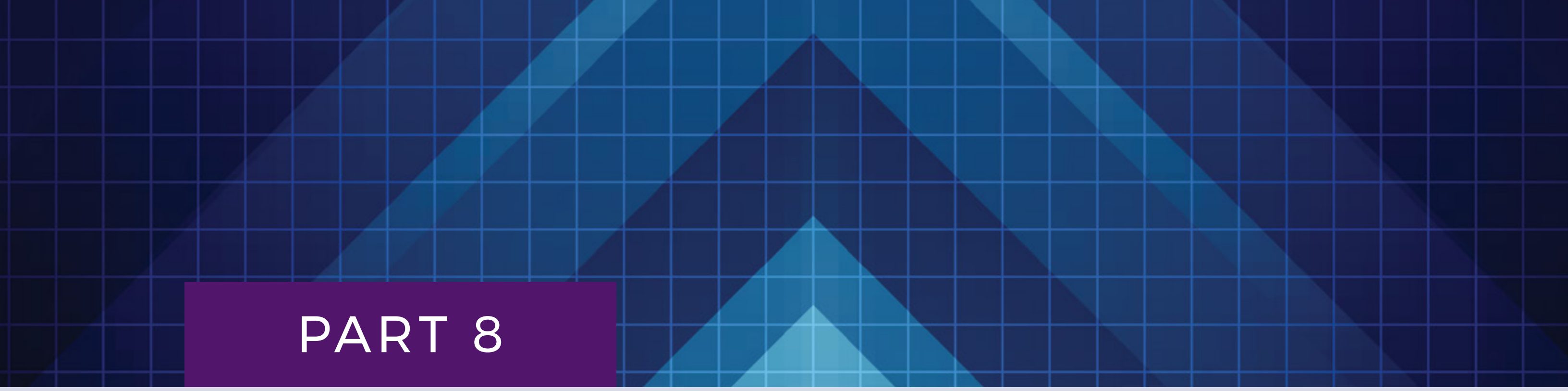
Decision-Making Questions for Mr. Tomlinson

1. What factors in Mr. Tomlinson's health history will be critical for the dental team to consider during treatment?
2. What signs of inflammation are evident on Mr. Tomlinson's clinical photographs?
3. What etiologic risk factors for gingival and periodontal diseases are evident from Mr. Tomlinson's clinical evaluation?
4. Does Mr. Tomlinson's periodontal evaluation indicate that he has attachment loss on some of his teeth? How did you arrive at your conclusion?
5. What etiologic risk factors for gingival and periodontal diseases are evident Mr. Tomlinson's radiographs?
6. On Mr. Tomlinson's radiographs, what specific findings indicate that the alveolar bone level is normal or abnormal?
7. How should you characterize Mr. Tomlinson's periodontal condition? Do you think that he has gingivitis, periodontitis, neither or both? What clinical or radiographic findings did you use to reach your conclusion?
8. Develop a step-by-step nonsurgical treatment plan for Mr. Tomlinson.
9. What information should your team give Mr. Tomlinson about his periodontal condition?
10. What should Mr. Tomlinson be told about the possible need for periodontal surgery later in his treatment?
11. What should Mr. Tomlinson be told about the need for continuing treatment such as periodontal maintenance?



PART 8

Online Resources

CHAPTER 35 Periodontal Disease in the Pediatric Population. 3

CHAPTER 36 Iatrosedation: Easing and Managing Pediatric Patient Fears 1

CHAPTER 37 Oral Malodor, Xerostomia, and Hyposalivation. 1

CHAPTER 38 Patient Cases: Radiographic Analysis 1

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CHAPTER 35

Periodontal Disease in the Pediatric Population

- Section 1 Periodontal Anatomy of the Primary Dentition
- Section 2 Gingival Diseases in Pediatric Patients
  - Eruption Gingivitis and Pericoronal Abscess
  - Plaque-Induced Gingival Disease
  - Gingival Diseases Modified by Medications
- Section 3 Common Types of Periodontitis in Pediatric Patients
  - Periodontitis
  - More Aggressive Forms of Periodontitis
  - Necrotizing Periodontal Diseases
- Section 4 Common Acute Periodontal Conditions in Pediatric Patients
  - Herpes Virus Infections and Aphthous Ulcers
- Section 5 Focus on Patients
  - Clinical Patient Care
  - Ethical Dilemma

CLINICAL APPLICATION

In 2021, the National Institutes of Health (NIH) issued a comprehensive follow-up to the landmark 2000 report *Oral Health in America: A Report of the Surgeon General*. Among the many important themes addressed in the 2021 NIH report (titled *Oral Health in America: Advances and Challenges*) is that oral health promoted early on in life, then maintained throughout childhood and adolescence, is the foundation to establishing good oral health *and* general health later on in life. As such, efforts to manage and preserve oral health at an early age is integral to ensuring normal orofacial development, general health, intellectual progression, and social maturation. This chapter aims to highlight the differences in periodontal anatomy associated with deciduous teeth versus that of adult teeth and will review the common types of gingival and periodontal diseases/conditions that affect the pediatric population. It will also present treatment strategies that can be employed when managing child/adolescent patients with these conditions.

LEARNING OBJECTIVES

- Describe the normal clinical appearance of the periodontal anatomy surrounding the primary dentition and be able to explain how these features differ from the clinical appearance of the periodontium surrounding the permanent dentition.
- Describe the normal radiographic appearance of the periodontal anatomy surrounding the primary dentition and be able to explain how these features differ from the normal radiographic appearance found around the permanent dentition.
- Explain the differences between a "cold sore" and a "canker sore" and describe treatment strategies for each condition.

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- Explain the importance of educating pediatric patients and their caretakers about the relationship between periodontal health and oral development.
- Describe the potential implications of untreated periodontal disease in the pediatric patient.
- Describe the common types of acute periodontal conditions that are seen in the pediatric population.
- Recognize the common forms of periodontal diseases that affect the pediatric patient and detail the treatment regimens to manage each of these conditions.

KEY TERMS

Eruption gingivitis
Pericoronitis
Operculum
Plaque-induced gingivitis
Puberty gingivitis
Drug-influenced gingivitis
Drug-influenced gingival enlargement
Familial aggregation
Episodic
Molar/incisor pattern
Generalized pattern
Primary herpetic gingivostomatitis
Recurrent herpes simplex labialis
Recurrent aphthous ulcers

Section 1

Periodontal Anatomy of the Primary Dentition

The 2021 NIH report *Oral Health in America: Advances and Challenges* reinforces the important concept that oral health instilled at early childhood influences the outcome of oral health and systemic health at a later age.[1] Conversely, common oral diseases that impact the quality of life of adults (such as untreated periodontal disease and untreated dental caries) often start out earlier in life when the individual is in childhood or adolescence.[1] Hence, within the clinical setting, an oral health care provider must be able to recognize and intercept a wide array of commonly encountered pediatric periodontal conditions. At the same time, the oral health care provider must be able to distinguish the key clinical and radiographic features of periodontal health versus periodontal disease in the child and adolescent population.

The clinical and radiographic characteristics of the periodontium in children and adolescents differ somewhat from those seen in adults. The periodontium during childhood and puberty is in a constant rapid state of change because of the growth of the jaws, the exfoliation of deciduous teeth, and the eruption of permanent teeth. This section outlines the important characteristics of pediatric and adolescent periodontium.

1. **Periodontium of the Primary Dentition**
   a. **Clinical Assessment.** The clinical appearance of the oral soft tissues of the primary (deciduous) dentition differs from that of the permanent dentition in several aspects. **Figure 35-1** shows a typical clinical appearance of the healthy gingiva of a child. Some of the key differences in the periodontal anatomy of children and adults are listed below and summarized in **Table 35-1**.
      1. Gingival color appears more reddish because of increased vascularity and a thinner overlying gingival epithelium, which increases the visibility of the underlying blood vessels.[2,3] In contrast, the healthy periodontium of an adult has a coral pink color.
      2. Gingival surface texture appears less stippled or smoother than that of an adult. Stippling appears at about 3 years of age and is present in about 56% of children between ages 3 and 10 years.[4]
      3. Gingival contour of a healthy pediatric patient is characterized by a free marginal gingiva that meets the tooth with a rounded margin. The prominence of the bulbous cervical ridge of the crown of the deciduous tooth accounts for the thick rounded form of the overlying free marginal gingiva. In contrast, an adult with a healthy periodontium has a free gingival margin that has a sharp knife-like edge that is closely adapted around the necks of the teeth.
      4. Gingival consistency of a healthy pediatric patient may have a flabby or loose consistency due to the comparatively less well developed net of collagen fibers than in adults.
      5. The gingival sulcus (as measured with a periodontal probe) starts off as being shallow at an early age but becomes deeper as pediatric patients become older. In other words, there is a tendency for the probing depths to increase as the pediatric patient's age increases. In a study that was conducted to

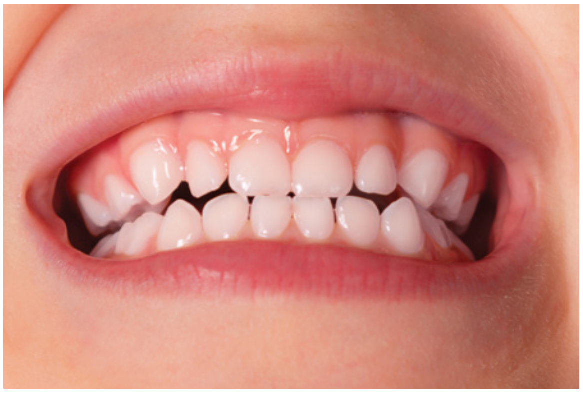

Figure 35-1 **A Healthy Pediatric Periodontium.** This clinical photograph shows a child with the clinical features characteristics of a healthy periodontium. Note the minimal signs of inflammation, the smooth gingival texture, and the rounded contours of the free marginal gingiva.

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Table 35-1 **Differing Clinical Characteristics of the Healthy Periodontium in Children Versus Adults**

Features	Children	Adults
Gingival color	More reddish	Coral pink
Contour	Rounded free marginal gingiva	Sharp, knife-edged marginal gingiva that is closely adapted to the cervical neck of a tooth
Consistency	Flabby due to less connective tissue density and lack of organized fiber bundles	Firm and resilient
Surface texture	Stippling absent in infancy; may be seen by age 6 years. However, note that in many cases the healthy pediatric gingiva may not exhibit any gingival stippling but may appear smooth.	Stippling may be present; however, its pattern and extent varies in different areas of the mouth of the same individual.
Interdental area	Saddle-shaped interdental gingiva	Triangular papillary gingiva
Attached gingiva	Width increases with age	Width greater in adults

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evaluate the relationship between age and probing depth in each primary tooth, it was found that the mean probing depth of an upper central incisor on the facial side at 4 years of age was 1.26 mm and 1.55 mm at 6 years.[5] In addition, in the majority of facial and lingual sites measured in the same study, mean values increased from anterior to posterior positions in both arches. The mean probing depths values for all teeth examined showed the highest mean value in lingual sites of mandibular second molars (2.08 mm) and the lowest mean in lingual sites of mandibular central incisors (1.03 mm).[5]

6. The interdental papilla is rounded (saddle-shaped) with a broad facial to lingual dimension and a narrow form from the mesial to distal aspect.
7. The width of the attached gingiva is narrower in the mandible than in the maxilla, and both widths increase with age as children transition from primary to permanent dentition.[6] Several studies confirm that an increase in attached gingiva is correlated with age.[5]

b. **Radiographic Assessment.** Differences in periodontal anatomy are also noted upon radiographic assessment. These include (**Figure 35-2**):

1. The periodontal ligament (PDL) spaces surrounding deciduous teeth are wider than those surrounding adult teeth.[2,3,7]
2. Alveolar bone in the primary dentition has less trabeculae and calcification with larger marrow spaces.[2,3,7]

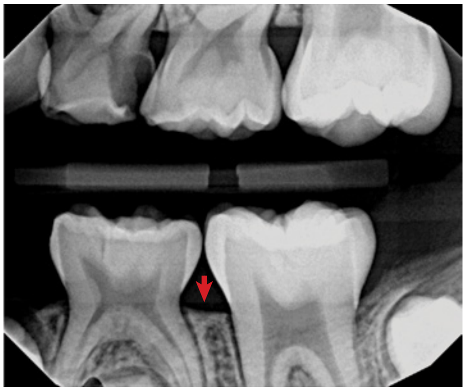

Figure 35-2 Radiographic Characteristics of a Healthy 8-year-old Child. Observe the pronounced lamina dura and the flat broad interdental septa between the deciduous first molar and the permanent first molar (red arrow).

3. Lamina dura (the radiopaque lining of an alveolus) is prominent in the primary dentition with a wider periodontal ligament space than in the permanent dentition.
4. Interdental septa of pediatric patients are generally broader and flatter than the interdental septa seen in healthy adults with bony crests within 1 to 2 mm of the cementoenamel junction.

Section 2

Gingival Diseases in Pediatric Patients

Although the prevalence of gingivitis among children and adolescents is high, a low percentage of children suffer from periodontitis. Nevertheless, although the percentage of children suffering from periodontitis is low, it is clear that oral health care providers need to examine every child's oral health with care because failure to identify periodontal conditions early can lead to unnecessary tooth loss. Further, considering the strong association between cigarette smoking and periodontal disease, the rise in the use of newer noncombustible nicotine products among youths, such as e-cigarettes, and its subsequent health ramifications is equally alarming.[8] For a review of all the potential adverse effects of e-cigarettes and other smoking-related products, the reader can refer to Chapter 17.

The American Academy of Periodontology and the American Academy of Pediatric Dentistry[9–11] have reported the following points related to gingival and periodontal diseases in children and adolescents:

- Gingivitis of varying severity is nearly universal in children and adolescents.
- In children, the amount of plaque biofilm often does not correlate with the level of inflammation; it is not uncommon to see copious amounts of plaque biofilm with little to no gingival inflammation.
- Loss of periodontal attachment and supporting bone is uncommon in ages 5 to 11 years but may increase in children ages 12 to 17 years. This may occur as the oral biofilm gradually matures, with elevated levels of orange and red complex microorganisms as an individual ages.[12] However, it is important to bear in mind that if the child or adolescent can practice and maintain good oral hygiene, then the risk of either gingivitis or periodontitis is low.
- The presence of severe attachment loss on multiple teeth among children and young adults has been reported to be approximately 0.2% to 0.5%.
- Children and adolescents should receive periodic periodontal evaluation as a component of routine dental visits. Both the American Academy of Periodontology and the American Academy of Pediatric Dentistry strongly recommend that all patients, regardless of age, receive a comprehensive periodontal evaluation as part of routine dental visits.

Eruption Gingivitis and Pericoronal Abscess

1. **Eruption Gingivitis. Eruption gingivitis** is a short-lived type of gingivitis observed in young children as the primary teeth are erupting. Additionally, eruption gingivitis can occur as the primary teeth are exfoliating (shedding) and as the permanent successors emerge.

a. **Characteristics of Eruption Gingivitis**
   1. Tooth eruption, by itself, is not a cause of gingivitis. Rather, this type of gingivitis is caused by plaque biofilm accumulation that occurs in areas of primary teeth shedding or permanent teeth erupting (**Figure 35-3**). The inflammatory response is further accentuated by the fact that the patient is unable to perform adequate oral hygiene in the eruption area because it is too painful or difficult to access.

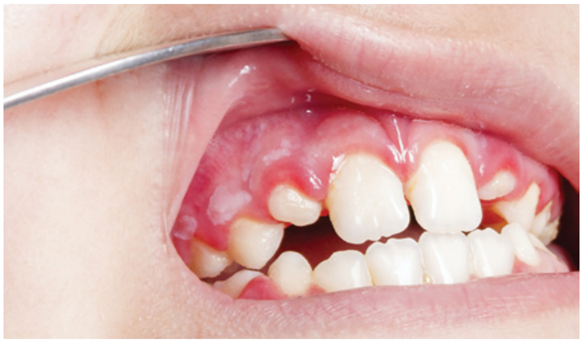

Figure 35-3 **Eruption Gingivitis.** Inflammation of the gingival margin is clinically evident around the erupting teeth of this child.

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   2. Also, as a normal part of exfoliation, the junctional epithelium migrates under the resorbing tooth, thereby increasing pocket depth and creating a niche for further bacteria plaque, food debris, and materia alba collection.[13]

b. **Treatment.** Eruption gingivitis is quickly reversible and subsides after the tooth has completely emerged into the oral cavity. It can also be managed with improved oral hygiene.

c. A first and/or second permanent molar that is in the process of tooth eruption may be associated with severe gingival inflammation which can develop into a pericoronal abscess.

2. **Pericoronal Abscess (Pericoronitis)**
   a. **Disease Characteristics of Pericoronal Abscess (Pericoronitis)**
      1. **Pericoronitis** is a localized purulent infection within the soft tissue surrounding the crown of a partially erupted tooth or a tooth that is in the process of tooth eruption.
      2. As mentioned above, this condition is most frequently seen around erupting permanent molars (**Figure 35-4**).

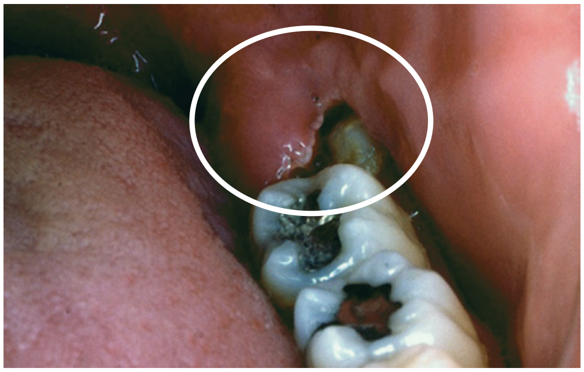

Figure 35-4 **Pericoronitis.** An adolescent patient with pericoronitis. Note the fold of soft tissue—the operculum—partially covering the occlusal surface of the third molar. The area is too painful for the patient to properly clean. This allows for further plaque accumulation which accentuates the inflammation seen around the operculum. The patient may also be experiencing pain whenever he occludes on it.

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   3. The fold of tissue partially covering the erupting tooth is termed an **operculum**. This enlarged tissue fold acts like a partially enclosed lid that covers the occlusal surface of the partially impacted molar (or tooth that is erupting) and collects and retains bacteria, food debris, and materia alba beneath the operculum. This leads to pericoronitis, which literally means "inflammation around the crown of a tooth."

b. **Clinical Manifestations of Pericoronal Abscess (Pericoronitis)**
   1. Some of the clinical signs and symptoms associated with pericoronitis are bad taste (from the pus that forms underneath the operculum), bad smell (halitosis), and tenderness and redness of the gingiva. The operculum can also be painful if the patient occludes on it.
   2. Over time, if acute pericoronitis is left untreated, there is the possibility that it can lead to more serious consequences, especially if the infection does not resolve and spreads to cause trismus (difficulty in jaw opening), dysphagia (difficulty in swallowing), or dyspnea (difficulty in breathing, especially if the infection spreads to the floor of the mouth and results in Ludwig angina).

c. **Treatment of Pericoronal Abscess (Pericoronitis)**
   1. The treatment for this condition is typically irrigation of the tissue with gentle debridement or surgical excision of the operculum. Depending on the severity of the case, antibiotic therapy may be required, especially in cases of lymph node involvement, fever, and malaise. Extraction of the involved molar tooth may also be a viable option.
   2. Pain may be managed with the use of over-the-counter acetaminophen and/or ibuprofen.

Plaque-Induced Gingival Disease

1. **Characteristics of Plaque-Induced Gingivitis in Children and Adolescents. Plaque-induced gingivitis** is characterized by the presence of inflammation limited to the marginal gingiva without detectable loss of clinical attachment or alveolar bone (**Figures 35-5** and **35-6**).

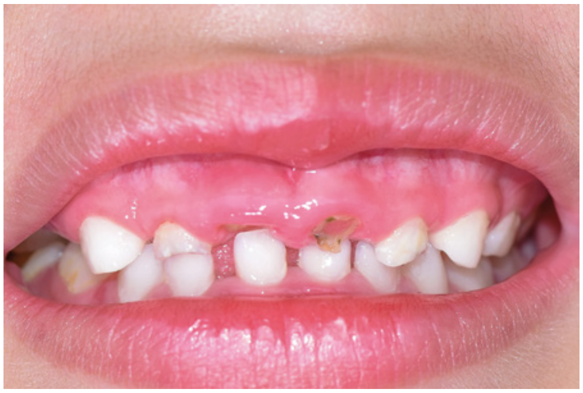

Figure 35-5 Inadequate Self-Care. At his first dental appointment, this young child exhibits plaque-induced gingivitis. Note the accumulation of plaque biofilm at the gingival margins and the presence of exfoliating teeth interfering with self-care efforts.

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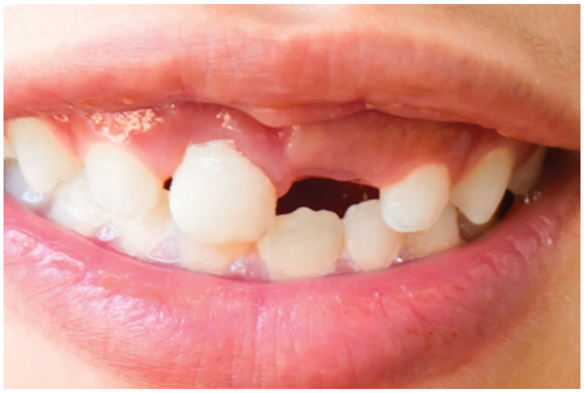

Figure 35-6 Plaque-Induced Gingivitis. This child exhibits gingival erythema and edema associated with moderate plaque biofilm accumulation around the central incisor tooth.

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   a. Gingivitis is extremely common among children and adolescents. As children age, their tendency to develop gingivitis increases.[13] The prevalence is lowest during the preschool years and peaks during puberty. Approximately 60% of teenagers exhibit gingival bleeding on probing.[11]
   b. The degree of gingival inflammation generally is less intense than what would be expected in an adult with a similar amount of plaque biofilm formation.[7,13] The junctional epithelium of the primary dentition tends to be thicker than in the permanent dentition, potentially reducing the permeability of the gingival tissues to bacteria that initiate the inflammatory response.[13]

c. The incidence of gingivitis peaks at ages 9 to 14 years of age and then decreases slightly after puberty.[13] Such gingivitis often is termed **puberty gingivitis** (**Figure 35-7**).

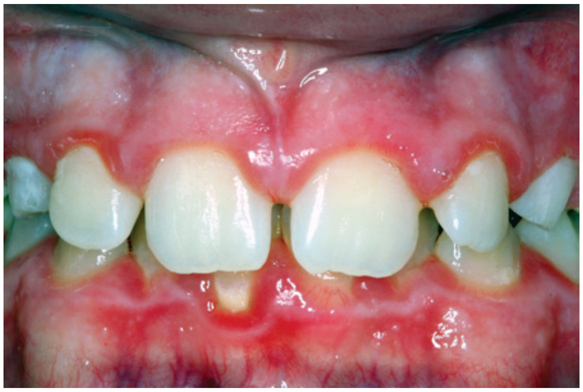

Figure 35-7 Puberty Gingivitis. Puberty gingivitis is an exaggerated inflammatory response of the gingiva to a relatively small amount of plaque biofilm. The exaggerated response is modulated by hormones released during puberty.

Courtesy of Dr. Richard J. Foster, Educational Solutions and Design, Greensboro, NC.

d. Puberty gingivitis is believed to be related to hormonal changes during this developmental stage that intensify the inflammatory response to plaque biofilm.[7,13]

2. **Treatment for Plaque-Induced Gingivitis in Children**
   a. Early gingivitis is quickly reversible with professional care and daily plaque biofilm control.
   b. Professional care may involve removal of plaque biofilm (**Figure 35-8**). About 9% of 4- to 6-year-old children exhibit calculus deposits. By age 7 to 9 years, 18% of children present with calculus deposits, and by the age of 10 to 15 years, 33% to 43% have some calculus formation.[14]

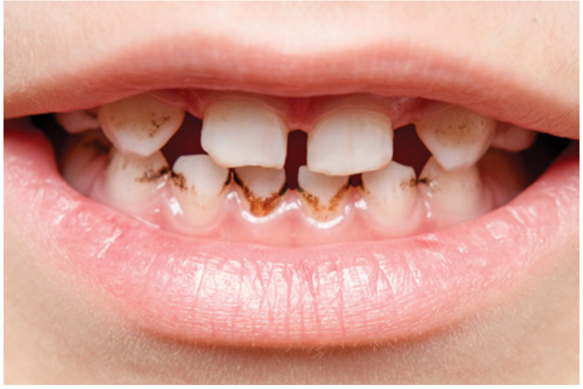

Figure 35-8 Dental Calculus. This young patient exhibits heavy tooth staining and dental calculus.

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Gingival Diseases Modified by Medications

Due to systemic disease, it is necessary for some pediatric patients to take systemic medications that have unwanted side effects on the periodontium. One of the most common medication side effects manifested in the periodontal tissues of pediatric population is gingival overgrowth.

With respect to gingival diseases modified by medications, note that there are two separate and distinct conditions that sound similar but are not the same. **Drug-influenced gingivitis** results from any systemic medication that causes an exaggerated inflammatory response to plaque biofilm. Oral contraceptives are an example of a drug class that triggers an exaggerated inflammatory response to plaque biofilm; the gingival inflammation that is seen in drug-influenced gingivitis is similar to that seen in pregnancy gingivitis or puberty gingivitis. In contrast, **drug-influenced gingival enlargement** is an increase in size (overgrowth) of the gingiva resulting from systemic medications, most commonly anticonvulsants, calcium channel blockers, and immunosuppressants. Unlike inflammatory gingivitis and drug-influenced gingivitis, the distinguishing clinical feature of drug-influenced gingival enlargement is that the gingival tissues do not bleed easily and typically appear firm, resilient, and pale pink in color when uncomplicated by inflammation. **Figure 35-9** shows an example of a child with phenytoin-influenced gingival enlargement. Plaque biofilm accumulation is not necessary for the initiation of gingival enlargement, but it will exacerbate the gingival disease. Meticulous plaque biofilm control can reduce but will not eliminate gingival overgrowth so long as the patient remains on the medication.

1. **Characteristics of Drug-Influenced Gingival Enlargement**
    a. The clinical manifestations of drug-influenced gingival enlargement frequently appear within 1 to 3 months after initiation of treatment with the associated medications.
    b. Gingival overgrowth first typically affects the facial interdental papillae of anterior teeth and progresses to affect the marginal lingual papilla. The interdental papillae overgrow, forming firm triangular tissue masses that protrude from the interdental area.
    c. Gradually, the enlarged papilla from one interdental area may unite with the adjacent enlarged papilla to partially cover the anatomical crown with marginal gingiva (Figure 35-9). Overgrowths are most commonly seen on the facial aspect of the maxillary and mandibular anterior teeth. The gingival overgrowth may serve as plaque-retentive areas which will lead to secondary inflammation.

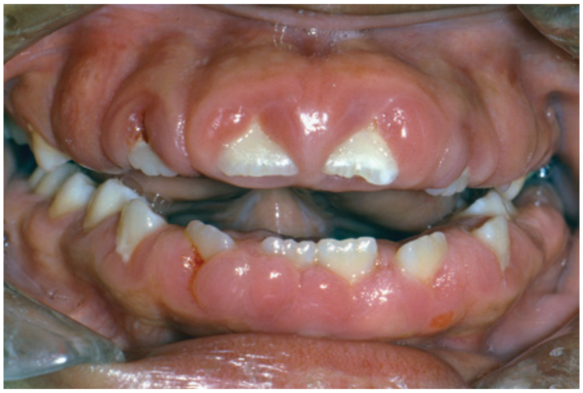

Figure 35-9 Phenytoin-Influenced Gingival Enlargement. Severe enlargement of the gingiva associated with phenytoin (Dilantin) medication in an individual with epilepsy.

Courtesy of Dr. Ralph Arnold, San Antonio, TX.

    d. Ultimately, the enlargement may transform into a massive tissue fold covering a considerable portion of the crowns.
    e. In severe cases, the fibrotic enlargement normally may extend coronally and interfere with esthetics, mastication, or speech. The tissue may completely cover the crowns of the teeth making it more difficult to keep the teeth clean and increase the patient's susceptibility to oral diseases, such as caries and periodontal disease.
    f. In the presence of good biofilm control, the enlarged tissue is pink in color and firm and rubbery in consistency. In the presence of poor biofilm control, the tissue appears red, edematous, and spongy.
2. **Treatment for Drug-Influenced Gingival Enlargement in Children and Adolescents**
    a. Close collaboration between medical and dental clinical teams is necessary for the joint management of individuals being treated with anticonvulsants, calcium channel blockers, or immunosuppressants.
    b. Medication-influenced gingival enlargement may resolve either partially or completely when the medication is discontinued. However, if the medication cannot be discontinued, a gingivectomy for surgical elimination of the tissue overgrowth may be required. If plaque biofilm control is inadequate, the

re-growth will occur rapidly. The patient should be advised of the likelihood of the recurrence of the gingival overgrowth following surgery.

c. As this condition is somewhat worsened by the level of biofilm accumulation on the teeth, effective oral hygiene measures should be implemented to reduce the severity.

d. Thus, it is important that the dental clinician continually reinforce the importance of stringent home care to the patient with drug-influenced gingival enlargement. Gingival diseases modified by medications are discussed in Chapters 5 and 14.

Section 3

Common Types of Periodontitis in Pediatric Patients

Periodontitis

As already discussed, periodontitis is far less common in children and adolescents than in adults.[15] Nevertheless, although it is rare to see this condition in children and adolescents, periodontitis can indeed occur in this subset of the population.[15] When periodontitis occurs in pediatric patients, the disease has similar clinical features, etiology, and pathophysiology to the adult version of the disease. However, the age of onset, location of the periodontal destruction, and the rapidity of the periodontal breakdown are unique features of periodontitis affecting the pediatric patient. **Figure 35-10** is a clinical photograph of a pediatric patient with periodontitis.

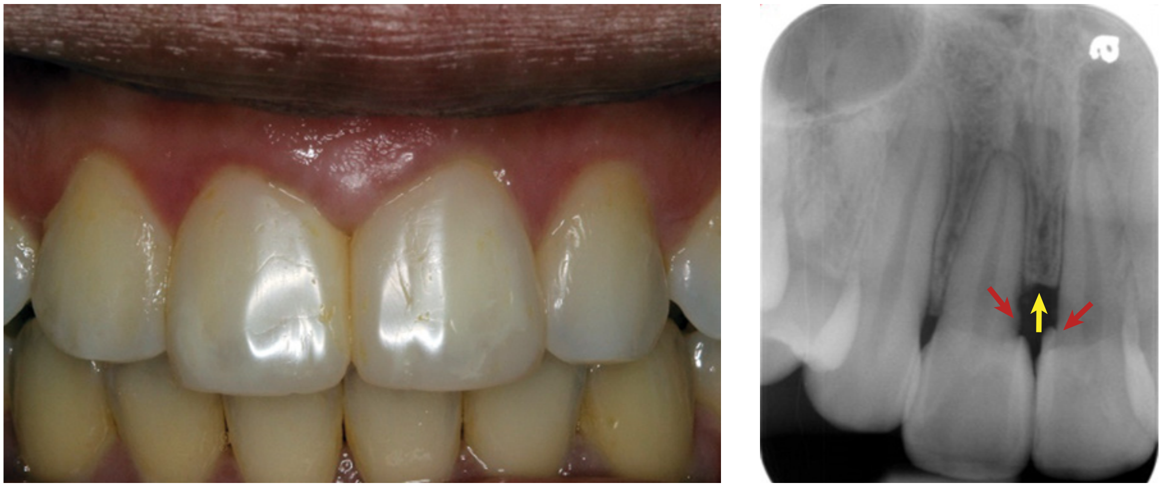

Figure 35-10 Periodontitis. 14-year-old adolescent with periodontitis localized between the central incisors. Observe the edematous gingival margin and papilla between the two teeth. Accompanying radiographic image reveals bone loss (yellow arrow) between the central incisors along with calculus deposits (red arrows).

Case courtesy of Dr. Nicole McKee, Indianapolis, IN.

More Aggressive Forms of Periodontitis in the Pediatric Population

1. **Characteristics of More Aggressive Forms of Periodontitis in Children/Adolescents.** The more aggressive forms of periodontitis seen in children and adolescents (**Figure 35-11**) has several well-recognized primary and secondary clinical features.[15]
   a. **Primary Features.** The primary features of the more aggressive forms of periodontitis in the pediatric population are:[16–18]
      1. Rapid destruction of periodontal ligament and supporting alveolar bone. Baer estimates that the loss of attachment—in what was previously known as localized juvenile periodontitis, then localized aggressive periodontitis—progresses three or four times faster than in periodontitis affecting the adult population.[19]
      2. No obvious signs or symptoms of systemic disease.[20]
      3. Other close family members (parents, siblings) also exhibiting this form of periodontitis.[21–23] **Familial aggregation** refers to the occurrence of more cases of the disease in close family members than can be readily accounted for by mere chance.

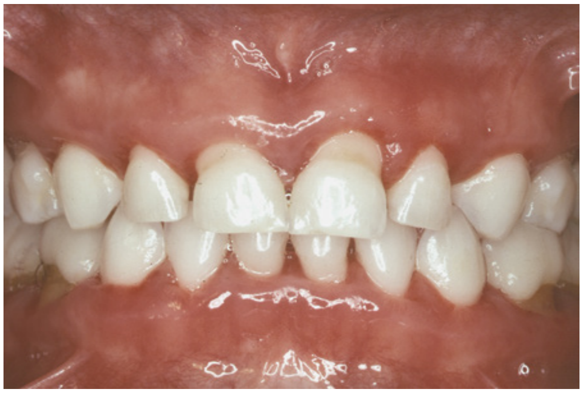

Figure 35-11 Child with a More Aggressive Form of Periodontitis. Periodontitis in a 5-year-old child with attachment loss on all teeth. Note the minimal amount of plaque buildup that is not consistent with the degree and severity of attachment loss.

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- b. **Secondary Features.** Secondary features that are generally but not always present are:
  1. Relatively small amounts of bacterial plaque biofilm; the disease severity seems to be exaggerated given the light amount of plaque biofilm.[16,24]
  2. Elevated proportions of *Aggregatibacter actinomycetemcomitans* (Aa).[21,25]
  3. Impaired phagocytosis.[26–28]
  4. Hyperinflammatory response to bacterial endotoxins (bacterial lipopolysaccharide).[29,30]
  5. A lack of clinical signs of disease.[17,31]
     - a. Affected tissue may have a normal clinical appearance with no signs of inflammation. Calculus deposits are minimal.
     - b. Probing reveals deep periodontal pockets on affected teeth.[32]
  6. A poor response to periodontal therapy.
  7. **Episodic Disease Progression**
     - a. Periodontitis in the adult population is a very slowly progressing disease.
     - b. In more aggressive forms of periodontitis, attachment loss is **episodic**, occurring in a succession of acute destructive phases with intermittent inactive phases.[17]

2. **Two Patterns of the More Aggressive Forms of Periodontitis Affecting the Pediatric Patient**
   - a. **Molar/Incisor Pattern.** The **molar/incisor pattern** of periodontitis (previously called localized juvenile periodontitis and localized aggressive periodontitis in prior disease classification systems) is characterized by localized attachment loss affecting the first molars and/or incisors and involving no more than two teeth other than first molars and incisors.[17,19,33–36] It has been generally accepted that this type of periodontitis can affect both the primary and permanent dentition. In fact, a child affected with the molar/incisor pattern of periodontitis may experience severe periodontitis of the permanent dentition later in his or her life.[37]
     1. **Features of the Molar/Incisor Pattern of Periodontitis**
        - a. In children, the onset of the molar/incisor pattern of periodontitis can occur in the primary dentition as early as age 4. One early indication is radiographic evidence of bone loss around the primary molars and/or incisors. In young individuals, the onset of this form of periodontitis may occur around the time of puberty. However, based on the amount of periodontal destruction seen at the time of detection, it is assumed that the disease process may have begun earlier.[38]
        - b. There is minor tissue inflammation and minimal amounts of plaque biofilm that seem inconsistent with the amount of periodontal destruction.[17,31]
        - c. The molar/incisor form frequently is associated with *Aggregatibacter actinomycetemcomitans* (Aa) in combination with *Porphyromonas* spp.[25,39,40] Yet, to date, no single microorganism has been found to be the causative agent responsible for this form of periodontitis (**Figure 35-12AB**).

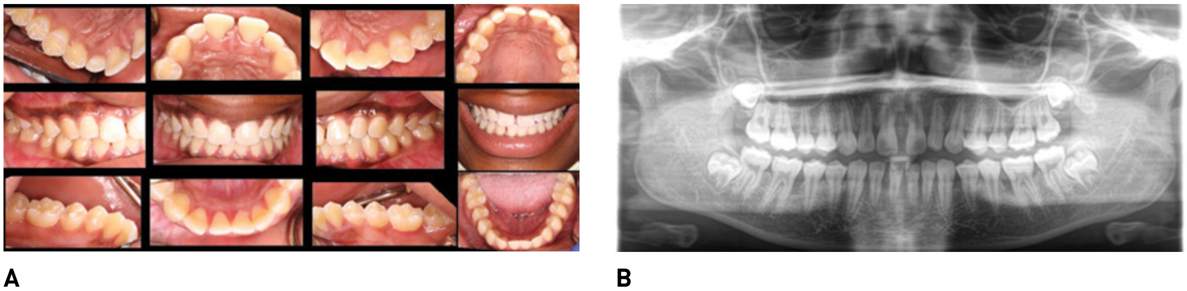

Figure 35-12AB **Molar-Incisor Pattern of Periodontitis. A.** A systemically healthy 17-year-old adolescent presents with molar-incisor pattern of periodontitis. From the clinical photographs, the gingiva exhibits minimal tissue inflammation with scant plaque biofilm accumulation. **B.** However, from the accompanying radiograph, note the localized periodontal destruction affecting the first molars, maxillary incisors, and mandibular incisors.

Case courtesy of Dr. Nicole McKee, Indianapolis, IN.

   d. Vertical bone loss around the first molars and incisors is a classic radiographic sign of the molar/incisor form of periodontitis.[41]
   e. Functional defects of neutrophils are suspected as a systemic contributing factor that makes certain patients more susceptible to this form of periodontitis.[42]
2. **Prevalence of the Molar/Incisor Pattern of Periodontitis.** Less than 1% of the population is affected with this condition. This form of disease may first affect the deciduous dentition but then progress to affect the permanent dentition as the pediatric patient ages, if the disease is left untreated.
3. **Screening of Primary and Mixed Dentitions for the Molar/Incisor Pattern of Periodontitis**
   a. The measurement of attachment loss on primary teeth or partially erupted teeth may be difficult.
   b. Measurement of the distance between the cementoenamel junction (CEJ) and the alveolar bone crest on bitewing radiographs is a useful screening approach with children (**Figure 35-13**).

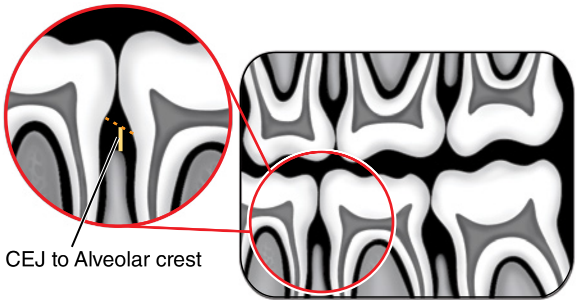

Figure 35-13 **Use of Bitewing Radiographs in Screening for the Molar/Incisor Pattern of Periodontitis.** The distance from the CEJ and the alveolar bone crest is measured from a line connecting the CEJs of the two adjacent teeth. Measurements are taken for each mesial and distal surface. Normal CEJ-to-alveolar bone crest distances for 7- to 9-year-old children are less than 2 mm.

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   c. The "normal" distance between the CEJ and the alveolar bone crest has been evaluated by recent investigations.[31,43]
      1. The median distance between the CEJ and the alveolar crest of primary molars in 7- to 9-year-old children is 0.8 to 1.4 mm. The CEJ of permanent molars is 0 to 0.5 mm coronal to the alveolar crest in 7- to 9-year-old children.
      2. Greater distances between the CEJ and alveolar crest are seen at sites with caries, restorations, or open contacts. These local intraoral factors may contribute to localized bone loss in children in a similar manner to that seen in adults and are not necessarily indicative of aggressive periodontitis.

3. A distance of more than 2 mm between the CEJ and alveolar crest, in the absence of local contributing factors, should cause the clinician to suspect periodontitis. If the measurement exceeds this value, periodontitis should be suspected, and a comprehensive periodontal examination should be performed.

4. **Treatment of the Molar/Incisor Pattern of Periodontitis**
   a. The successful treatment includes early diagnosis and surgical or nonsurgical periodontal therapy in combination with systemic antibiotic therapy.[11,44]
   b. According to a number of studies, the most successful antibiotics in the treatment of the molar/incisor pattern of periodontitis are broad-spectrum antibiotics, such as azithromycin alone or a combination regimen of amoxicillin and metronidazole.[45,46] However, administering tetracycline to a child should be prescribed with caution as there is the possibility that the antibiotic may discolor and stain the developing dentition. Furthermore, most periodontal pathogens have developed resistance to tetracycline due to the previous overuse of the antibiotic.
   c. Because there may be an underlying systemic condition that makes the child more susceptible to the disease, medical consultation with the child's pediatrician is recommended to coordinate medical care with periodontal therapy.
   d. If the primary teeth are affected, then the child must be diligently monitored at subsequent recall visits for the possibility of this form of periodontitis affecting his/her permanent dentition.
   e. More frequent recalls and dental checkups for pediatric patient with this pattern of periodontitis may be recommended to provide maintenance care, reinforce the importance of a good oral hygiene regimen, verify that the disease is arrested, or intercept the disease reoccurrence at its early stages.

b. **Generalized Pattern of More Aggressive Forms of Periodontitis.** The **generalized pattern** of the more aggressive forms of periodontitis is characterized by generalized interproximal attachment loss affecting at least three permanent teeth other than the first molars and incisors. Most of the permanent teeth usually are affected in this form of periodontitis (**Figure 35-14**).[17,35]

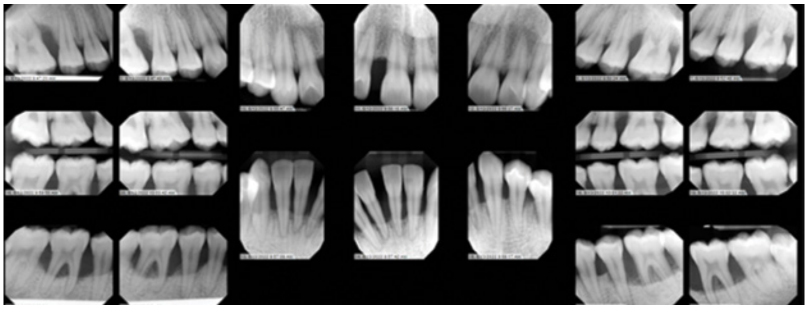

Figure 35-14 Generalized Pattern of Periodontitis. A systemically healthy 16-year-old adolescent presents with generalized pattern of periodontitis. Family history reveals that her father, grandfather, and paternal uncles lost teeth due to periodontitis. Note the extensive bone loss that has occurred at such a young age. Additionally, note the periodontal destruction affecting at least three permanent teeth other than the first molars and incisors- namely, the mandibular second molars, mandibular premolars, and mandibular canines.

1. **Features of the Generalized Pattern of More Aggressive Forms of Periodontitis**
   a. Usually occurs in persons younger than 30 years of age, but patients may be older.[17,47]
   b. Destruction of attachment and alveolar bone is very episodic, occurring in a succession of acute phases rather than in a gradual progression.[17]
   c. The appearance of the gingival tissues varies in this type of periodontitis.
      1. The gingival tissues may be acutely inflamed, ulcerated, and fiery red.[17,47] This tissue response is believed to occur in the destructive phase of disease progression.
      2. The gingival tissues may appear pink and free of inflammation. However, deep pockets can still be detected with conventional periodontal probing. This tissue response may coincide with periods of disease inactivity.[48]

2. **Treatment of the Generalized Pattern of More Aggressive Forms of Periodontitis.** In general, the treatment regimen for the generalized pattern of more aggressive forms of periodontitis is similar to that used to treat the molar/incisor pattern of periodontitis.
    a. Medical evaluation of the patient is warranted to determine if any underlying undiagnosed systemic conditions are putting the patient at higher risk of having a refractory or recurrent form of this type of periodontitis.
    b. The generalized pattern does not always respond well to conventional nonsurgical periodontal therapy or to antibiotics commonly used to treat periodontitis.[49–51]
    c. Microbiological assaying may be considered to identify the specific putative pathogen and determine the specific type of antibiotic to prescribe.[46]

Necrotizing Periodontal Diseases

Necrotizing periodontal diseases occur with low frequency (less than 1%) in North American and European children.[11] Factors that predispose children to necrotizing periodontal diseases include viral infections (including HIV), malnutrition, emotional stress, lack of sleep, and a variety of systemic diseases.[11] Necrotizing periodontal diseases are discussed in Chapter 9.

Section 4

Common Acute Periodontal Conditions in Pediatric Patients

Herpes Virus Infections and Aphthous Ulcers

1. **Primary Herpetic Gingivostomatitis. Primary herpetic gingivostomatitis** is a severe reaction to the initial viral infection with—first exposure of an individual to the Herpes Simplex Virus Type-I (HSV-1).
    a. **Disease Characteristics**
        1. According to the World Health Organization, approximately 3.7 billion people younger than age 50 are infected with HSV-1.
            a. In most cases, the virus never causes symptoms during the primary HSV-1 infection. This is known as a subclinical (i.e., symptom-free) infection.
            b. In some individuals, however, this initial infection presents with intensely painful gingival inflammation and multiple vesicles that easily rupture to form painful ulcers. This severe reaction to the initial HSV-1 infection is known as primary herpetic gingivostomatitis (**Figures 35-15** and **35-16**).

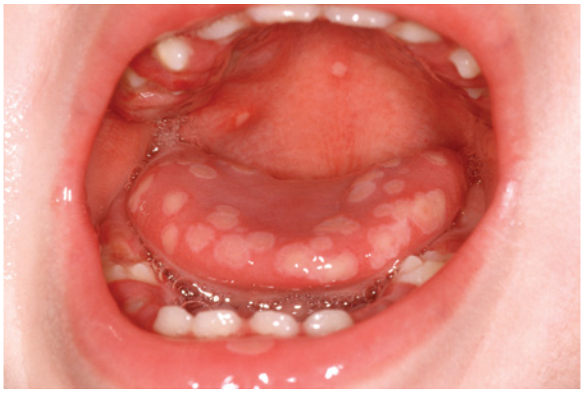

Figure 35-15 Primary Herpetic Gingivostomatitis. This clinical photograph shows a young child with oral lesions associated with primary herpetic gingivostomatitis. Note the oral ulcerations surrounded by red halos.

© Mediscan/Alamy Stock Photo

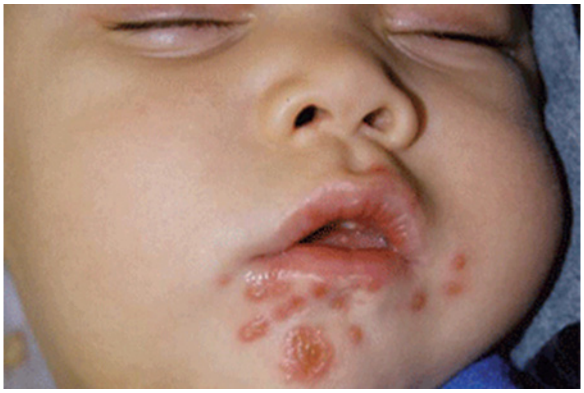

Figure 35-16 Facial Lesions of Primary Herpetic Gingivostomatitis. Oral lesions of primary herpetic gingivostomatitis can occasionally be accompanied by skin lesions. This clinical photograph shows an infant with primary herpetic gingivostomatitis with vesicle formation on the face.

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    c. Once infected, most individuals develop immunity to the virus. In certain individuals, HSV-1 can remain latent in the trigeminal ganglion and cause recurrent oral herpetic lesions (cold sores).
  2. Initial infection with HSV-1 usually occurs in young children, with heightened incidence from 1 to 3 years of age. However, initial infection may also occur in adolescents and adults.
    a. Of children with primary infections, 99% are symptom free or the symptoms are attributed to teething.
    b. The remaining 1% develops significant gingival inflammation and multiple painful ulcerative lesions found on keratinized tissue, such as the lips, gingiva, hard palate, and dorsal side (top) of the tongue.[52]
  3. The infection is contagious during the vesicular stage because the virus is contained in the clear fluid in the vesicles. The virus may easily spread through transmission of viral-infected saliva from one part of the body to another in the same individual (i.e., perioral involvement or herpetic whitlow) or from one infected individual to another individual.
  4. Primary herpetic gingivostomatitis is associated with severe pain that makes eating and drinking difficult. This may result in nutritional deficiencies during the active stage of the disease.
  5. Systemic symptoms associated with primary herpetic gingivostomatitis are headache, swollen lymph nodes, and sore throat (**Figure 35-17**). Because this condition is a viral infection, there may be a low-grade fever, usually not above 101°F (38.3°C).

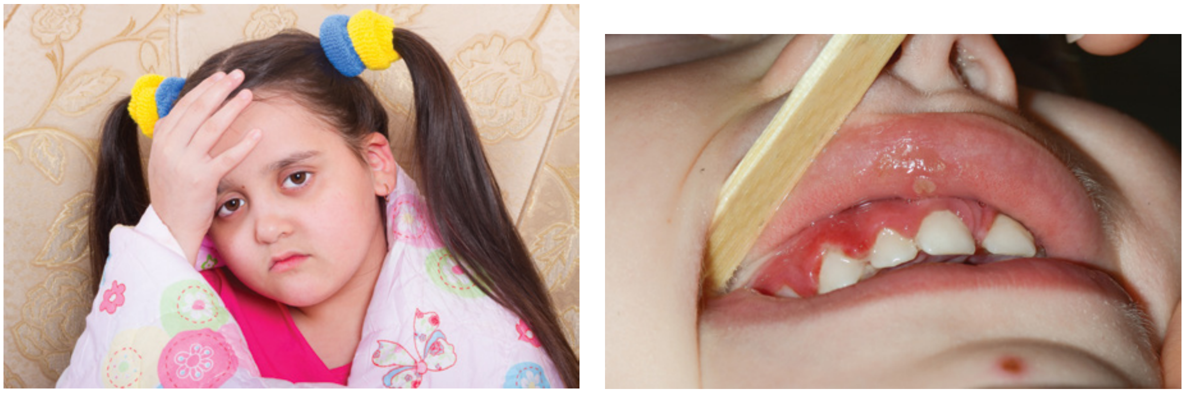

Figure 35-17 Symptoms of Primary Herpetic Gingivostomatitis. This child demonstrates common symptoms of primary herpetic gingivostomatitis, including fever and multiple ulcerative lesions of the oral cavity. Other common systemic symptoms associated with primary herpetic gingivostomatitis are lymphadenopathy, sore throat, and general malaise.

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  6. Primary herpetic gingivostomatitis regresses spontaneously within 10 to 20 days without scarring.

b. **Clinical Manifestations of Primary Herpetic Gingivostomatitis.** Primary herpetic gingivostomatitis may occur anywhere on the free or the attached gingiva.
  1. Primary herpetic gingivostomatitis is characterized by widespread inflammation of the marginal and attached gingiva.
  2. The gingiva exhibits intense gingival inflammation.
  3. Small clusters of yellowish-white liquid-filled vesicles rapidly form throughout the mouth.
  4. Later, these vesicles burst, forming yellowish ulcers that are surrounded by a red halo.
  5. Systemic involvement, such as headache, fever, swollen cervical and submandibular lymph nodes (lymphadenopathy), and sore throat, usually is present.

c. **Treatment for Primary Herpetic Gingivostomatitis**
  1. If possible, treat an outbreak within the first 48 to 72 hours with a systemic antiviral medication such as acyclovir, valacyclovir, or famciclovir.
  2. Counsel the child and the child's caregiver that adequate intake of fluids is important. Because eating and drinking are painful, dehydration is a major concern with these individuals. Athletic drinks, such as Gatorade, can be consumed to replenish electrolytes lost due to dehydration.

3. A dietary replacement drink, such as PediaSure or Ensure, can be a good source of nutrition if eating is difficult. The patient may be able to eat foods processed in a blender. Vitamin supplementation may also be beneficial so as to maintain healthy levels of micronutrients during the course of the disease.
4. Pain may be relieved by acetaminophen, ibuprofen, or viscous lidocaine regimen.
5. An antimicrobial mouthwash such as Listerine or Peridex should be recommended to prevent a secondary infection.
6. Precautions should be taken to prevent the spread of the virus to the patient's eyes or transmission from an infected individual (child) to a noninfected individual (parent). The infected individual should wash with soap and water frequently.
7. Avoid any procedure that results in the airborne spread of viral-infected aerosol (i.e., handpiece, ultrasonic scaler, and air polisher) until the HSV lesion has scabbed over.
8. Avoid any procedure that involves the manipulation of soft tissues infected with HSV (i.e., periodontal probing and periodontal instrumentation).
9. Implement strict infection control procedures to reduce the risk of spreading the virus from an infected patient to a dental health care worker.

2. **Recurrent Oral Herpes Simplex Labialis.** **Recurrent herpes simplex labialis**—commonly referred to as a fever blister or cold sore—is an infection of the mouth area caused by the reactivation of Herpes Simplex Virus Type-I (HSV-1). It is a common and contagious infection that spreads easily. According to the American Sexual Health Association, over half of adults in the United States carry this virus.
   a. **Characteristics of Recurrent Oral Herpes Simplex Labialis**
      1. This lesion can occur in patients of all ages, including children. After the primary infection, the virus lays dormant inside the nerve cells of the face for the rest of a person's life. This means that symptoms are not always present. However, certain events can make the virus reawaken and lead to a recurrent herpes infection.
      2. Events that trigger a recurrent infection of oral herpes might include excessive exposure to sunlight, physical/emotional stress, lack of sleep, hormonal changes, fever, a weakened immune system, lowered tissue resistance resulting from various types of trauma, or recent dental treatment.

   b. **Clinical Manifestations of Recurrent Oral Herpes.** The majority of recurrent oral herpes occurs on the vermillion border of the lip, hence the condition also being referred to as recurrent herpes labialis, whereas only 5% of recurrent oral herpes occurs in intraoral locations, such as the nonmovable mucosa (keratinized tissue) of the hard palate and/or the attached gingiva (**Figure 35-18**). Like primary herpetic gingivostomatitis, these lesions may spread to perioral areas, such as the nose, cheek, and chin. Although recurrent oral herpes is milder than the primary attack, it can be dangerous if the infection spreads to the eyes and causes blisters or sores to form close to the cornea. If such a situation arises, immediate consultation with an ophthalmologist is warranted.

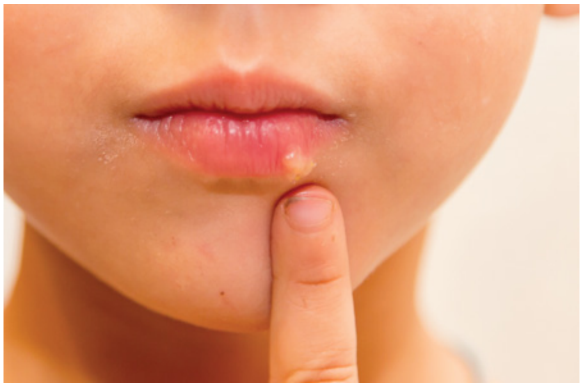

Figure 35-18 **Recurrent Oral Herpes.** Child with a recurrent oral herpes blister on her lower lip. The fluid-filled blister will eventually rupture and leave superficial ulcers that crust and heal without scaring within 2 weeks.

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1. Tingling on or near the lips is usually a warning sign that the cold sores of recurrent oral herpes lesions are about to appear in 1 to 2 days.
2. Symptoms of a recurrent episode may include:
    a. Blisters or sores on the mouth, lips, tongue, nose, or gingiva.
    b. Burning pain around the blisters.
    c. Tingling or itching near the lips.
    d. Outbreaks of several small blisters that grow together and may be red and inflamed.

c. **Treatment for Recurrent Oral Herpes**

1. Recurrent oral herpes has a spontaneous resolution in 10 to 14 days; therefore, it is managed by palliative care.
2. Systemic antivirals (such as acyclovir, valacyclovir, and famciclovir) can be prescribed to treat an outbreak within the first 48-72 hours of onset.
3. Over-the-counter topical medicaments, such as Abreva, can be used to treat outbreaks that occur on the lip when applied at the first sign of outbreak.
4. Applying a whole-milk compress to an erupted cold sore has been a natural home remedy for over a century to mitigate the spread of the virus, speed up the healing process, and ease the pain. While the exact reason is unknown, it is believed that the L-lysine and immunoglobulins in whole milk help the body fight off the virus.
5. Parents should be cautioned to monitor the child to prevent him or her from touching ulcerations and spreading the contagious virus to other mucous membranes or to other individuals. Educating the child and the parent of the importance of meticulous hygiene is critical in minimizing disease transmission.

3. **Recurrent Aphthous Ulcers (also known as recurrent aphthous stomatitis)**

a. **Disease Characteristics.** **Recurrent aphthous ulcers** are reoccuring, painful ulcers of the oral mucosa that occur in school-aged children and adults. This condition also is referred to as recurrent aphthous stomatitis.

1. Aphthous ulcers, or "canker sores," are small recurrent, painful, round or ovoid ulcers with well-defined erythematous margins, like a halo, and a central yellow or gray floor that typically develop on nonkeratinized tissue such as the buccal mucosa, the mucobuccal fold, the floor of the mouth, the ventral side (bottom) of the tongue, and on the soft palate (**Figure 35-19**). Unlike oral herpetic lesions, aphthous ulcers are not contagious, are not caused by a virus, and generally are not associated with detectable systemic involvement. Moreover, while HSV-1 is characterized as a vesiculoulcerative lesion (meaning that a vesicule stage precedes the ulcerative stage of the lesion), aphthous ulcers do not have a preceding vesicle stage.

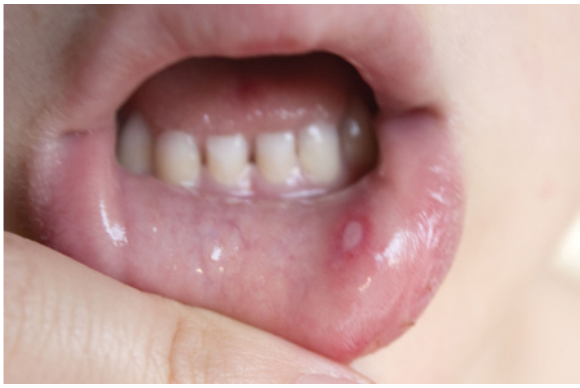

Figure 35-19 **Aphthous Ulcer.** A young child with an aphthous ulcer on the mucosal lining of the lip. Although it clinically appears to look like an intraoral herpetic lesion, its location on nonkeratinized mucosa (inner lip) is typical of an aphthous ulcer.

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    2. The precise cause of aphthous remains unclear, though researchers suspect that a combination of local factors (such as trauma, allergy to toothpaste constituents, or salivary gland dysfunction) and systemic factors (such as autoimmune dysfunction, bacterial or viral infection, or nutritional deficiencies) contribute to outbreaks, even in the same person.
    3. The peak age is between 10 and 19 years of age.

b. **Aphthous Ulcer Triggers.** Possible triggers for recurrent aphthous ulcers include:
    1. A minor injury to the oral cavity from dental work, overzealous brushing, sports mishaps or an accidental cheek bite.
    2. Toothpastes and mouth rinses containing sodium lauryl sulfate.
    3. Food sensitivities, particularly to chocolate, coffee, strawberries, eggs, nuts, cheese, and spicy or acidic foods.
    4. A diet lacking in vitamin B12, zinc, folate (folic acid) or iron.
    5. Emotional stress.

c. **Treatment of Aphthous Ulcers**
    1. Ulcers usually heal spontaneously within 4 to 14 days.
    2. Treatment focuses on promoting ulcer healing, reducing ulcer pain, and maintaining the patient's nutritional intake.

Chapter Summary Statement

The comprehensive 2021 NIH *Oral Health in America: Advances and Challenges* report is a powerful reminder of the extensive toll oral, dental, and craniofacial disorders/diseases continue to have on the American (and global) population, in spite of the many advances made in medicine and dentistry. One subset of our population that the report highlights as being vulnerable is children. Children present unique challenges and are at an increased risk for oral infections, delays in tooth eruption, periodontal disease, enamel irregularities, and malocclusion. Their exposure to certain medications and therapies, special diets, and their difficulty in maintaining daily oral self-care further compromises their oral health. According to estimates highlighted in the report, the ramifications of untreated oral diseases account for serious general health problems, significant pain, interference with eating (which affects growth and development), improper utilization of hospital emergency rooms, and lost school time. One of the most critical themes repeatedly emphasized in the report is that effective prevention requires an early start.

To meet this challenge head on, oral health care providers, especially dental hygienists, must be ready to take the lead in educating pediatric patients and their caregivers on multiple oral health issues—dental caries, periodontal health, injury prevention, dental development, oral habits, common soft tissue ulcerative lesions, and bite development—as well as promoting the child's complete oral health by performing a comprehensive periodontal examination at each routine dental visit. In cases where preventative therapy has not been effective in suppressing the disease, early interventional therapy makes a big difference in ensuring the greatest chance for success in preserving the dentition and periodontium while maintaining the normal developmental progression and maturation of the child. Although it is true that the pediatric population has a lower occurrence of destructive periodontal diseases (i.e., periodontitis) compared to the adult population, it is essential that all participants invested in the care of the pediatric patient, including the dental practitioner, the child, and the caregiver, not let their guard down in preserving a healthy lifestyle all throughout childhood and adolescence.

Section 5

Focus on Patients

Clinical Patient Care

CASE 1

A 28-month-old child is brought to your office for an emergency appointment. The parent informs you that her child has had a fever for the past 2 days, has not been eating well, and barely wants to drink fluids. The parent informs you that the child has no significant medical history and no known drug allergies. The child's immunizations are up to date. Examination of the child reveals an elevated temperature; swollen cervical and submandibular lymph nodes; inflammation of the marginal and attached gingiva; small clusters of blisters on the lips, tongue, and buccal mucosa; and some ulcers on oral tissues surrounded by red halos.

1. What might be a likely diagnosis for this child's condition?
2. What kind of therapy would be appropriate for this child?
3. What information about this condition should be given to the parent?

CASE 2

A 13-year-old female presents for a routine preventive visit in your office. The patient tells you that she recently had a painful blister form on her lip, and she indicates with her finger that the blister had been on the vermillion border of her upper lip. She explains that the blister lasted about 2 weeks and disappeared after that 2-week period. She also tells you that she has had painful blisters like this before. Examination reveals that there is no evidence of a lip lesion at the time of this visit and no other oral pathology.

1. What might be a likely diagnosis for this blister?
2. What information should be given to the patient about this condition?

Ethical Dilemma

A mother brings her 8-year-old daughter to your office for an examination. The mother explains that Mary woke up 2 days ago with a slight fever and a headache. She explains that she took her daughter to a pediatrician who prescribed amoxicillin and referred Mary to your dental office. The mother explains that Mary's symptoms are not improved after 48 hours. When you examine Mary's oral tissues, you notice small vesicular lesions on her gingiva and on the dorsal surface of her tongue. Her oropharynx appeared clear of lesions. She has palpable cervical lymph nodes that are a bit tender. Mary's temperature is elevated. The mother is frustrated and asks you to tell her if the pediatrician managed her daughter's care properly.

1. How should you respond to the mother's frustration?
2. How should you respond to her question about the care provided by the pediatrician?

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CHAPTER 36

Iatrosedation: Easing and Managing Pediatric Patient Fears

- Section 1 Dental Anxiety and the Pediatric Dental Patient
- Section 2 Behavioral Assessment of the Pediatric Patient
- Section 3 Iatrosedation Techniques with Pediatric Patients
- Section 4 Focus on Patients
  - Evidence in Action
  - Clinical Patient Care
  - Ethical Dilemma

CLINICAL APPLICATION

"No man stands so tall as when he stoops to help a child." This James Dobson quote helps to frame the special relationship that exists between adults who serve children. Likewise, this quote is appropriate for adults who serve children in a pediatric dental setting. It has been demonstrated in the literature that early oral health experiences serve as an influential and foundational role for patients' decisions and motivations to seek, access, and utilize dental care as adults.[1–3] As a vital member of the dental team, the hygienist is in a key position to promote a positive and productive dental experience for pediatric dental patients. This chapter introduces several topics relevant to a concept of fear/anxiety management known as iatrosedation and behavioral guidance. Additionally, this chapter will provide an outline of practical iatrosedation strategies based on evidence-based guidelines from behavioral psychology, child developmental, and behavioral dentistry principles.

LEARNING OBJECTIVES

- Define *pharmacosedation* and *iatrosedation*.
- Discuss the importance of iatrosedation and behavioral guidance for the pediatric dental patient.
- Describe the role and effect of growth and development in patient behavior and outcomes.
- Discuss the variables that may impact the behavior of pediatric dental patients.
- During role plays or in the clinical setting, demonstrate effective communication with pediatric dental patients and their parents or caregivers.
- Discuss the behavioral guidance strategies for a range of pediatric dental patients representing all stages of growth and development.
- Review techniques for safe and effective application of distraction techniques with pediatric patients.
- Demonstrate how to perform a knee-to-knee oral screening exam for infants and toddlers, gain access to a child's mouth, and restrain a child's body movements during an oral screening exam.

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KEY TERMS

Objective fear
Subjective fear
Pharmacosedation
Iatrosedation
Engagement behaviors
Disengagement behaviors
Nonpharmacological therapy
Frankl Behavioral Rating Scale
Tell–Show–Do
Positive reinforcement
Distraction
Modeling
Systematic desensitization
Pediatric dentistry triad
Knee-to-knee position

Section 1

Dental Anxiety and the Pediatric Dental Patient

Dental fear and anxiety are major contributors to the reluctance of people in North America and across the world to obtain dental services. Most patients are afraid of one of three things at the dental office: fear of loss of control, fear of embarrassment, or fear of pain. The overall effect of dental anxiety appears to be multifaceted, such that the individual not only avoids his or her dental appointments but also tends to have worse oral health. The incidence of dental fear and anxiety appears to be relatively consistent throughout the world, with some subgroups reporting higher levels than others. This chapter addresses the general concept of iatrosedation or how the dental clinician utilizes his or her behaviors to help alleviate pediatric dental patient fears.

1. **Dental Anxiety in Relation to Dental Treatment.** According to the literature, the prevalence of dental fear and anxiety among pediatric patients ranges between 5% and 20%.[4–7] In a recent study assessing the prevalence of dental fear and anxiety among 308 adult patients in various private practice settings, White et al. identified several sources of dental fear and anxiety in adults: (1) fear of dental experience, (2) previous negative dental experience, (3) cost of treatment, (4) gag reflex, and (5) fear of bad news.[8] Although this study examined dental fear and anxiety among an adult cohort, it is possible that these same factors may also play a role in evoking similar responses from a fearful and anxious pediatric patient.
    a. **Causes of Dental Fear in Children.** Children experience dental anxiety just as adults do, and this fear is intensified by a sense of the unknown. They just do not know what to expect during the dental appointment, and that is scary.
        1. It is important for the practitioner to understand where dental anxiety originates in children. There are essentially two types of dental fear: objective and subjective.
            a. **Objective fear** is fear resulting from a direct experience. Objective fear is created when a child has a negative experience in the dental office, such as feeling pain or not understanding precisely what is happening. The next time the child is scheduled for a dental visit, he or she would be fearful due to this prior painful or frightening experience. The only way to overcome objective fear is to return to the dentist and replace these negative experiences with positive ones. Over time, as a child becomes more comfortable with the dental setting and more familiar with the dental practitioner, his her anxiety will subside.
            b. **Subjective fear** is fear that does not spring from a prior experience. This fear may result from images of terrifying dentists on television, playground horror stories from classmates, or by sensing mom's anxiety before her own dental appointment.
        2. McElroy was responsible for the earliest reference to behavior management in the dental literature in 1895.[9] He wrote "Although the operative dentistry may be perfect, the appointment is a failure if the child departs in tears." This was the first time the success of a dental visit for a child was not measured by simply completing the dental treatment at hand.
    b. **Managing Dental Fear.** When helping fearful dental patients cope with anxiety, the two primary techniques used in contemporary dentistry are (1) pharmacosedation and (2) iatrosedation.
        1. **Pharmacosedation** is defined as the act of making calm with the administration of anxiety reduction medications, such as a barbiturate or benzodiazepine.

        a. Managing pediatric dental patients with medication can be quite challenging, even for the most experienced dental clinician. It requires a strong understanding of human anatomy and physiology, a deep familiarity with the pharmacologic mechanisms of action of anxiolytic/sedative agents, and a thorough grasp of current pharmacosedative techniques and technologies.
        b. In some pharmacosedative cases, even a skilled dental practitioner may require the help of an individual who has undergone advanced specialized training in anesthesiology (i.e., a dental anesthesiologist, nurse anesthetist).
    2. **Iatrosedation** was developed and defined by Dr. Nathan Friedman as the act of making calm by the doctor's behavior (*iatro* means "pertaining to a doctor or treatment"; *sedation* means "the act of calming").[2] Behavior in this sense includes a broad spectrum of verbal and nonverbal communication (behavior). *Mosby's Dental Dictionary* defines *iatrosedation* as a relaxed state induced by actions rather than drugs; a method of anxiety reduction that is psychologically based.
        a. Iatrosedation encompasses a range of nonpharmacological techniques in reducing the dental patient's anxiety. **Nonpharmacological therapy** means any therapy that does not include sedative or anxiolytic drugs or medications.
        b. Iatrosedation is a technique for helping anxious patients of any age; however, this technique is particularly useful when treating pediatric and special needs individuals.

2. **Factors That Influence Appointment Outcomes in Pediatric Patients.** The pediatric patient is shaped by his or her environmental influences. These influences include family and external influences, patient influences, and the influence of the clinician and office setting.[1,3,10–15]
    a. **Family and External Influences**
        1. Parents or caregivers who exhibit high anxiety in their daily life or the dental office setting typically can affect the child's behavior negatively. This is especially true of the effect of maternal anxiety (**Figure 36-1**).

Figure 36-1 Parental Anxiety. A child's behavior may be adversely influenced by a parent or caregiver who exhibits high anxiety in the dental setting.

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        2. Parental/caregiver factors such as parental age, health, or education.
        3. Stability of the child's day-to-day life, including emotional stability of parents/caregivers or such factors as the stability of the marital relationship.
        4. Family culture and beliefs. Is health a priority in the family value system?
        5. Parental or caregiver attitudes have the potential to impact the child's demeanor. This influence is based on parenting styles such as overprotective, overindulgence, under affectionate, rejecting, and authoritarian.

b. **Patient Factors**
   1. **Developmental Factors.** In 1959, Dr. Erik Erikson articulated a comprehensive theory that identifies a series of eight stages that a healthy developing individual should pass through from infancy to late adulthood—infant, toddler, preschooler, grade-schooler, teenager, young adult, middle-age adult, older adult.[16] The following is an outline description of Erickson's stages of development from infant to adolescent and the impact in the dental setting.
      a. **Infants—Babies: Birth to age 2**
         1. Children learn to sit, stand, walk, and run.
         2. Infants progress from babbling to using simple sentences.
         3. They can identify familiar faces and progress through periods of being friendly and then fearful of strangers.
         4. At this age, a child is too young to cooperate with dental procedures.
      b. **Toddlers—Preschoolers: Ages 3 to 5 years**
         1. Children develop autonomy (self-confidence and independence) and initiative.
         2. Toddlers thrive in a controlled and structured environment.
         3. They can follow simple instructions.
         4. At this age, children welcome an active role in the dental treatment experience.
      c. **School-Age Children: Ages 6 to 12 years**
         1. School-age children have smooth and strong motor skills with variable physical abilities.
         2. Children develop a sense of body image around age 6.
         3. This is the period of socialization; children are learning to get along with others.
         4. They are learning the rules and regulations of society.
         5. At this stage, children begin to overcome fears of new objects and situations.
      d. **Adolescent—Teenagers: Ages 13 to 18 years**
         1. At this stage in development, the individual should respect the dental team member as a person.
         2. The hygienist should treat adolescents respectfully according to their advanced level of maturity.
         3. If an adolescent is acting out in a negative way, it is likely the result of his or her own self-esteem or self-image issues.
         4. During this period of development, the individual is responsive to direct focus and empathy.
   2. **Patient Temperament.** The temperament of the pediatric patient plays a major role in determining or gauging their appointment outcomes.
      a. **Easy.** These children have a positive attitude, are pleasant, adaptable, and willing to try new things.
      b. **Slow to Warm-up.** Typically, this describes the shy patient. This child is slow to adapt to new things but will become comfortable once he or she gets acquainted with the environment.
      c. **Difficult.** This group of children are challenging dental patients. They have difficulty with routines and sleeping, are disengaging, and are neither accepting nor adaptable to change. They may be awkward in social settings.
   3. **Patient's Medical History.** Patients who have had positive medical experiences in the past are more likely to cooperate during a dental visit. Those who have had medical challenges or negative medical experiences may have a difficult time at the dental office.
   4. **Patient's Awareness of Dental Concern.** A child with an existing dental concern (such as pain, inflammation, or trauma) may have a tendency toward negative behavior because the child is aware there is a problem that needs to be treated.

c. **Dental Team Factors.** Behavioral guidance and management of the child and adolescent patient are necessary to establish a relationship between the patient, parent/caregiver, and the hygienist. Oftentimes, there are aspects of the relationship that are beyond the control of the dental hygienist. Aspects of the relationship that are within the control of the clinician include:
   1. Being mindful of personal and office appearance.
   2. Acknowledging the parent while not allowing a parent/caregiver to interfere with direct communication with the child.
   3. Maintaining interest, concern, humor, and confidence when interacting with the patient and parent/caregiver.

4. Using subtle commands, rather than making suggestions or asking questions.
5. Maintaining a readiness and willingness to be firm.
6. Avoiding fear-promoting words and always explaining before doing.
7. Keeping appointments short and at an optimum time for the patient.

d. **Additional Factors.** Other factors that may influence appointment outcomes are summarized in **Box 36-1**.

Box 36-1 Examples of Additional Patient Factors

Variables that can influence the outcomes for the pediatric dental patient:

- Readiness for learning
- Motivation
- Language development
- Dental anxiety of parents/caregivers and siblings
- Behavioral diagnosis or problems (ADHD, autism)
- Physical handicaps
- Emotional development
- Academically gifted

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Section 2

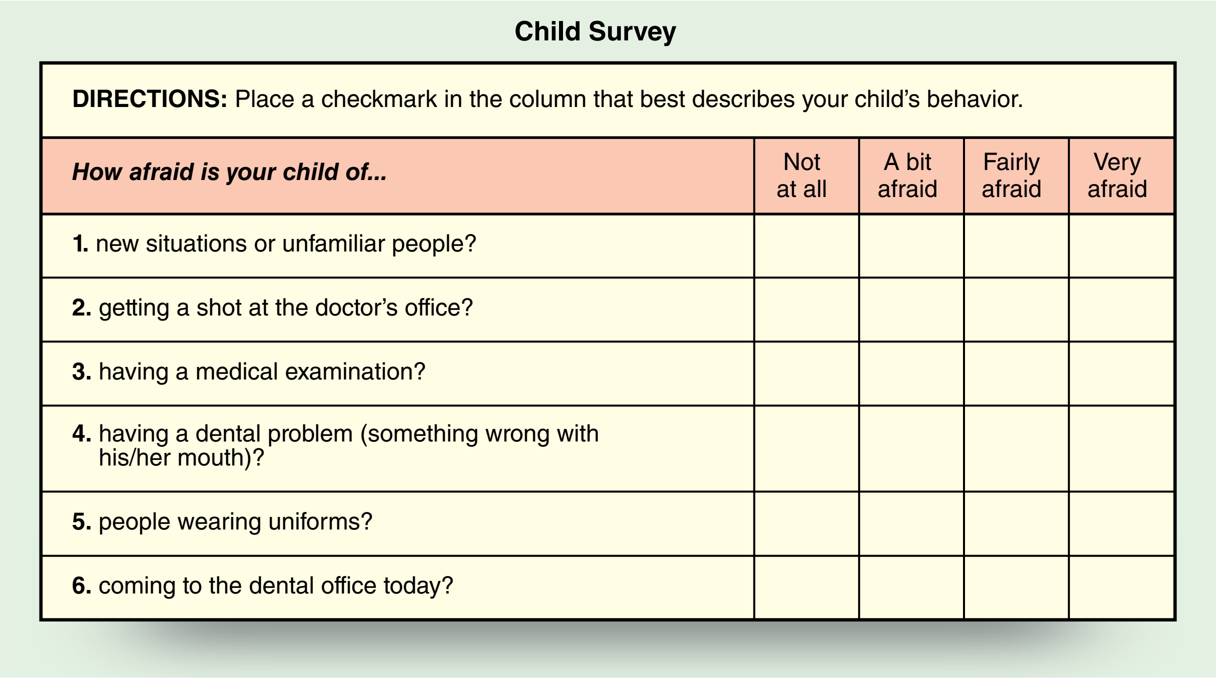
Behavioral Assessment of the Pediatric Patient

Dental fear has been related to personality, increased general fears, previous painful dental experiences, parental dental fear, age, and gender. When treating a pediatric patient, the first issue of concern is the child's behavior. One of the most challenging issues for the clinician is to anticipate what type of behavior can be expected from a child or adolescent patient. A child who arrives at the dental office crying or screaming is obviously fearful. In contrast, a child who is quiet or withdrawn is exhibiting behavior that is difficult to read. To prevent disruptive behavior, it is imperative to identify the dentally anxious child at the earliest possible age. For this purpose, different types of behavioral measuring scales have been used to assess dental fear. Behavioral assessment tools are helpful tools to classify behavior and more effectively care for children and adolescents.

1. **Behavioral Management**
   a. **Impact of Clinician Behaviors.** The success or failure of iatrosedation can hinge on the behavior and demeanor of the dental practitioner. For example, a dental practitioner who has a soothing demeanor and a good bedside manner will be able to allay and calm an anxious pediatric patient much better than a jittery and nervous practitioner. These same behaviors can also be used by the dental practitioner to manage the anxiety of an adult patient or a patient with special needs.
      1. As a vital member of the dental team, the dental hygienist plays a critical role in the success of the dental team by establishing and shaping the pediatric dental patient's perceptions, beliefs, and experiences around dentistry.
      2. It is imperative for the clinician to be able to recognize and address the dental fears and anxieties of a pediatric patient, then utilize effective behavioral management strategies to promote a positive dental experience (i.e., helping the child patient cope and find success in the dental setting).

   b. **Body Language Assessment.** Behavior management begins the moment the child enters the dental environment. Engagement and disengagement are important signals to monitor in the patient's body language.
      1. **Engagement behaviors** indicate interest, receptivity, or agreement. Good eye contact, smiling, and open body posture are all signs of engagement.
      2. **Disengagement behaviors** signal that a person is bored, angry, or defensive. If someone is disengaged, the amount of eye contact decreases; people tend to look away from things that distress them. Disagreement also shows up in compressed lips, clenched jaw muscles, or a head turned slightly away. If the child in the waiting room sits with his feet wrapped around the legs of the chair, he may be anxious and disengaged.

c. **Pre-appointment Evaluations.** Pre-appointment evaluations can help with predicting behavior outcomes.[17] The medical history–taking aspect of the first visit provides an excellent opportunity for the dental team to assess the potential behavior of the child. The objectives of a behavioral assessment tool are twofold: (1) to learn about patient and parental concerns and (2) to gather information that would allow the clinician to estimate the cooperative ability of the child. **Figure 36-2** provides examples of questions used to predict a child's behavior in a dental setting.



Child Survey

DIRECTIONS: Place a checkmark in the column that best describes your child's behavior.

How afraid is your child of...	Not at all	A bit afraid	Fairly afraid	Very afraid
1. new situations or unfamiliar people?				
2. getting a shot at the doctor's office?				
3. having a medical examination?				
4. having a dental problem (something wrong with his/her mouth)?				
5. people wearing uniforms?				
6. coming to the dental office today?				

Figure 36-2 Behavioral Assessment Tool. Shown here is an example of a short questionnaire to assess the potential behavior of a child before the first dental visit. While taking the medical history, the parent or caregiver can be asked to complete a brief behavioral questionnaire. Several negative responses on the behavior assessment tool can indicate a potentially challenging outcome for the patient's visit and alert the clinician to a potential behavioral problem that may complicate the dental appointment.

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2. **Behavioral Assessment Tools.** Behavioral assessment scales are helpful tools to classify and document behavior of children and adolescents. Documentation of a child's behavior during a series of appointments, or over a period of years, can assist in behavior management.
   a. **Frankl Behavioral Rating Scale**
      1. One of the most widely used systems was introduced by Frankl et al. in 1962. It is referred to as the **Frankl Behavioral Rating Scale**.[1,18] The scale divides observed behavior into four categories: definitely negative, negative, positive, and definitely positive. A detailed description of the scale is provided in **Table 36-1** and **Figures 36-3A-D**.
      2. The Frankl scale lends itself to a shorthand form of documentation in the chart or computerized record. A child displaying positive cooperative behavior can be identified by jotting down (+) or (+/+). Conversely, uncooperative behavior can be noted by (−) or (−/−).
      3. When documenting the behavioral assessment at the conclusion of the visit in the patient record, it is important to be very descriptive on key aspects of the responses. Sample observations could be "(−) the patient cried, was withdrawn and required lots of reminders to stay open for most of the visit" or "(+/+) the patient was pleasant and talkative. She hopped up into the big girl chair and was very interested in having her favorite bubble gum tooth sparkles."
      4. If behavior ranges from negative to positive during a visit, a simple notation could be (− > +). The management technique can also be recorded: "patient responded well to the T (Tell), S (Show), D (Do) technique."

Table 36-1 **Frankl Behavioral Scale**

Patient Record Notation		Patient Behavior
−/−	© BestPhotoStudio/Shutterstock	**Figure 36-3A** Refusal of treatment; forceful crying, fearfulness, or any overt evidence of extreme negativism
−	© XiXinXing/Shutterstock	**Figure 36-3B** Reluctance to accept treatment; uncooperativeness, some evidence of negative attitude, sullen and withdrawn
+	© Kzenon/Shutterstock	**Figure 36-3C** Acceptance of treatment; cautious at times; willingness to cooperate with the dentist, at times with reservation, but the patient follows directions cooperatively
+/+	© wavebreakmedia/Shutterstock	**Figure 36-3D** Good rapport with the clinician, interest in dental procedures, laughter and enjoyment

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Section 3

Iatrosedation Techniques with Pediatric Patients

The dental hygienist plays a critical role in shaping the pediatric dental patient's perceptions and beliefs about dental care. Often, the hygienist is the first member on the dental team to have a meaningful interaction with the child patient during his or her initial office visits. Iatrosedation is an effective behavioral management approach to overcome dental fear and anxiety in adults and children. The end-result of effective iatrosedation is to make the dental visit more pleasant for both the patient and members of the dental team, facilitate the delivery of high-quality dental care, and improve outcomes of treatment.

Several methods of timely and appropriate behavioral interventions with pediatric and special needs patients are available.[1,19–27] This section presents seven common behavioral guidance practices that are effective when working with pediatric patients in the dental setting. Many of the behavioral guidance techniques prescribed for the pediatric patient population would also serve to ease the fears in adult patients. Nevertheless, regardless of the patient's age, behavioral guidance always begins with the clinician! See **Table 36-2** for a summary of key points.

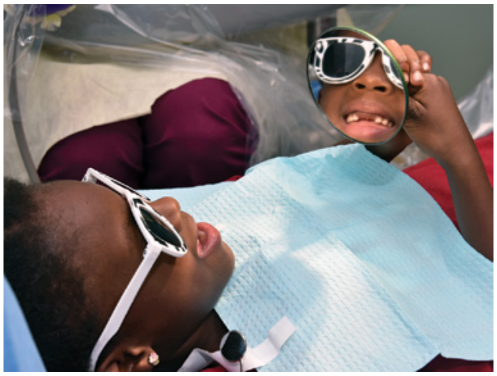
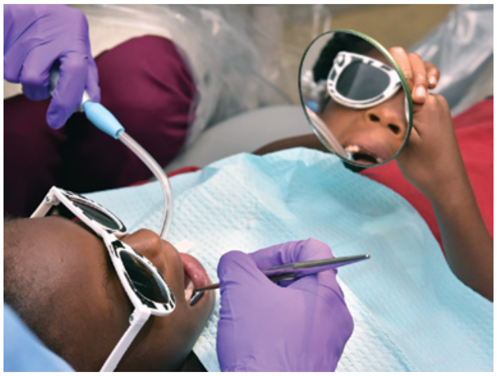
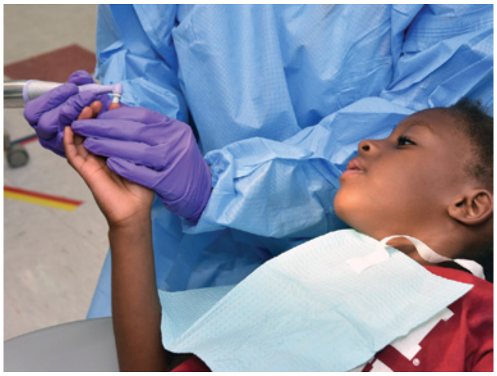
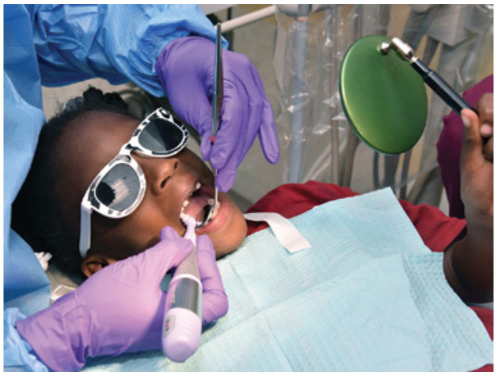
Table 36-2 **Behavioral Guidance to Reduce Anxiety**

Technique	Description
Tell–Show–Do	Informing the patient of what will happen, demonstrating it, and then performing that part of the procedure
Nonverbal communication	The clinician reinforces desired behavior and guides behavior through body language, contact, posture, and facial expression
Voice control	A controlled modulation of the clinician's voice volume, tone, or pace to influence and direct the patient's behavior
Positive reinforcement	Offering a tangible or social reward in response to the patient having a desired behavior
Distraction	Directing the patient's attention from a behavior, thought, or feeling to something else
Modeling	Providing the patient an example or demonstration about how to behave constructively
Systematic desensitization	Reducing the patient's anxiety by initially presenting a situation or object that evokes a little fear, and then progressively presenting stimuli that are more fear-provoking

Avery DR, Dean JA, McDonald RE. *McDonald and Avery's Dentistry for the Child and Adolescent*. 10th ed. Elsevier; 2016:700; Dentistry AAOP. Behavior guidance for the pediatric dental patient. *Pediatr Dent*. 2017;39(6):246–259.

1. **Effective Techniques for Easing Patient Fears**
   a. **Tell–Show–Do**
      1. The **Tell-Show-Do** approach is the cornerstone method of behavioral shaping used to help children. Tell–Show–Do is one of the most effective approaches when working with pediatric patients. The approach is illustrated in **Table 36-3** and **Figures 36-4A-D**.
      2. The objective of the Tell–Show–Do approach is to familiarize the patient with the dental setting and expose the child to the dental procedure and instruments. This reinforces desired behavior by gaining the patient's trust and empowers the child regarding the experience.

Table 36-3 **Tell–Do–Show Approach**

 © Wolters Kluwer	**Figure 36-4A** **TELL.** ■ A verbal explanation of a procedure using words that are appropriate to the stage and level of development of the patient. ■ Explain to the patient what you are going to do and orient them in the patient mirror.
 © Wolters Kluwer	**Figure 36-4B** **TELL.** ■ Use visual, auditory, olfactory, and tactile aspects of the procedure to help the child to understand. ■ Explain to the patient what you are going to do and introduce the procedure using the mouth mirror and saliva ejector or "straw."
 © Wolters Kluwer	**Figure 36-4C** **SHOW.** ■ A demonstration of the procedure for the patient. ■ For example, lightly applying a rubber prophy cup on the child's fingernail allows the patient to see, hear (whirring sound of the handpiece), and feel this procedure.
 © Wolters Kluwer	**Figure 36-4D** **DO.** ■ Begin the rubber cup polishing procedure.

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b. **Nonverbal Communication**
   1. The clinician models and guides the desired behavior through his or her own body language, contact, posture, and facial expression (**Figure 36-5**).

Figure 36-5 Behavioral Guidance Starts with You. A positive experience for a child patient starts with the clinician's body language, posture, facial expression, and tone of voice

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   2. The objective of nonverbal behavioral modeling is to establish positive rapport with the patient to promote desirable behavior.

c. **Voice Control**
   1. When speaking with the patient, the clinician uses a controlled volume, tone, and pace to influence and direct the patient's behavior.
   2. The objective is to gain the patient's attention and confidence.

d. **Positive Reinforcement**
   1. **Positive reinforcement** is the addition of a tangible or social reward following a desired behavior that makes it more likely that the behavior will occur again in the future.
      a. The clinician rewards appropriate behaviors to strengthen the recurrence of those behaviors. For example, the clinician rewards a child with a colorful sticker for sitting still while the clinician takes a digital radiograph.
      b. Social rewards include positive voice modulation, facial expression, verbal praise, and appropriate physical demonstrations of affection by members of the dental team.
      c. Nonsocial rewards include tokens, toys, a toothbrush, and stickers.
   2. The objective of positive reinforcement is to reinforce desired behaviors.

e. **Distraction**
   1. **Distraction** is the technique of diverting the patient's attention away from a procedure that the child may perceive as unpleasant. Giving patients something else to focus on is especially effective. Examples of distraction techniques are listening to music through ear buds, watching videos on a ceiling-mounted monitor, squeezing a stress ball, or cuddling a stuffed toy.
   2. The objective is to build trust between the clinician and patient and avert patient avoidance behavior.

f. **Modeling**
   1. **Modeling** is the technique of learning new skills by observing and then imitating another person, such as a parent or another patient, who performs the behavior to be acquired. This approach works well in a dental office with an open bay concept where the child can watch another patient demonstrating positive behavior during the procedure. For example, a child observes the dental clinician using dental instruments in another patient's mouth.
   2. The objective of modeling is to reinforce desired behavior by allowing a child to observe a patient who is tolerating the treatment well.

g. **Systematic Desensitization**
   1. **Systematic desensitization** is the technique for reducing a patient's anxiety by initially presenting a situation or object that evokes a little fear, and then progressively presenting stimuli that are more fear-provoking.
      a. Oftentimes, having the patient do a "getting acquainted" visit to the office is helpful if a child has a history of anxiety in new situations or with the pediatrician. For example, the first visit might consist of visiting a treatment room, riding up and down in a dental chair, and having water sprayed in the mouth from a dental "water squirter" (with no actual treatment being performed at this visit).
      b. It is also helpful to educate the parents on the language used in the office as well as establish expectations for the future visit. **Box 36-2** provides some examples of word substitutions for dental terminology.

Box 36-2 Examples of Word Substitutions of Dental Terminology for the Pediatric Patient

Dental Terminology	Word Substitute
Dental light	Flashlight
Saliva ejector	Straw
High-evacuation suction	Mouth vacuum
Air syringe	Air dryer
Water syringe	Water squirter
Explorer	Tooth counter
Scaler	Tooth scrubber
Prophy angle	Tooth tickler
Prophy paste	Tooth sparkler
Sealant	Tooth paint
Etchant	Tooth soap
Radiographs	Tooth picture
Fluoride	Tooth vitamin

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      c. "Mapping out" the experience of a dental appointment with visual aids as brochures or "My First Dental Visit" books can help desensitize the patient and ensure a successful visit.
   2. The objective of systematic desensitization is to create a positive experience by slowly introducing a child to the dental setting.

h. **The Pediatric Dentistry Triad: Clinician–Patient–Parent/Caregiver**
   1. With most adult patients, the clinician–patient interaction should be a one-to-one relationship.
   2. For pediatric or special needs patients, the clinician interacts not only with the patient, but also with parents and caregivers. This three-way relationship is known as the **pediatric dentistry triad**.[1,22,25] It is important for the hygienist to establish a positive rapport with parents and caregivers as **Figure 36-6** illustrates. Parents and caregivers need to know that all members of the dental team are competent and care about the best outcomes for the child.

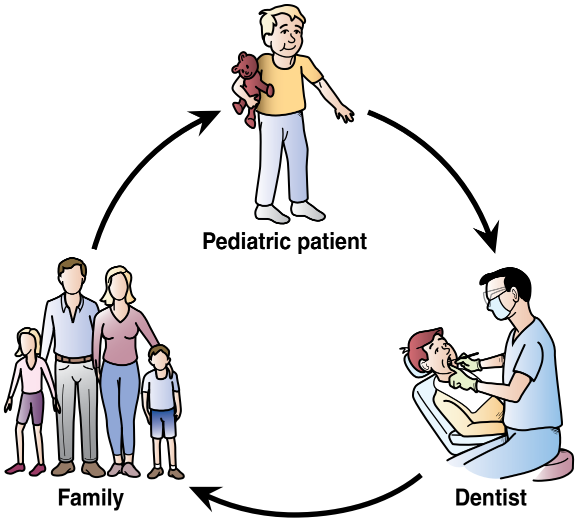

Figure 36-6 The Pediatric Dentistry Triad. When treating pediatric or special needs patients, the clinician establishes a relationship with the patient and his or her parents or caregivers.

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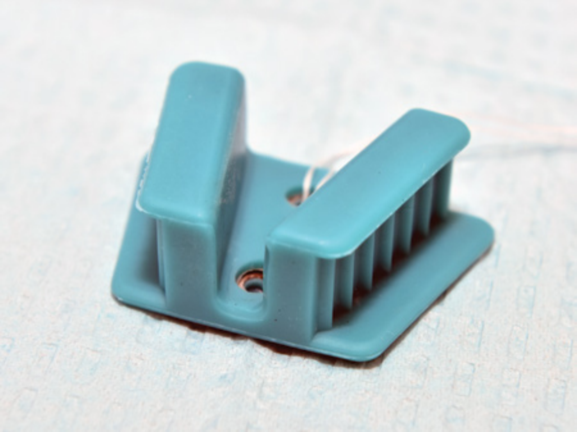
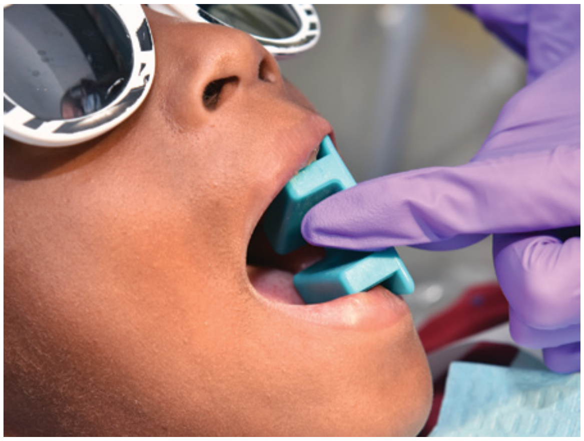
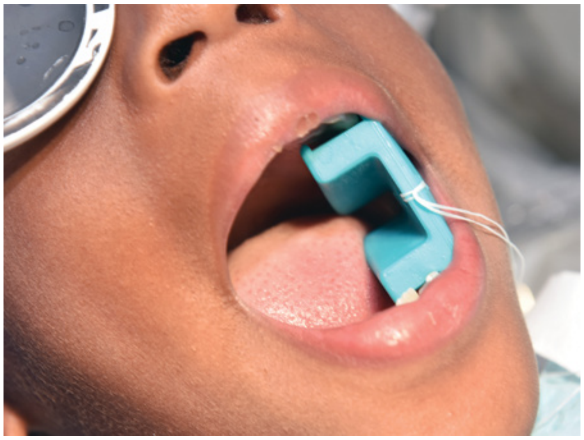
a. Partnering with the child and the parent/caregiver facilitates achieving a positive outcome to the dental visit for the child.
b. Educating the parent/caregiver about the planned treatment and anticipated treatment outcomes is helpful in setting expectations and avoiding overinvolvement of the parent/caregiver during the appointment.
c. Involving the parent/caregiver as a silent observer is a means to support a one-on-one relationship between the child and the dental clinician.

3. The most important goal of the dental visit is for the child to have a positive experience. To accomplish this all members of the dental team should:
   a. Establish positive communication with the patient and parent/caregiver.
   b. Build a trusting relationship with the patient and parent/caregiver.
   c. Strive to alleviate fear and anxiety for the child patient.
   d. Promote a positive attitude towards oral health in general with the entire family.
   e. Deliver quality and competent dental oral health care.

2. **Adaptive Support.** On some occasions, it becomes necessary to utilize an adaptive support tool such as a bite block when working with pediatric dental patients.
   a. A bite block is a wedge-shaped device with the rubber-like texture that is placed between a patient's teeth and used to assist the patient to keep the mouth open wide during a procedure. The bite block also serves a "resting place" that allows the patient to relax his jaws when holding the mouth open for prolonged procedures such as application of pit and fissure sealants.
   b. Bite blocks come in different sizes; therefore, it is important to select the proper fit. To achieve maximum mouth opening, the bite block should be placed more posteriorly.
      1. Informed consent should be obtained prior to using a bite block. For patients under the age of consent or adults with diminished mental capacity, informed consent should be obtained from a parent or caregiver.[28–30]
      2. Prior to placing a bite block in the patient's mouth, it is imperative to securely attach a length of dental floss to the device. The dental floss should be of an adequate length to aid in retrieval of the bite block if it is dislodged and falls into the posterior pharyngeal area.
      3. Use of a bite block is demonstrated in **Table 36-4** and **Figure 36-7A-C**.

Table 36-4 **Application of a Dental Bite Block**

 © Wolters Kluwer	**Figure 36-7A** 1. Select a bite block of appropriate size for the patient's mouth. Attach approximately a 12-inch length of dental floss to the bite block.
 © Wolters Kluwer	**Figure 36-7B** 2. Gently guide the bite block along the occlusal surfaces of the teeth.
 © Wolters Kluwer	**Figure 36-7C** 3. Ensure a comfortable fit of the bite block in the patient's mouth. Note that the dental floss is positioned to aid in retrieval of the bite block.

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3. **The Infant Oral Exam**
   a. Guidelines for Infant Oral Health Care. The American Academy of Pediatric Dentistry[31,32] makes recommendations that relate to the role of the dental hygienist in infant oral health care:
      1. Every infant should receive an oral health risk assessment from his or her primary medical care provider or dental health care provider by 6 months of age.

2. Parents or caregivers should establish a dental home for infants by 12 months of age.
3. Health care professionals and all stakeholders in children's health should support the identification of a dental home for all infants at 12 months of age.

b. **Role of the Hygienist in Guidance and Counseling**

1. The guideline that the infant should have the "first dental visit by the child's first birthday" places the dental hygienist in a prime position to engage in infant oral health care. A benefit of this philosophy is that it exposes pediatric patients to the dental setting at an early age. The hygienist will be able to educate parents and caregivers on setting the pediatric patient on an early path to oral health.
2. Anticipatory guidance/counseling is a proactive counseling process whereby the clinician provides practical, developmentally appropriate information about a child's health so as to prepare the parents and caregivers for upcoming physical, emotional, and psychological milestones that will impact the dental needs of the child. The recommended topics for parent and caregiver counseling that relate to the role of the dental hygienist include oral hygiene and dietary habits.

c. **The Knee-to-Knee Technique for the Initial Infant Dental Visit**

1. Proper positioning of the child is critical to conducting an effective and efficient clinical exam in a young child. In general, the **knee-to-knee position** should be used with children ages 6 months to 3 years, or up to age 5 with children who have special health care needs. Children older than 3 may be able to sit alone in the dental chair. The knee-to-knee positioning is one of the most secure and effective approaches for the infant dental visit.
2. To perform the infant oral exam, the child needs to be in a safe and secure position, usually held by his or her parent or caregiver.
   a. The dental hygienist and the parent/caregiver sit on two chairs facing one another and take a seat with knees touching to make a table. They can use a patient pillow; however, this is not necessary.
   b. Knee-to-knee positioning may be accomplished with the child on the dental chair and the parent and clinician on dental stools. Alternatively, the child sits on the parent's lap with the parent and clinician seated on dental stools or office chairs.
   c. It is important that there is adequate light available, or an assistant could hold a flashlight to help visualize the patient's mouth.
   d. Refer to **Figures 36-8** through **36-13** for a detailed illustration of this approach.

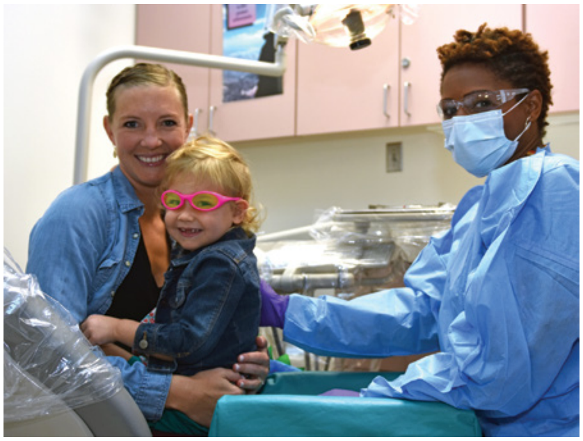

Figure 36-8 Step 1. Knee-to-Knee Technique: Make Positioning Fun.

- Have the child sit on the parent's lap in a straddle position with the child facing the parent/caregiver.
- The clinician faces the parent and approaches the parent until their knees touch, forming a bridge. A pillow may be used to help secure the patient.
- Ask the parent to gently lean the child back onto the clinician's lap.
- The parent may hold the child's hands to avoid having the child interfere with the clinical examination.
- Another important tip: keep child's legs around the caregiver's waist during the exam. This will also aid in restraining the child's body movement.

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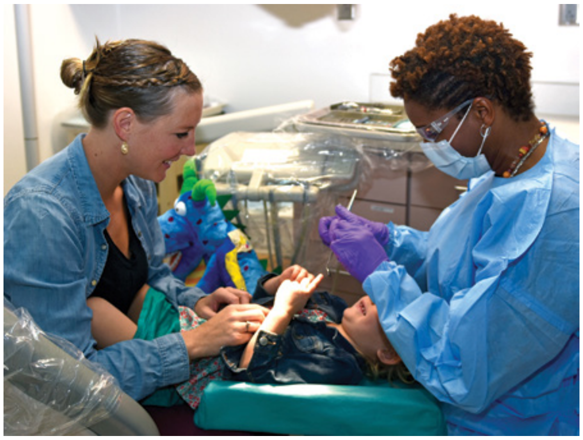

Figure 36-9 **Step 2. Knee-to-Knee Technique: Use Tell–Show–Do.**

- Even when approaching the knee-to-knee position with the young child, utilizing Tell–Show–Do is critical. This allows the patient to warm up to the "fun" instruments that will be used during the visit.
- Beginning with introducing the dental mirror also aids in desensitizing the patient.

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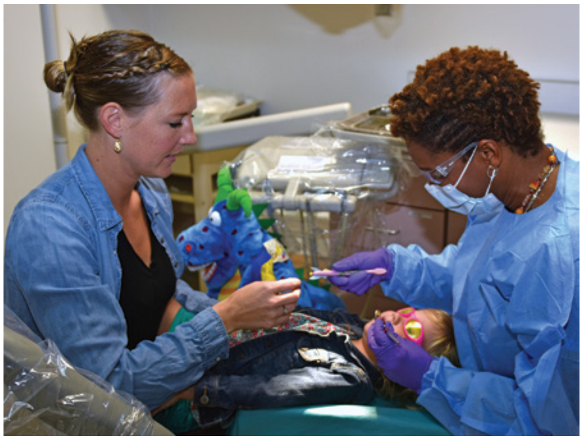

Figure 36-10 **Step 3. Knee-to-Knee Technique: Partner with the Parent or Caregiver.**

- Invite the parent to partner with you with the Tell–Show–Do approach.
- Allow the parent to demonstrate how he or she brushes the child's teeth at home.
- This allows the patient to become more comfortable with having dental instruments in the mouth.

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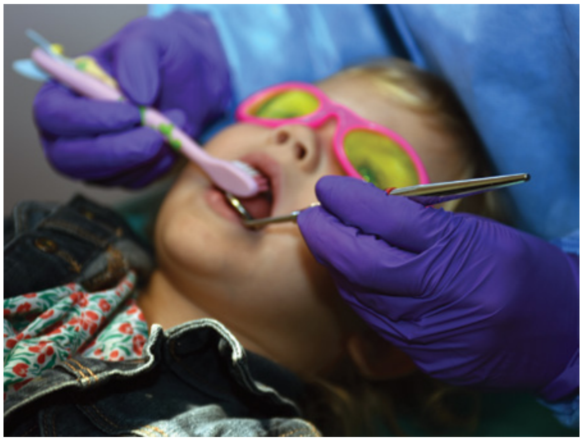

Figure 36-11 **Step 4. Knee-to-Knee Technique: Toothbrush Prophylaxis.**

- A toothbrush is effective in removing plaque biofilm in most young children. It is also nonthreatening to young children and serves to demonstrate the proper brushing technique to the caregiver.
- Clinical examination of the teeth and soft tissues can be accomplished while counting the child's teeth aloud. Many clinicians make a game of this task, singing songs, engaging the child's attention, and if all else fails, distracting the child with a brightly colored toy.
- Praise the child at each step for cooperation and good behavior.

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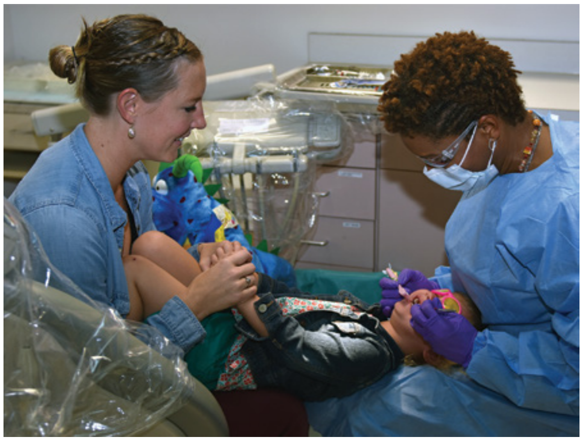

Figure 36-12 **Step 5. Knee-to-Knee Technique: Alternate Position for Unhappy Patient.**

- Controlling the child's body movement, particularly leg movements, can be achieved by the parent gently resting his or her arms on the child's bent legs, enfolding the child's body in a snug "hug."
- The parent holds the patient's hands and secures the patient's legs with his or her forearms. The patient remains safe even in this position and when the patient is unhappy.

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Figure 36-13 **Step 6. Knee-to-Knee Technique: Celebrate.**

- Celebrate the dental visit by highlighting the positives.
- If the patient is unhappy, still try to celebrate the end of the visit.
- Celebrate the positives.
- Provide lots of praise and prizes!

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d. **Celebrate the End of the Visit.** Accentuate the positives; do not focus on the negatives.
   1. Keep in mind the patient's level of growth and development. Crying at this stage and age is to be expected and within normal limits.
   2. Take this opportunity to also celebrate with the parent/caregiver who helped the child have a successful visit. Reassure the parent/caregiver that an uncooperative or tearful child is to be expected and that behavior should improve over time with increased maturity of the patient.
   3. Reinforce the need for good oral hygiene, fluoride applications, and healthy nutrition habits.

e. **Practical Considerations for Providing Care for Pediatric Patients.** The primary aim is to provide quality care for the pediatric patient that will minimize disruptive behavior, promote a positive psychological response to treatment, and promote patient safety and welfare. To achieve this end, a consideration should be made in three practical areas: the practice environment, patient scheduling, and parental presence or absence in the exam room.
   1. **Office Environment and Design.** The overall office environment should be comfortable and inviting for families. To achieve this effect, some offices have themes, bright colors, designated areas for play with toys and/or video games, as well as adult areas with beverage centers.
   2. **Scheduling.** When scheduling it is helpful to consider the patient's age and development. Children ages 5 and under as well as those with special health care needs typically benefit from appointment times in the morning. Avoiding nap time or other disruptive times of the day helps the dental team support positive behavioral outcomes.
   3. **Parental Involvement**
      a. The literature has demonstrated that children's behavior is unaffected by parental presence or absence. However, the exception to this effect has been found in young children younger than 4 years of age. Typically, this demographic of children demonstrates better behavior with their parent or caregiver present.[24–27]
      b. It is normal for a child younger than 4 years to experience separation anxiety at this stage in development. It has been shown that this is a strong indicator of dental anxiety. Therefore, it appears that parental presence is helpful with the younger dental patient, but has no positive impact on older children. It is helpful for all members of the dental team to discuss these expectations with parents and caregivers.
      c. If the decision is made to allow parents into the dental treatment room, the clinician needs to establish positive communication with the adult and set expectations. In doing so, the clinician should avoid allowing the parent or caregiver to become a distraction during treatment by:
         1. Disrupting direct clinician communication with the child by controlling the conversation or providing incorrect instructions.
         2. Repeating (echoing) the clinician's instructions to the child.
         3. Attempting to "coach" or "cheer the patient on" during the appointment in a disruptive manner.

Chapter Summary Statement

Iatrosedation and behavioral guidance are effective strategies to implement for the child and adolescent patient population. Therefore, it is critical that all members of the dental team understand the importance of behavioral guidance and be comfortable with implementing these techniques as part of patient care when indicated. The ability to communicate well with the child and the parent/caregiver, express empathy, and project calmness are necessary skills to promote positive behavior and reduce disruptive behavior. The behavioral guidance assessments and techniques described in this chapter are universal strategies and approaches used in pediatric dentistry. When appropriately used, iatrosedation and behavioral guidance techniques set the tone of the dental appointment as a positive experience and provide the child patient with a rewarding opportunity to gain faith in their clinician and acquire confidence in their own abilities to overcome previously conceived fears and anxiety associated with dental treatment.

Section 4

Focus on Patients

Evidence in Action

Answer the following questions for Cases 1–4:

1. How would you rate this patient's behavior based on the Frankl Scale of Behavioral Assessment?
2. What behavior guidance techniques would you use to help this patient have a successful visit?

Case 1: A 4-year-old female patient with past medical history of allergy to penicillin and eczema happily greets you in the lobby. She hops into the dental chair and is eager to have dental treatment. During the procedure, she is very cooperative and talkative.

Case 2: A healthy 3-year-old male patient walks into the treatment area clenching his mother's hands and offers you little eye contact. He is hesitant to sit in the dental chair and requires a great deal of coaxing to participate with the treatment. He begins to cry out during the prophy and during the entire appointment but keeps his mouth open.

Case 3: A healthy 2-year-old male patient and his older sister arrive for their appointments. The older sister is seen first, and the younger brother is able to watch the entire visit. After completion of the older sister's appointment, you now ask the 2-year-old to sit in his very own "big boy chair." After modeling with his older sister, the patient sits in the chair by himself. The patient is attentive and cooperative for the visit.

Case 4: A healthy 6-year-old female patient reluctantly greets you in the lobby. As you approach the treatment area, the patient announces loudly that she doesn't want to have her teeth brushed today. She then refuses to sit in the dental chair, sits on the floor and holds on to the base of an extra chair for parents in the room. Mom can convince the patient to sit in the chair with promises to buy a treat at McDonald's following the visit if the treatment is completed. The patient attempts to bite your finger and intentionally spits out the prophy paste in your face.

Case 5: A 4-year-old new patient presents to your dental team's office and has a past medical history of asthma that is controlled as needed (prn) with Claritin and albuterol. In the lobby, he screams and kicks when asked to put down a video game and come to the treatment room. Once back in the treatment area he enjoys playing with the toys but won't let go of mom's arm when it's time to sit in the dental chair.

1. Consider this patient's stage of development both physically and emotionally and assess his current behavior.
2. Explain the approaches to behavioral guidance strategies and alternatives that may be utilized to help this patient have a successful visit.

Clinical Patient Care

CASE 1

You plan to take bitewing radiographs on a healthy 3-year-old female patient. She refuses to open her mouth for the radiograph after happily sitting in the chair.

- What behavioral guidance approaches might you use with this 3-year-old patient?
- If the patient decides to cooperate and accomplishes having the radiographs taken, how would you assess her behavior based on the Frankl scale?

CASE 2

A patient with Down syndrome is excited about his recall exam today. He gives you a big hug in the lobby, eagerly walks into the treatment area. During the oral exam, you notice there is moderate gingivitis in the anterior sextants of the maxillary and mandibular teeth. When you attempt to instrument these areas, the patient tightly purses his lips and refuses to keep his mouth open.

- How would you proceed with this visit?
- What special considerations should you make with a child diagnosed with Down syndrome?
- Based on the Frankl scale, how would you rate this patient's behavior?

Ethical Dilemma: 1

Mom presents with her 17-year-old son, George, with a medical history of Asperger syndrome, developmental delay, and dairy allergy. George's caregiver, Nona, usually comes to all medical and dental visits, but is going to be running late for today's appointment. George's mother has multiple appointments scheduled for the day and insists that her son's appointment begin without Nona's assistance.

George is cooperative about walking to the treatment room but is hesitant to sit in the dental chair. George's mother begins to physically push George into the chair stating he just needs a gentle nudge. George's mother asks the hygienist to help her to "force her son into the chair" and becomes upset when the hygienist declines to do so.

Fortunately, Nona arrives. It takes 10 minutes to coax George into the dental chair. Nona suggests that the appointment be rescheduled because George is "having a bad day." George's mother refuses to reschedule saying that "George only requires a bit of tough love."

George remains uncooperative, squeezing his lips closed, and struggling. The entire time, George's mother is adamant that the dental team help her force the George to complete his treatment today. She states she had put a lot of effort and time into bringing George to the dental office today. She asks the hygienist for his opinion about trying to continue with treatment today.

1. What is the best approach for behavioral guidance for a special needs patient?
2. Should a dental clinician continue to "push" a patient to complete treatment? Does this change in the case of an emergency?
3. Are there ethical principles in conflict in this case?

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CHAPTER 37

Oral Malodor, Xerostomia, and Hyposalivation

- Section 1 Oral Malodor
- Section 2 Xerostomia and Hyposalivation
- Section 3 Focus on Patients
  - Clinical Patient Care
  - Ethical Dilemma

CLINICAL APPLICATION

This chapter discusses two of the most difficult disorders to treat in dentistry: oral malodor and xerostomia/hyposalivation, largely because of the different etiologies attached to the disorders as well as the fact that there is no cure to these conditions. Hence, treatment focuses on relieving symptoms and preventing complications. This chapter seeks to describe how dental practitioners can work more effectively with their patients to address these challenges head-on and implement more focused and targeted interventional strategies to address these disorders.

LEARNING OBJECTIVES

- Define the term *oral malodor*.
- Explain the significance of oral malodor.
- List the chemical compounds found in oral malodor.
- Explain the association between oral malodor and other oral conditions.
- List some extraoral causes of oral malodor.
- Be able to explain the diagnosis of oral malodor to a patient.
- Describe the treatment management of a patient with oral malodor.
- Distinguish *xerostomia* from *hyposalivation*.
- Explain the importance of saliva
- Describe some causes of xerostomia and hyposalivation.
- Be able to explain the diagnosis of hyposalivation to a patient.
- List some health care providers who might be involved in the comanagement of a xerostomic/hyposalivation patient.
- Describe some principles of the dental management of patients with xerostomia and hyposalivation.

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KEY TERMS

Halitosis
Oral malodor
Genuine halitosis
Pseudo-halitosis
Halitophobia
Volatile sulfur compounds (VSCs)
Tongue coating
Organoleptic test (OLT)
Xerostomia
Hyposalivation
Mouth breathing
Sjögren's syndrome
Polypharmacy
Medication-Induced Xerostomia and Hyposalivation
Sialometry

Section 1

Oral Malodor

The odor of a patient's breath can be pleasant or unpleasant. If the odor is unpleasant, the terms *oral malodor*, *halitosis*, *breath malodor*, or simply *bad breath* have all been used to describe the perceived unpleasant odor. In general, malodor can be classified into one of three categories depending on its origin: (1) intraoral malodor, (2) nonoral malodor, or (3) extraoral malodor. Intraoral malodor is caused by anaerobic microorganisms residing within a dental biofilm that enzymatically break down sulfur-containing amino acids into volatile, noxious, foul-smelling sulfur-containing substrates known as volatile sulfur compounds (VSCs). In contrast, a nonoral malodor is a malodor that originates from exogenous sources, rather than from the oral cavity. Examples of nonoral malodors include the consumption of certain pungent food/drink products (e.g., onions, garlic, or coffee) and smoking. Extraoral malodor is a manifestation of a serious extraoral pathologic systemic condition(s), such as gastrointestinal disturbances, upper respiratory infections, and diabetic ketoacidosis. An extraoral malodor can be difficult and challenging for an oral health care provider to manage alone. As such, in the absence of any apparent intraoral source or nonoral source of the malodor, the oral health care provider should suspect that the patient is suffering from a potentially life-threatening underlying or undiagnosed medical condition(s) that warrants immediate consultation with the patient's physician and/or nurse practitioner.

1. **Implications of Oral Malodor**
    a. **Halitosis.** Socially, **halitosis** is a worrisome problem that can stigmatize an individual suffering from this condition. In the United States alone, the magnitude of this problem is underscored by a reported consumer expenditure of between $1 billion and $3 billion per year on over-the-counter products purported to minimize bad breath.[1] Malodor is a common complaint, with up to 30–50% of people worldwide self-identify as having frequent incidents of malodor.[2,3] The American Dental Association Council on Scientific Affairs counsels that **oral malodor** is an identifiable condition that should be treated by the oral health care professional.[4] Along with identifying the malodor, the oral health care provider will need to be able to distinguish two forms of oral malodors: genuine halitosis and pseudo-halitosis. **Genuine halitosis** is a form of malodor that can be objectively verified.[5] In contrast, **pseudo-halitosis** is a form of malodor where the affected individual simply imagines that he or she has malodor but there is no objective evidence to verify it.[5] An individual suffering from pseudo-halitosis should be comanaged by a team of medical, dental, and mental care experts to determine whether the patient's perceived chief complaint is due to an actual underlying or undiagnosed psychosomatic condition.
        1. It is safe to assume that the problem of halitosis will increase as the population of elderly adults worldwide increases. A Thai study of more than 800 subjects reported that being 30 years of age or older significantly correlated with a higher prevalence of self-reported oral malodor.[6] Additionally, in a study assessing the prevalence of halitosis among the elderly living in Swedish nursing homes, over 50% of residents were found to suffer from halitosis. Possible reasons for the high prevalence correlates to the elderly having a higher prevalence of periodontal disease, hyposalivation, and mental cognitive decline, which may hinder the daily self-care regimen.[7]
        2. No gender predominance seems to exist for halitosis, and the age of patients with halitosis includes all ages from childhood to the elderly.

b. **Halitophobia.** Some individuals have an extreme fear and irrational preoccupation of bad malodor that does not actually exist. This is known as **halitophobia**. Halitophobia is a variant of pseudo-halitosis.
   1. Halitophobia is also known as delusional halitosis.
   2. Halitophobia is not a benign condition. Instead, it can be a sign of obsessive-compulsive disorder (OCD). People with OCD who suffer from halitophobia tend to overbrush or excessively floss. Although patients are encouraged to brush and floss (or use any type of interdental devices) daily, an OCD patient with halitophobia may overdo performing the self-care task. This may lead to irreversible damage to the teeth (such as tooth abrasion) and/or irreversible loss of periodontal tissue (such as recession of the gingival margin or gingival ulcerations).
   3. Management of a patient with this disorder warrants a multidisciplinary approach that involves the input of physicians, including psychiatrists and ENT (ear, nose, and throat) specialists; dentists; and mental health care experts.

c. **Causes of Halitosis**
   1. In the majority of halitosis patients (approximately 90%), the underlying source of the halitosis originates in the oral cavity (oral malodor). Dental caries, gingivitis, periodontitis, low salivary flow, and tongue coating are most commonly associated with malodor.[4]
      a. In most cases, oral malodor is most evident upon awaking from sleep.[8] For most individuals, the malodor is resolved with brushing, flossing, eating, and/or drinking water.[8]
      b. Malodor that is not resolved by simple self-care techniques usually is associated with untreated periodontitis, dental caries, or other factors such as gross dental neglect, smoking, or xerostomia.[8]
   2. Even though the source of halitosis is usually intraoral, in some patients the oral malodor may be a manifestation of extraoral systemic conditions that can be difficult to identify. Approximately 10% of halitosis cases are extraoral—originating from systemic conditions or a location other than the oral cavity.[4,9] Dental professionals, therefore, should be diligent in completing a thorough medical history to understand all possible origins.[4]
   3. These extraoral causes are discussed briefly in this chapter and can include ear, nose, and throat pathology; certain systemic diseases; hormonal problems; liver disease; kidney disease; bronchial carcinoma; or gastroenterological pathology.[2]

d. **Chemical Origin of Oral Malodor**
   1. The oral malodor is produced by microbial proteolytic degradation of peptides present in the oral cavity, sloughed epithelial cells, food debris, and bacteria from the tongue, teeth, gingival crevicular fluid, and blood. Previously, it was thought that only a select group of periodontopathic Gram-negative anaerobic microorganisms such as *Tannerella forsythus*, *Porphyromonas gingivalis*, *Aggregatibacter actinomycetemcomitans*, and *Prevotella intermedia* were associated with oral malodor.[2,8,10] However, recent studies using more sensitive microbial assaying techniques have identified a wider range of microbial diversity than was previously thought. For example, a recent investigation found significantly higher percentages of both Gram-negative anaerobic bacteria, such as *Prevotella* spp., *Alloprevotella* spp., and *Leptotrichia* spp., and Gram-positive anaerobic bacteria, such as *Peptostreptococcus* spp. and *Stomatobaculum* spp., in the tongue coating samples of periodontally healthy halitosis subjects compared to tongue coating samples of periodontally healthy subjects without halitosis.[11] Additionally, many of the aforementioned bacteria significantly were correlated with the total number of volatile sulfur compounds (discussed below).[11] Hence, based on our current understanding of the microbial etiology of oral malodor, no single microbial species and no single cause of halitosis can be established. Rather, it is suspected that oral malodor may be due to the complex interactions of a vast array of different microbial culprits.[12]
   2. The aforementioned bacteria have the ability to degrade sulfur-containing amino acids to release noxious, foul-smelling **volatile sulfur compounds (VSCs)** in the breath, such as hydrogen sulfide (H_2S), methyl mercaptan (CH_3SH), and dimethyl sulfide [$(CH_3)_2S$].[2,4,8] With respect to the characteristics of the odors of these chemicals, hydrogen sulfide has a smell that equates to rotten eggs; methyl mercaptan has a putrid, musty smell; and dimethyl sulfide has a sweet, musty smell of cooked onion.[12]
   3. In addition to the volatile sulfur compounds, many non-sulfur-containing volatile compounds have been implicated in contributing to oral malodor. These chemicals include putrescine (the smell has

been described as being similar to spoiled fish or rotten meat), cadaverine (the smell has been described to be akin to spoiled fish, rotten meat, or urine), indole/skatole (the smell of these two chemicals has been equated to a strong fecal-like odor), butyric or propionic acid (the smell of these two chemicals have been described as rotten cheese smell, human vomit, sweat, or feet odor), and ammonia (which has a urine-like scent).[12]

4. **Box 37-1** is a list of some of the main sulfur-containing and non-sulfur-containing chemical compounds that have been found in oral malodor.

Box 37-1 Chemical Compounds Found in Oral Malodor

Hydrogen sulfide
Methylmercaptan
Dimethylsulfide
Putrescine
Cadaverine
Indole
Skatole
Butyric acid
Propionic acid
Ammonia

2. **The Association Between Oral Malodor and Other Oral Conditions.** Oral malodor is frequently associated with other common oral conditions. Examples of some of these other conditions are discussed below. It is important for members of the dental team to identify these conditions and address them as part of the strategy for managing oral malodor.
    a. **Oral Malodor and Dental Caries**
        1. In some patients, oral malodor can be associated with existing dental caries.
        2. Patients with deep carious lesions may be at risk of developing oral malodor because of putrefaction of sulfur-containing food products that lodge and remain stagnant within the confines of the carious lesion. (**Figure 37-1**) Food rich in sulfur-containing amino acids (e.g., beef, chicken, seafood, nuts, eggs, legumes, and allium vegetables) can be broken down by anaerobic bacteria residing within the dental biofilm to produce VSCs.

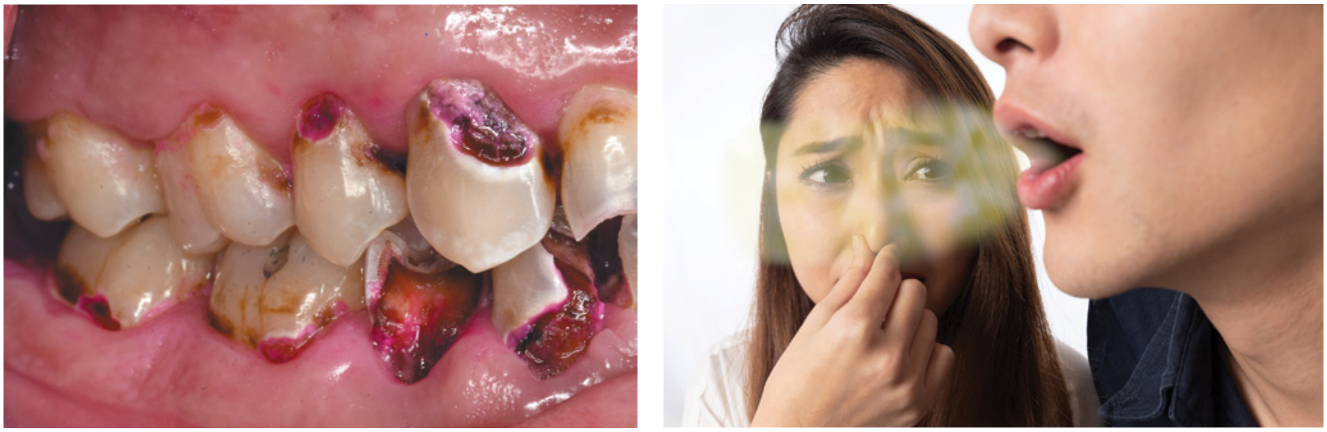


Figure 37-1 Oral Malodor and Dental Carious Lesions. Carious lesions tend to harbor both plaque and food debris. Note the pink dye disclosing the accumulated plaque deposits within the carious lesions. The sulfur-containing food that accumulates within the confines of the carious lesion will be enzymatically broken down by the bacteria to emit VSCs.

Right: © kei907/Shutterstock

b. **Oral Malodor and Periodontal Disease**
   1. Bacteria associated with periodontitis are almost all Gram-negative anaerobes that are capable of producing VSCs (**Figure 37-2**).
   2. In periodontitis patients, the level of VSCs (and therefore the unpleasant breath odor) correlates with the depth of periodontal pockets (i.e., the deeper the periodontal pockets, the greater the number of VSC-producing microorganisms within the subgingival plaque biofilm).
   3. In addition, some individuals suffering from oral malodor may have periodontal pathogens that can break down certain amino acids to release other non-sulfur noxious compounds, such as cadaverine and putrescine.

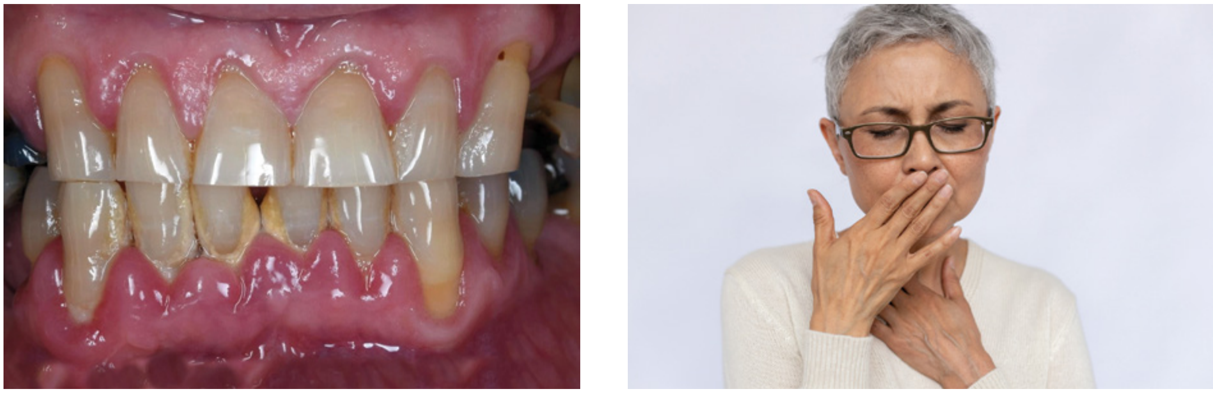


Figure 37-2 **Oral Malodor and Periodontal Disease.** Patients with periodontal disease present with deep pocket depths which harbor high levels of anaerobic bacteria. Some anaerobic bacteria associated with periodontal disease have the capability to break down sulfur-containing amino acids into VSCs or break down certain amino acids to release non-sulfur containing noxious compounds.

Right: © Mangostar/Shutterstock

c. **Oral Malodor and Dry Mouth**
   1. Dry mouth, will be discussed later in this chapter. At this point, however, it should be at the forefront of every clinician's mind that patients with dry mouth can have an increased level of oral malodor because the VSCs are more likely to be released as gases as the saliva dries.
   2. Thus, these two conditions (oral malodor and dry mouth) can be related to some degree in selected patients.

d. **Oral Malodor and Tongue Coatings**
   1. The dorsal (upper) tongue surface is another bacterial reservoir for bacteria associated with halitosis. The irregular surface of the dorsal tongue is an ideal niche for uninhibited bacterial growth.[1]
   2. In addition to the bacteria growing on the tongue surface, food remnants can be trapped in the irregular contours of the dorsum of the tongue, thus providing nutrients for bacteria already present on the surface of the tongue.
   3. In some patients, a benign anatomical variation of the surface of the tongue characterized by deep fissures on the dorsum of the tongue can harbor bacteria that contribute to oral malodor (**Figure 37-3**). This anatomical variation is known as a fissured tongue.
   4. The dorsal surface of the tongue frequently accumulates an off-colored brownish-gray or grayish-white **tongue coating** that consists of bacteria, food debris and desquamated epithelial cells.[2,4,8]
      a. This tongue coating cannot be removed without special efforts during self-care because of the irregular contours of the surface. Craters, fissures, and peaks on the surface of the tongue are covered with a fine sticky substance that harbors the malodorous bacteria. Researchers report that a single epithelial cell in the oral cavity can harbor up to 25 bacteria, whereas a single epithelial cell on the dorsum of the tongue can harbor up to 100 bacteria.[13]
      b. A close correlation has been reported in the literature between the presence of tongue coating and oral malodor.

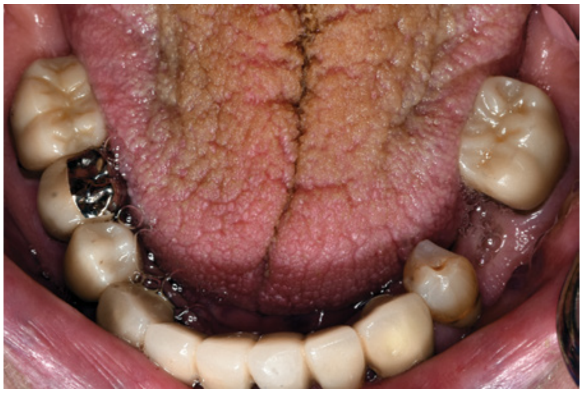


Figure 37-3 **Oral Malodor and Tongue Anatomy.** A patient with oral malodor presents with a benign, painless anatomical condition of the tongue known as fissured tongue. Characteristically, a fissured tongue has a deep, prominent groove that runs downs the center of the tongue with multiple smaller furrows (of varying depths) branching off the main body of the fissure. Consequently, a fissured tongue has been described as having a wrinkled appearance. Additionally, note the discolored tongue coating of the fissured tongue. This may be attributed to the accumulation of food particles and the growth of anaerobic bacteria in the deep fissures and furrows. The anaerobic bacteria, in turn, enzymatically break down sulfur-containing food products to emit the foul-smelling noxious smell characteristic of VSCs. For fissured tongue patients who present with malodor, the patient should mechanically debride the tongue daily with a toothbrush or tongue scraper. In this patient's case, the oral malodor resolved following 2 weeks of incorporating a tongue scraper in her daily self-care regimen.

e. **Oral Malodor and Oral Infections.** In patients with acute oral infections leading to foul-smelling purulent (pus) discharges into the mouth, oral malodors may be noticeable. This is attributed to the noxious odors emitted from the discharge of pus which contains putrefying host immune cells and bacteria. For review of acute periodontal infections that lead to foul-smelling discharge, please review Chapter 9.

f. **Oral Malodor and Acrylic Dental Appliances**
   1. The surface of an acrylic dental appliance is quite porous; the resulting surface irregularities can harbor bacteria.
   2. Patients with certain oral appliances, such as acrylic dentures, can also suffer from oral malodors.
   3. Acrylic appliances can lead to oral malodor if the patient fails to keep the appliance clean as a part of his or her daily self-care regimen.

g. **Box 37-2** summarizes the oral conditions associated with oral malodor.

Box 37-2 Oral Conditions Associated with Oral Malodor

Dental caries
Periodontal disease
Dry mouth
Tongue coatings
Purulent infections
Acrylic dental appliances

3. **The Association Between Oral Malodor and Extraoral Conditions.** While the vast majority of malodor causes have an intraoral etiology, halitosis can arise from systemic disorder(s) in some individuals. **Box 37-3** lists several extraoral conditions associated with malodor. Clinicians must cast a high degree of suspicion in any patient where malodor does not improve significantly following elimination of possible intraoral causes of oral malodor.

a. **Oral Malodor and Nose and Throat Conditions**
   1. Oral malodor can result from nose and throat conditions such as acute pharyngitis, sinusitis, and postnasal drip. Postnasal drip resulting in oral malodor can also be associated with esophagitis resulting from damage to the nasopharynx following regurgitation of acidic stomach content.
   2. In patients with tonsillitis, the deep crypts of the tonsils can accumulate food debris and bacteria that can lead to oral malodor.

b. **Oral Malodor and Diabetes**
   1. In diabetic patients whose diabetes is not controlled, chemicals called ketones can build up in the blood, leading to a potentially life-threatening condition known as diabetic ketoacidosis.
   2. Characteristically, a rotten, fruity oral malodor is a good indication of ketoacidosis.

c. **Oral Malodor and the Lung Disease.** Oral malodor has been associated with certain pulmonary (lung) conditions, including chronic bronchitis, bronchiectasis, and bronchial carcinoma.

d. **Oral Malodor and the Gastrointestinal Tract**
   1. In a very small number of patients with oral malodor, malodors can arise because of problems in the gastrointestinal tract.
   2. Among these gastrointestinal causes of oral malodor are herniated esophageal walls and gastric hernias.
   3. Malodor can also emanate from intestinal gas production where some of the sulfide compounds can be transported to the lungs and exhaled in the breath.

e. **Oral Malodor and Liver Disease.** One of the many adverse consequences of liver disease is that the liver is unable to filter out compounds that contain sulfur. Consequently, sulfur accumulates in the body to produce a distinct sulfur-like malodor known as fetor hepaticus.[14] Fetor hepaticus is a noticeable form of oral malodor that signifies liver impairment. Fetor hepaticus malodor has been described as having a rotten, sweetish, musty, and fecal-like odor; it is known as "breath of the dead." If all intraoral causes of the oral malodor have been ruled out and fetor hepaticus is suspected, the patient should be immediately referred to a hepatologist for evaluation. Another consequence of liver impairment is hyperammonemia. Note that the liver filters out toxic metabolites such as ammonia. If the liver is impaired, ammonia levels will build up in the body, thus generating an ammonia-scented oral malodor.[15]

f. **Oral Malodor and Kidney Disease.** Along with the liver, the kidneys play a major role in filtering out metabolites and chemicals from the body. Additionally, the kidneys play an additional role in excreting these metabolites/chemicals. If the kidneys are impaired, harmful chemicals, such as uric acid or ammonia, can build up in the blood, thereby contributing to a foul, ammonia-scented odor. If all intraoral causes of the malodor have been ruled out and kidney impairment is suspected, the patient should be immediately referred to a nephrologist for evaluation.

g. **Oral Malodor and Hormonal Changes**
   1. In some women, oral malodor can develop when progesterone levels are increased as part of the normal menstrual cycle.
   2. VSC levels have also been reported to increase in the expired air near the time of ovulation.

h. **Oral Malodor and Medications**
   1. Some medications have been reported to result in oral malodor. For example, the antimicrobial drug metronidazole can cause oral malodor.
   2. The drug metronidazole can also be associated with patients' perceptions of a metallic taste in the mouth, which some patients may confuse with oral malodor.

Box 37-3 Extraoral Conditions Associated with Malodor

Nose and throat conditions
Diabetes
Lung disease
Gastrointestinal tract problems
Liver disease
Kidney disease
Hormonal changes

4. **Diagnosis of Oral Malodor**
    a. **The Medical History as a Foundation for Diagnosis**
        1. The foundation for establishing a proper diagnosis of malodor is taking a thorough medical history.
        2. When a patient complains of oral malodor, some directed questions can help clarify whether the malodor originates from the oral cavity or from nonoral (extraoral) sites. Such questions can also help clarify whether the malodor is due to pathologic reasons (e.g., systemic conditions, dental caries, or periodontal disease) or nonpathologic reasons (e.g., "morning breath" or breath due to consumption of pungent, odoriferous food/drink).
        3. In patients who complain of halitosis, questions should be asked about the frequency of occurrence, the time of day the problem appears, whether others have noted the malodor, and whether the patient is aware of other symptoms such as dry mouth.
    b. **Techniques for Quantifying Halitosis**
        1. Several techniques have been recommended for quantifying malodors.[5,16,17] These techniques primarily include using organoleptic tests or the use of diagnostic tests.
        2. Organoleptic testing is the standard assessment method for oral malodor.
            a. An **organoleptic test (OLT)** is a sensory test that involves having a specially trained judge smell a patient's expired air to assess the odor. This assessment depends upon the olfactory organs of the judge.
            b. Various scoring systems have been developed. However, most are based on a numerical scale of 1 to 5, with 1 being barely noticeable odor and 5 being extremely foul odor. The typical OLT rating scale is shown in **Box 37-4**.

Box 37-4 Categories of the Organoleptic Test (OLT) Rating Scale

0 = no odor present
1 = barely noticeable odor
2 = slight but clearly noticeable odor
3 = moderate odor
4 = strong offensive odor
5 = extremely foul odor

Data from Allison VC, Katz SH. An investigation of stenches and odors for industrial purposes. *Ind Eng Chem*. 1919;11(4):336–338. doi: 10.1021/ie50112a024

            c. Because some adults have partial loss of smell acuity, testing of smell acuity is a part of the training for a judge.
            d. As part of a typical organoleptic evaluation, a trained judge would evaluate oral cavity odor, breath odor, tongue coating odor, and nasal breath odor.
        3. Diagnostic tests have been developed to detect volatile odor–causing compounds, but specially trained clinicians using their olfactory senses often are more accurate than these machines. Examples of diagnostic tests are presented below.
            a. A portable electronic volatile sulfur monitor has been used to detect hydrogen sulfide and methylmercaptan in the breath.[17] A major disadvantage of this instrument is that it does not detect unpleasant odors caused by other VSCs, such as dimethyl sulfide,[9] or other volatile nonsulfur compounds, such as putrescine or cadaverine. Because of this limitation, the sulfur monitor may underestimate malodor.[18]
            b. A portable gas chromatograph has been developed for use in typical clinical settings to measure VSCs in the breath and can identify hydrogen sulfide, methyl mercaptan, and dimethyl sulfide.[19] With these devices, the measurement of VSCs can be obtained and differentiated with samples from saliva, tongue coating, and breath. This assists in determining the origin of the malodor.[17]
            c. The electronic nose is a handheld instrument that has the ability to "smell" and produce unique fingerprints for odors.[20] This instrument has made odor analysis fast and simple. Although the electronic nose is used in a number of medical fields, few studies have been done to assess its effectiveness in clinically assessing oral malodor.[20,21]

d. A saliva incubation test has been used to detect unpleasant odors in the air immediately above the incubated saliva. This test involves collection of a saliva sample in a glass tube and incubating the saliva in the tube over a 3-hour period. A gas chromatograph can be used to detect odor-producing compounds, but an organoleptic evaluation has also been recommended as part of this test.

4. **Figure 37-4** is a flowchart that illustrates a step-by-step process for determining the origins of oral malodor.

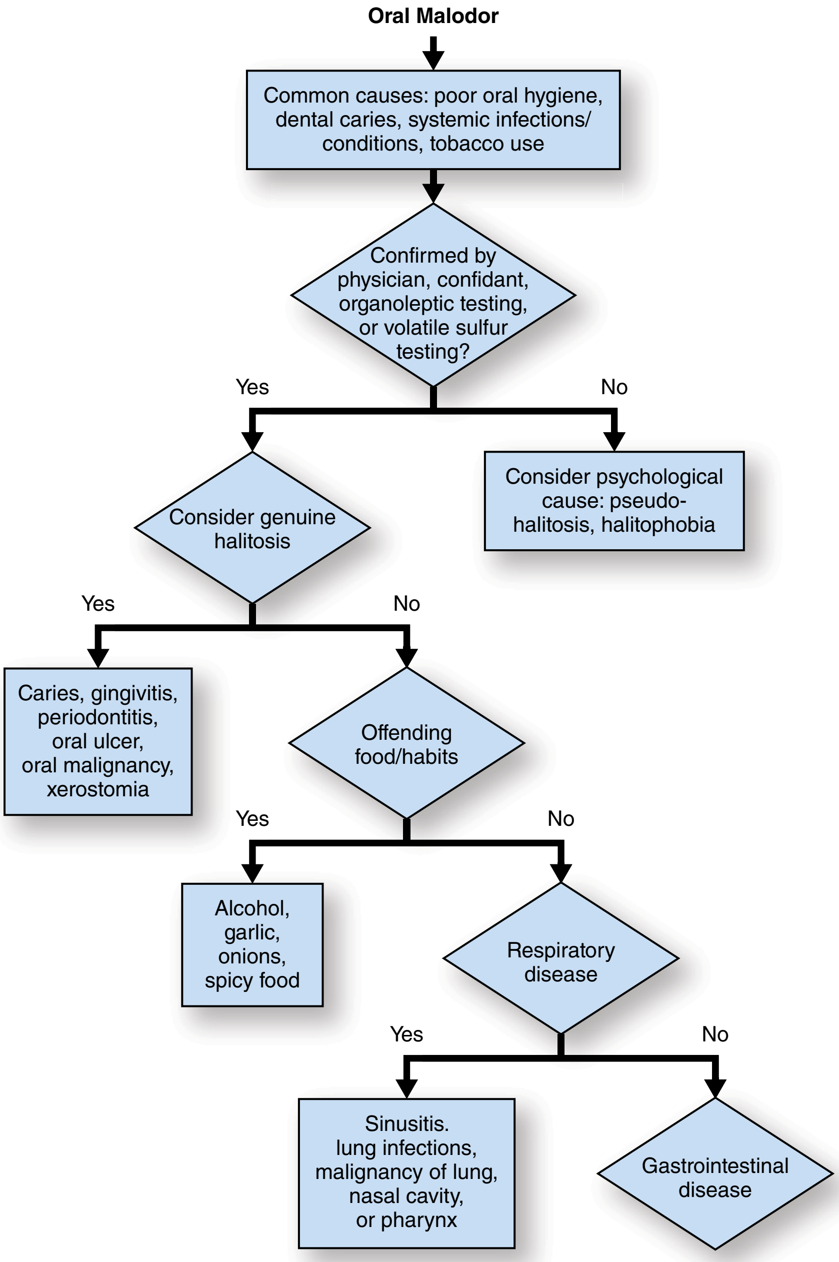

Oral Malodor
Common causes: poor oral hygiene, dental caries, systemic infections/ conditions, tobacco use
Confirmed by physician, confidant, organoleptic testing, or volatile sulfur testing?
Yes
No
Consider psychological cause: pseudo-halitosis, halitophobia
Consider genuine halitosis
Yes
No
Caries, gingivitis, periodontitis, oral ulcer, oral malignancy, xerostomia
Offending food/habits
Yes
No
Alcohol, garlic, onions, spicy food
Respiratory disease
Yes
No
Sinusitis. lung infections, malignancy of lung, nasal cavity, or pharynx
Gastrointestinal disease

Figure 37-4 Diagnosing the Source of Oral Malodor. This step-by-step process can be used to assess and determine the source of oral malodor.

Data from Kapoor U, Sharma G, Juneja M, Nagpal A. Halitosis: current concepts on etiology, diagnosis and management. *Eur J Dent*. 2016;10(2):292–300. doi:10.4103/1305-7456.178294

5. **Management of Patients with Oral Malodor.** The strategies for management of patients with malodors can be complex. But because most malodors are oral malodors, dental providers are ideally suited to manage most of these patients. At the same time, the dental team should always be on alert for any oral malodor that fails to respond to the standard management protocols. For patients where the malodor persists, a referral to the medical practitioner(s) is strongly advised. It is helpful for members of the dental team to use the following therapeutic strategies to manage and minimize the impact of oral malodor.
   a. **Reduction of Contributing Oral Factors**
      1. Patients with dental caries should have carious lesions restored and the decay problem controlled.
      2. Patients with periodontal diseases should have the periodontal disease controlled through conventional periodontal therapy.
   b. **Patient Education for Malodor Control**
      1. Regardless of what may be the cause, mechanical removal of biofilm and microorganisms—meticulous daily self-care—is the first step in control of malodor.[17,22] Various self-care techniques and regimens have already been reviewed in Chapter 26. Although it is extremely important that all patients adhere to a properly tailored self-care regimen to control both dental caries and periodontal disease, it should also be emphasized that self-care in the patient with oral malodor must include cleaning the dorsal surface of the tongue. It is estimated that approximately 60% of the VSCs originate from the dorsal surface of the tongue.[5,17,23]
      2. Cleaning the dorsal surface of the tongue can be accomplished with the use of a toothbrush or a tongue scraper.
         a. The technique for cleaning the tongue surface should include a gentle scraping with the brush or tongue scraper.
         b. The posterior part of the tongue collects the heaviest tongue coatings, and the gagging reflex can be a problem in some patients. Gagging seems easier to control with tongue scrapers than with toothbrushes. In a clinical trial comparing different tongue-cleaning methods, patients reported that they preferred the tongue scraper to the toothbrush.[24]
         c. Because patients often neglect tongue cleaning in their daily self-care routines as the result of lack of familiarity with the practice, dental hygienists should demonstrate the appropriate use of tongue scrapers.
         d. Although mechanical tongue cleaning with or without chemical intervention can reduce bacterial load on the tongue, this effect is transient, and regular tongue cleaning is required to provide a long lasting (overnight) reduction in bacterial numbers.[25]
         e. In addition, for patients with acrylic oral appliances, the appliances should be removed at night and cleaned thoroughly before replacing them the next morning.
         f. Nevertheless, the evidence supporting the use of mechanical tongue debridement to manage halitosis remains uncertain. In a recent Cochrane systematic review that included 44 trials with 1809 participants comparing an intervention to a placebo or control, Nagraj et al. found that there is a lack of evidence to support the contention that mechanical tongue cleaning is superior to no tongue cleaning, when using the OLT scores as an outcome.[26]
   c. **Chemotherapeutic Reduction.** Some chemical agents useful in plaque biofilm control have also been studied as aids to control oral malodor.[27] A Cochrane systematic review concluded that mouth rinses containing antibacterial agents such as chlorhexidine and cetylpyridinium chloride may play an important role in reducing the levels of halitosis-producing bacteria on the tongue, and mouth rinses containing chlorine dioxide and zinc can be effective in neutralization of odoriferous sulfur-containing compounds. However, a more recent Cochrane systematic review found that there is currently a lack of evidence to support the contention that chlorhexidine mouth rinses are superior to a placebo mouth rinse or no mouth rinse. Further, the same review concluded that there is currently a lack of evidence to support the contention that brushing plus cetylpyridinium mouth rinse was anymore superior to toothbrushing alone in managing haltiosis.[26] More well-designed randomized controlled trials with larger sample sizes, longer interventions, and longer follow-up periods are needed to cast more light on this matter.
   d. **Alter Volatility of Sulfur Compounds**
      1. Certain metal ions can be used to trap some of the odor-producing sulfur-containing gases before they can be released.[28–33]

    2. Zinc ions can readily bind to sulfur-containing gases, thus reducing the release of the volatile sulfur compounds.
        a. Because zinc is relatively nontoxic and does not accumulate in the body, it has been suggested for use in the control of oral malodor.[34,35]
        b. Zinc chloride has been reported to reduce volatile sulfide compounds, and zinc chloride is available in some over-the-counter products.
    3. Toothpastes containing baking soda have been shown to reduce volatile sulfur compounds. While the exact reason is unclear, this may be due to the antibacterial properties of baking soda which neutralize VSC-producing bacteria. Further, the alkaline properties of baking soda neutralizes the high acidic levels which play a role in malodor.
e. **Increase Salivary Flow**
    1. Simply increasing the volume of saliva allows for more retention of larger volumes of sulfur compounds in the saliva, thus preventing their release.
    2. Strategies for increasing saliva are discussed in the next part of this chapter.
    3. These strategies include steps such as maintaining a proper liquid intake by the patient and encouraging the patient to use sugarless candies or sugarless chewing gum to stimulate saliva production.
f. **Mask the Oral Malodor.** Masking oral malodors with products containing volatiles with pleasant smells such as rinses, sprays, or lozenges does not have a lasting effect on oral malodor, but this strategy may be employed as part of a more comprehensive treatment plan to deal with the problem of oral malodor.
g. **Involve the Patient in Odor Management**
    1. Involving the patient in the assessment of control of oral malodor is helpful.
    2. Patient involvement can be critical in motivating the patient to perform the high level of self-care needed to control this condition.
    3. Spitting some saliva into a cup, allowing it to dry a bit, then smelling the saliva can be a simple and fast way for a patient to self-assess for oral malodor. As the saliva dries, it releases volatile sulfide compounds if present.
    4. It is sometimes helpful for a patient to scrape the back of the tongue gently with a metallic spoon, then smell the utensil to assess for oral malodor; this is especially helpful if it appears that tongue coating is a major contributing factor to the unpleasant odor.
h. **Reassess Malodor Control**
    1. Reassessment following dental treatment of a patient with a complaint of oral malodor is critical.
    2. If oral malodor is not improved following conventional treatment strategies, then other sources of the odor should be investigated.
    3. If intraoral sources have been ruled out and all other attempts to control the oral malodor have failed, then the individual with persistent malodor should be referred to his or her physician for a comprehensive medical evaluation.

Section 2

Xerostomia and Hyposalivation

1. **Introduction to Xerostomia and Hyposalivation**
    a. Dry mouth is a broad disorder which encompasses two similar, yet distinct terms—xerostomia and hyposalivation. **Xerostomia** is the *subjective* sensation of dry mouth, as expressed by the patient. This should not be confused with the term **hyposalivation**, which is the *objective* reduction in salivary flow rate verified by actual measurements of stimulated or unstimulated salivary flow.[36] As such, one can think of xerostomia as being the *symptom* of dry mouth while hyposalivation is a *sign* of dry mouth. Of note, a patient with xerostomia may not necessarily exhibit the objective signs of hyposalivation; rather, his/her symptom may be secondary to qualitative and/or quantitative changes in saliva composition.[37,38] Hence, xerostomia patients may not present with a demonstrable change in the amount of saliva to warrant a diagnosis of hyposalivation. Conversely, a patient who presents with the objective signs of dry

mouth (hyposalivation) may not necessarily complain of the subjective sensation of having a dry mouth (xerostomia).

1. Xerostomia is a common patient complaint. Epidemiologic studies estimate that xerostomia affects about 1 in every 4 to 5 adults, with the prevalence of dry mouth increasing with age.[39–43]
2. Xerostomia and hyposalivation are known side effects of many medications. Since older adults tend to take more prescribed medications, it should not be surprising that dry mouth is a common complaint among this age group.
3. Reduced salivary flow may be associated with decreased salivary gland function. But it may also be present in patients where the salivary glands are capable of producing an adequate flow of saliva under the right conditions.

b. **Importance of Saliva for Quality of Life**

1. Saliva has many functions in addition to moistening and lubrication of the entire mouth.
    a. Saliva is critical for its antimicrobial properties, in buffering the pH of the oral cavity, for removal of food debris, in remineralization of teeth, and for maintaining the health of the oral mucosa.
    b. In addition, saliva plays an important role in chewing, taste, swallowing, digestion, and speech.
2. Reduced salivary flow decreases the oral pH, which creates an environment favorable for the growth of cariogenic bacteria, thereby significantly increasing the risk for dental caries. Patients with dry mouth will need to be carefully managed to reduce the risk of dental caries.
3. As discussed, dry mouth may be associated with halitosis because it creates a favorable ecological oral niche for the growth of VSC-producing anaerobic bacteria.
4. Hyposalivation can also be an indication of several systemic diseases (to be discussed later in this chapter) that promote oral infections, such as candidiasis or dental caries.
5. Partially or fully edentulous patients with xerostomia and/or hyposalivation may experience pain from denture irritation, mucosal ulcerations, and fungal infections, in addition to loss of denture retention.[44,45] **Figure 37-5** is a case of an individual who complained of xerostomia and was later diagnosed as suffering from hyposalivation which was verified by sialometry (to be discussed later in this section). Note how dry the mucosal tissues covering the palate, floor of the mouth, edentulous ridges, and vestibular regions appear.
6. **Box 37-5** summarizes some of the important functions of saliva.

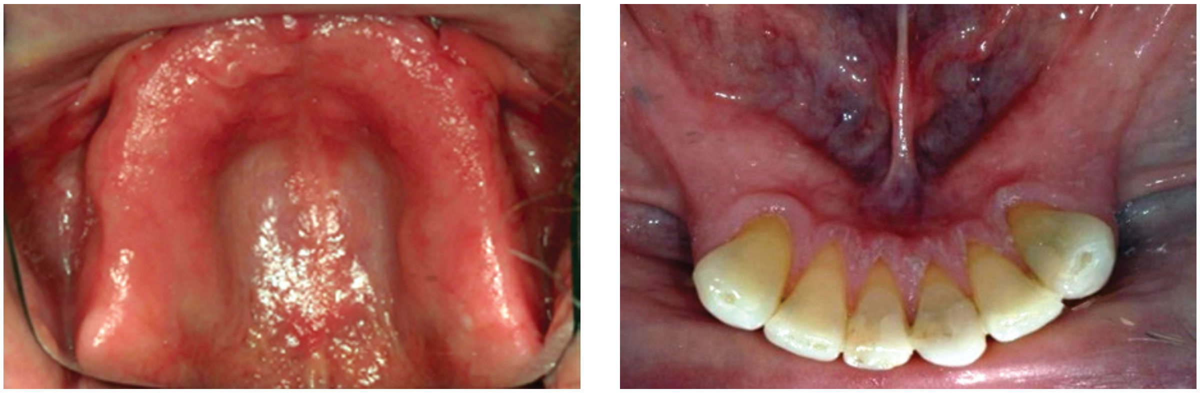
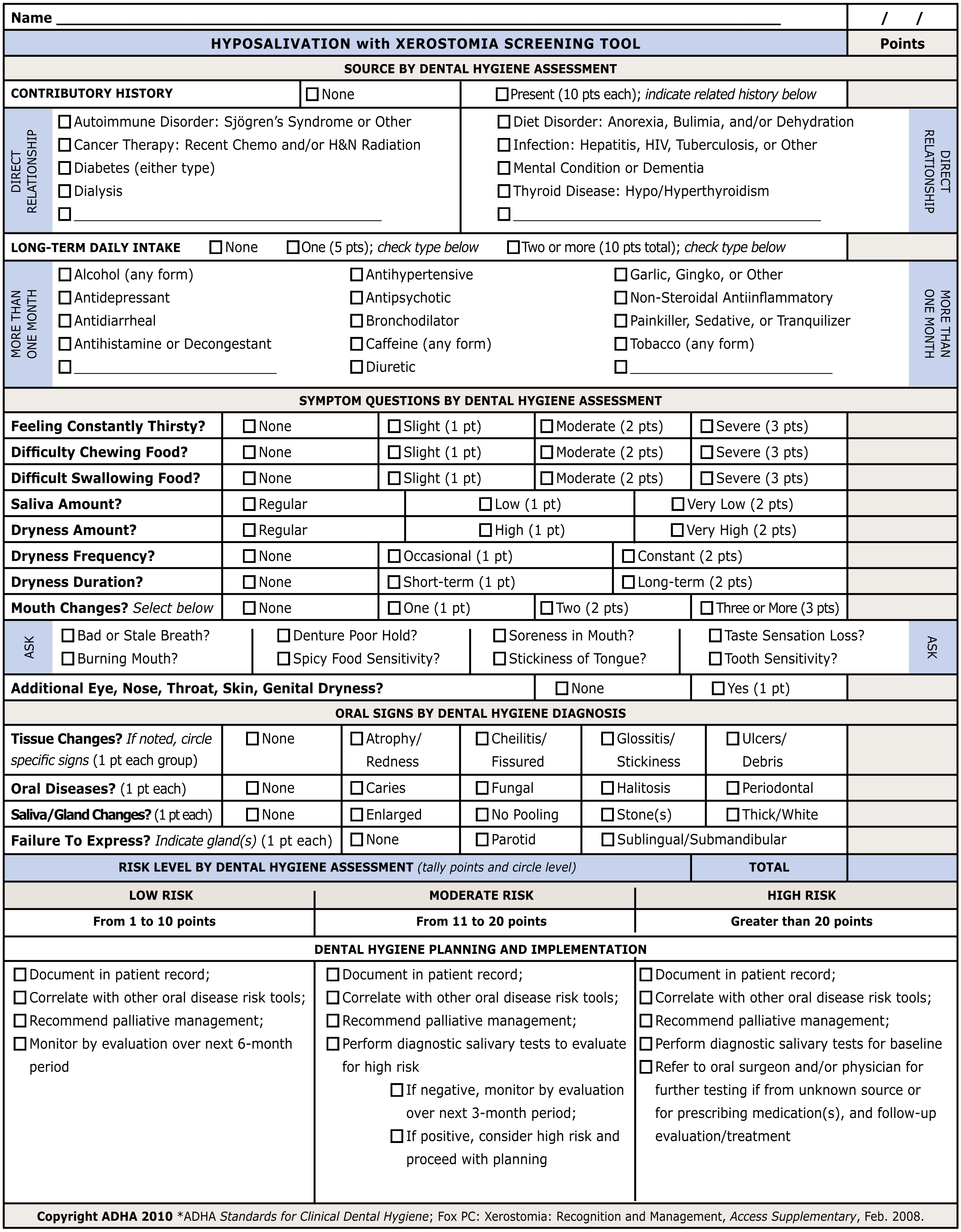


Figure 37-5 Implications of Xerostomia and Hyposalivation in a Denture-Wearing Individual. A 50-year-old denture-wearing individual presents with the following chief complaint: "The roof of my mouth feels so dry that my upper plate no longer fit comfortably." Sialometry later confirmed a diagnosis of hyposalivation. **A.** Clinical photo of the maxilla. Note the dry mucosal tissues and the complete lack of saliva coating the denture-bearing palate and edentulous ridges. Additionally, note the inflamed, irritated appearance of the mucosal tissues. **B.** Clinical photo of the mandible of the same individual. Although the mucosal tissues do not appear erythematous, the lack of pooled saliva in the floor of the mouth and the buccal vestibule is consistent with the patient's chief complaint.

Box 37-5 Functions of Saliva

Moistening and lubrication of the mouth
Antimicrobial properties
Maintaining pH of the oral cavity
Removal of food debris
Remineralization of teeth
Maintaining the health of oral mucosa
Aid in chewing
Enhancing taste
Aid in swallowing
Aid in digestion

2. **Overview of Causes of Xerostomia and Hyposalivation**
    a. **Local Conditions Associated with Xerostomia and Hyposalivation.** Several local and systemic causes may contribute to xerostomia and hyposalivation. The American Dental Hygienists' Association (ADHA) has developed a useful screening tool for the identification and management of xerostomia and hyposalivation. This screen tool is shown in **Figure 37-6**.
        1. As a general rule, diminished salivary flow resulting in dry mouth is due to systemic causes, not to intraoral problems. One exception to this general rule is mouth breathing.
        2. **Mouth breathing** is defined as inhaling and exhaling through the mouth rather than through the nose.
            a. Patients normally breathe through the nose when resting or while performing light exercise; they breathe through the nose and mouth only during more vigorous exercise.
            b. Continuous mouth breathing can cause a drying of the oral mucosal tissues which lead to a patient complaint of dry mouth.
        3. Research suggests that the use of cannabis increases the risk of hyposalivation and candidiasis.[46] With the increasing prevalence of cannabis use, dental health care providers should be aware of cannabis-associated oral side effects. To review other cannabis-associated oral side effects, the reader is asked to refer to Chapter 17, Section 5.
    b. **Systemic Conditions Associated with Dry Mouth**
        1. **Dry Mouth Associated with Sjögren's Syndrome**
            a. Sjögren's syndrome (pronounced "show-grins") is the most common systemic condition associated with dry mouth.[47–49]
            b. **Sjögren's syndrome** is an inflammatory autoimmune disease that results in a general dryness caused by damage to the salivary and other similar glands, such as tear glands.
                1. This syndrome occurs most frequently in postmenopausal women.
                2. One of the characteristics of the underlying pathology of this syndrome is lymphocytic infiltration of salivary as well as tear glands, which impairs the normal functions of these glands.
                3. A variety of other patient complaints are associated with Sjögren's syndrome; these can include symptoms such as blurred vision, oral soreness, smell alterations, taste alterations, fissures on the tongue and lips, fatigue, and dry nasal passages.
                4. There is no known cure for Sjögren's syndrome, so therapy is directed at alleviating the symptoms.
        2. **Dry Mouth as a Medication Side Effect**
            a. More than 1800 drugs have been reported to be associated with dry mouth. Further, the most frequently prescribed medications in the United States may play a contributing role in dry mouth.
            b. For example, medications used to treat common ailments such as high blood pressure, anxiety, allergies, and weight loss can contribute to dry mouth.
            c. In addition, patients taking multiple medications simultaneously to treat one or more conditions (known as **polypharmacy**) are at increased risk of developing dry mouth. When this occurs, this condition is referred to as **medication-induced xerostomia and hyposalivation**.



Name ____	/ /
HYPOSALIVATION with XEROSTOMIA SCREENING TOOL	**Points**

SOURCE BY DENTAL HYGIENE ASSESSMENT

CONTRIBUTORY HISTORY	☐ None	☐ Present (10 pts each); *indicate related history below*	

DIRECT RELATIONSHIP

- ☐ Autoimmune Disorder: Sjögren's Syndrome or Other
- ☐ Cancer Therapy: Recent Chemo and/or H&N Radiation
- ☐ Diabetes (either type)
- ☐ Dialysis
- ☐ ________________________________
- ☐ Diet Disorder: Anorexia, Bulimia, and/or Dehydration
- ☐ Infection: Hepatitis, HIV, Tuberculosis, or Other
- ☐ Mental Condition or Dementia
- ☐ Thyroid Disease: Hypo/Hyperthyroidism
- ☐ ________________________________

DIRECT RELATIONSHIP

LONG-TERM DAILY INTAKE	☐ None	☐ One (5 pts); *check type below*	☐ Two or more (10 pts total); *check type below*	

MORE THAN ONE MONTH

- ☐ Alcohol (any form)
- ☐ Antidepressant
- ☐ Antidiarrheal
- ☐ Antihistamine or Decongestant
- ☐ ____________________
- ☐ Antihypertensive
- ☐ Antipsychotic
- ☐ Bronchodilator
- ☐ Caffeine (any form)
- ☐ Diuretic
- ☐ Garlic, Gingko, or Other
- ☐ Non-Steroidal Antiinflammatory
- ☐ Painkiller, Sedative, or Tranquilizer
- ☐ Tobacco (any form)
- ☐ ____________________

MORE THAN ONE MONTH

SYMPTOM QUESTIONS BY DENTAL HYGIENE ASSESSMENT

Feeling Constantly Thirsty?	☐ None	☐ Slight (1 pt)	☐ Moderate (2 pts)	☐ Severe (3 pts)	
Difficulty Chewing Food?	☐ None	☐ Slight (1 pt)	☐ Moderate (2 pts)	☐ Severe (3 pts)	
Difficult Swallowing Food?	☐ None	☐ Slight (1 pt)	☐ Moderate (2 pts)	☐ Severe (3 pts)	
Saliva Amount?	☐ Regular	☐ Low (1 pt)	☐ Very Low (2 pts)		
Dryness Amount?	☐ Regular	☐ High (1 pt)	☐ Very High (2 pts)		
Dryness Frequency?	☐ None	☐ Occasional (1 pt)	☐ Constant (2 pts)		
Dryness Duration?	☐ None	☐ Short-term (1 pt)	☐ Long-term (2 pts)		
Mouth Changes? *Select below*	☐ None	☐ One (1 pt)	☐ Two (2 pts)	☐ Three or More (3 pts)	

ASK

- ☐ Bad or Stale Breath?
- ☐ Burning Mouth?
- ☐ Denture Poor Hold?
- ☐ Spicy Food Sensitivity?
- ☐ Soreness in Mouth?
- ☐ Stickiness of Tongue?
- ☐ Taste Sensation Loss?
- ☐ Tooth Sensitivity?

ASK

Additional Eye, Nose, Throat, Skin, Genital Dryness?	☐ None	☐ Yes (1 pt)	

ORAL SIGNS BY DENTAL HYGIENE DIAGNOSIS

Tissue Changes? *If noted, circle specific signs* (1 pt each group)	☐ None	☐ Atrophy/ Redness	☐ Cheilitis/ Fissured	☐ Glossitis/ Stickiness	☐ Ulcers/ Debris	
Oral Diseases? (1 pt each)	☐ None	☐ Caries	☐ Fungal	☐ Halitosis	☐ Periodontal	
Saliva/Gland Changes? (1 pt each)	☐ None	☐ Enlarged	☐ No Pooling	☐ Stone(s)	☐ Thick/White	
Failure To Express? *Indicate gland(s)* (1 pt each)	☐ None	☐ Parotid	☐ Sublingual/Submandibular			

RISK LEVEL BY DENTAL HYGIENE ASSESSMENT *(tally points and circle level)*	**TOTAL**	

LOW RISK	**MODERATE RISK**	**HIGH RISK**
From 1 to 10 points	**From 11 to 20 points**	**Greater than 20 points**

DENTAL HYGIENE PLANNING AND IMPLEMENTATION

☐ Document in patient record; ☐ Correlate with other oral disease risk tools; ☐ Recommend palliative management; ☐ Monitor by evaluation over next 6-month period	☐ Document in patient record; ☐ Correlate with other oral disease risk tools; ☐ Recommend palliative management; ☐ Perform diagnostic salivary tests to evaluate for high risk ☐ If negative, monitor by evaluation over next 3-month period; ☐ If positive, consider high risk and proceed with planning	☐ Document in patient record; ☐ Correlate with other oral disease risk tools; ☐ Recommend palliative management; ☐ Perform diagnostic salivary tests for baseline ☐ Refer to oral surgeon and/or physician for further testing if from unknown source or for prescribing medication(s), and follow-up evaluation/treatment

Copyright ADHA 2010 *ADHA *Standards for Clinical Dental Hygiene*; Fox PC: Xerostomia: Recognition and Management, *Access Supplementary*, Feb. 2008.

Figure 37-6 **Xerostomia and Hyposalivation Screening Tool.**

Copyright ADHA 2010. ADHA Standards for Clinical Dental Hygiene; Fox PC: Xerostomia: Recognition and Management, Access Supplementary, Feb. 2008.

   d. Medication-induced xerostomia and hyposalivation is often seen in older/geriatric patients who tend to take more medications simultaneously. As medicine and pharmacology make inroads in advancing our life expectancy, dental practitioners will need to be capable of recognizing and managing medication-induced xerostomia and hyposalivation.

3. **Dry Mouth Associated with Cancer Therapy**
   a. Many head and neck cancer patients who are either undergoing or have already completed radiation therapy to the head and neck region often complain of xerostomia. (**Figure 37-7**). Radiation therapy uses high-dose radiation to destroy cancer cells while minimizing as much harm as possible to normal cells.
   b. Radiation therapy can destroy the cells of salivary glands, which in turn leads to reduced salivary secretion, xerostomia, oral discomfort, altered taste, difficult with speaking, swallowing, chewing, and increased risk of dental diseases, such as dental caries, periodontal disease, and oral candidiasis.[50]
   c. As a result of radiation therapy, there can also be fibrosis of the salivary glands, which leads to radiotherapy induced hyposalivation.
   d. The degree of permanent dry mouth following radiation therapy depends on the precise volume of salivary glands exposed to the radiation as well as on the radiation dose used in the cancer treatment.
   e. Additionally, some cancer chemotherapeutic agents can cause salivary gland hypofunction which decreases the salivary flow.

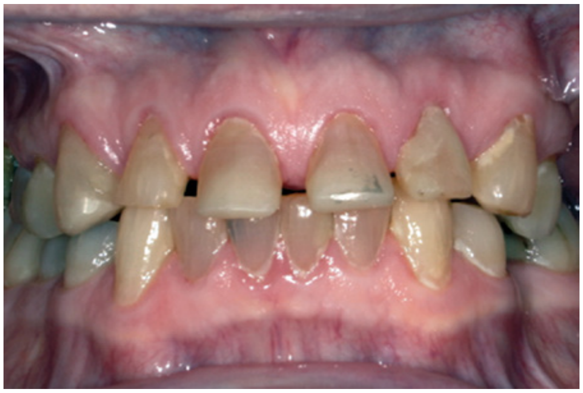


Figure 37-7 **Implications of Radiation Therapy on Xerostomia and Hyposalivation**. Two months prior to obtaining this photo, this individual completed a 6-week course of head and neck radiation to treat pharyngeal cancer. At the time of obtaining the photo, the patient presented to her dentist with the chief complaint: "My mouth feels dry since I started radiation." Visually, note the pronounced dryness of the mucosal tissues.

4. **Dry Mouth Associated with Aging**
   a. As patients age, there is a tendency to produce less saliva.
   b. In general, a reduction in salivary output associated with aging may not be dramatic enough to result in a complaint of xerostomia. But when combined with other factors, such as the effects of medications, the subjective sensation of xerostomia may become more noticeable to the patient.[51–53]
5. **Dry Mouth Associated with HIV Infection**
   a. In some young patients infected with HIV, there can be an associated salivary gland disease that results in the subjective feeling of xerostomia superimposed with hyposalivation.
   b. This dry mouth usually follows enlargement of the parotid salivary glands and less frequently the submandibular glands.

6. **Dry Mouth Associated with Other Systemic Conditions**
    a. An array of other systemic conditions can lead to patient's complaining of xerostomia with objective evidence of hyposalivation.
    b. Systemic diseases that have been associated with dry mouth include sarcoidosis, rheumatoid arthritis, lupus erythematosus, nephritis, scleroderma, diabetes mellitus, hypertension, cystic fibrosis, and endocrine disorders.
    c. In addition, other conditions associated with dry mouth include nutritional deficiencies, methamphetamine use, depression, anxiety, stress, and fear.
    d. Dry mouth is often exacerbated by activities such as smoking or drinking alcohol.

3. **Diagnosis of Hyposalivation in a Dental Setting.** Diagnosing hyposalivation is much easier than identifying the underlying cause. The diagnosis of hyposalivation in a dental office is based on evidence obtained from the three primary clinical steps outlined below.
    a. **First Diagnostic Step: Perform a Thorough Patient History**
        1. Patients with hyposalivation may complain of difficulty in eating, speaking, swallowing, or even wearing dentures.
        2. A typical complaint would be that dry foods, such as crackers, are difficult to chew and swallow.
        3. **Box 37-6** outlines some typical patient complaints related to reduced salivary flow.
    b. **Second Diagnostic Step: Perform a Comprehensive Examination of the Oral Cavity**
        1. During examination of the oral cavity in a patient with hyposalivation, the oral mucosa may appear dry and sticky; a wooden tongue depressor placed against the inner surface of the cheek may tend to stick to the mucosa.
        2. Oral examination may reveal dental caries, parotid gland enlargement, inflammation and fissuring of the lips, inflammation or ulcers of the tongue or cheek mucosa, oral candidiasis, or halitosis.
        3. There may be very little or no pooled saliva in the floor of the mouth.
        4. Along with affecting the *quantity* of saliva, hyposalivation alters the *quality* of saliva. In conditions of health, saliva has a watery, thin, clear consistency. However, hyposalivation patients present with saliva that has a thick, ropey, sticky consistency.
            a. This is a common postoperative sequela of patients exposed to head and neck radiation or chemotherapy for cancer treatment. Both types of therapy destroy viable, functioning salivary gland cells, thereby diminishing the output of protective salivary macromolecules, enzymes, and proteins.
            b. Another cause is dehydration. In dehydrated patients, the saliva has a thick, ropey, sticky consistency because of the lower-than-normal amount of water in the saliva.
            c. Lastly, smokers who complain of xerostomia may exhibit thick, ropey saliva because smoking has its primary effects on the parotid gland, which produces watery (serous) saliva. To compensate for this, the body's submandibular and sublingual glands offset the decreased output of the parotid gland by producing saliva, albeit mucous-rich saliva.[54] Moreover, smoking induces the body's mucous glands to produce higher-than-normal mucous levels, which ends up mixing with the saliva.

Box 37-6 Oral Complaints Associated with Reduced Salivary Flow Reported by Patients

Difficulty in eating dry foods
Problems with taste acuity
Difficulty with swallowing
Sensitivity of oral mucosa to food
Soreness of the tongue
Increased tooth decay
Difficulty in wearing dentures

    c. **Third Diagnostic Step: Measure the Salivary Flow Rate**
        1. In-chair tests can be utilized to determine the salivary flow rate; measuring the salivary flow rate is referred to as **sialometry**.

2. Sialometry usually involves placing a collection device over the parotid gland duct orifice or the submandibular/sublingual gland duct orifices and measuring saliva flow per minute with glands stimulated and with glands unstimulated.
3. A stimulated salivary flow rate of 0.5-0.7 mL/min and an unstimulated salivary flow rate of less than or equal to 0.1 mL/min is diagnostic of hyposalivation which is objective proof that confirms the patient's subjective feeling of dry mouth (xerostomia).
4. **Box 37-7** shows the normal stimulated/unstimulated salivary flow rate vs. the stimulated/unstimulated salivary flow rates that are indicative of hyposalivation.

Box 37-7 Diagnostic Information: Normal Salivary Flow Rates vs. Hyposalivation Flow Rates

	Normal Rate	Hyposalivation Rate
Stimulated salivary flow	1-2 mL/min	0.5-0.7 mL/min
Unstimulated salivary flow	0.3-0.5 mL/min	Less than or equal to 0.1 mL/min

d. **Identifying the Underlying Cause of Dry Mouth: An Interdisciplinary Approach**
   1. Once the diagnosis of hyposalivation is made, the actual identification of the underlying cause(s) of dry mouth should involve the input of a variety of health care providers. In addition to dentists, specialists in oral medicine, rheumatology, and ophthalmology (if the eyes are involved) should be included.
   2. The type of information gathered by health care practitioners to identify possible causes of dry mouth is outlined below, but the details of this type of diagnosis are beyond the scope of this book.
      a. Complete medical/dental history and clinical examination.
      b. Salivary tests to measure both the flow rate of the saliva and the condition of the salivary glands themselves.
      c. Tests on the eyes.
      d. Special blood tests, such as for antibodies associated with Sjögren's syndrome, for rheumatoid factor, and for immunoglobulins.
   3. Even prior to a definitive diagnosis, members of the healthcare team can implement strategies to manage a patient with reduced salivary flow. Strategies for managing patients with dry mouth are outlined in the next section.

4. **Dental Management of Patients with Dry Mouth**
   a. **Alleviate the Symptoms of Dry Mouth.** As already discussed, when possible, the management of patients with dry mouth should include the identification of the underlying cause of the decreased salivary flow.
      1. In the event that steps can be taken to minimize the effect of the underlying cause, this should be done; frequently, these steps will be in the hands of the patient's physician.
      2. For many patients, however, little can be done to alter the underlying cause of the dry mouth.
         a. For example, if the underlying cause is a side effect of medications taken to maintain the health of the patient, the patient must continue to take these medications, even though this may mean that the hyposalivation will persist.
         b. Hence, in many patients with dry mouth, management should be focused on palliative strategies to minimize the side effects and to improve the patient's quality of life.
      3. Strategies for dental management of patients with xerostomia and hyposalivation depend upon the severity of the disorder and may be patient-specific.[55,56]
   b. **Involve the Patient in the Management of Dry Mouth**
      1. Members of the healthcare team should make every effort to involve the patient in actively managing the condition.

   2. The collaborative effort should focus on identifying medications and/or behaviors that could be contributing to the patient's dry mouth.
   3. The patient should be encouraged to maintain his or her daily self-care regimen, and report any suspicious new findings to the healthcare team promptly. For example, patients should be instructed to watch for areas of redness or patches on the oral tissues that might indicate infection.
   4. Patients with hyposalivation must faithfully adhere to a self-care regimen to minimize potential side effects such as dental caries.

c. **Recommend Palliative Products.** Make the patient aware of over-the-counter palliative products that can minimize the subjective symptoms of xerostomia.
   1. Over-the-counter products are available to support patients with a dry mouth; these products include toothpastes, mouth rinses, sprays, gels, gums, and liquids.[57]
      a. An example of these products is the Biotene® brand of products; **Box 37-8** lists some of these products.
      b. A quick search of the internet will reveal a variety of over-the-counter products that can be helpful in some patients, and more products are available each year.
   2. Use of toothpastes and mouth rinses specially designed for individuals with dry mouth is recommended. Sodium lauryl sulfate—a foaming agent commonly found in toothpastes and mouth rinses—can further damage already fragile oral soft tissues. The use of sodium lauryl sulfate–containing products should be strongly discouraged for patients with xerostomia.[58,59]

Box 37-8 Examples of Biotene® Brand of Products Formulated for Patients with Dry Mouth

Biotene® Dry Mouth Toothpaste
Biotene® Gentle Mouthwash
Biotene® Dry Mouth Gum
Biotene® Dry Mouth Kit

d. Instruct the patient in simple techniques for increasing existing salivary flow. Some relatively simple techniques for increasing salivary flow (when there is some function of the salivary glands remaining) are outlined below.
   1. Eating foods that require mastication can stimulate salivary flow; examples of these types of foods include carrots or celery.
   2. Chewing sugarless gum can increase the flow of saliva.
   3. In addition, some patients benefit from using acid-tasting sugarless candy (as recommended for diabetics).
   4. For most individuals, daytime dryness is easily managed, however oral dryness is much more problematic at night while sleeping. One product, XyliMelts—a self-adhering, slowly dissolving disc that time-releases xylitol, a lubricant, and a humectant—is used to reduce oral dryness. XyliMelt discs adhere to tooth surfaces and can be used during sleep. In a 15-subject study, Burgess and Lee found that use of XyliMelts discs may be an effective strategy for managing oral dryness that occurs during sleep.[60]

e. Replace lost saliva with saliva substitutes. Over-the-counter saliva substitutes specifically formulated for patients with dry mouth are available as solutions, spray, chewing gum, and gels.[61,62]
   1. Saliva substitutes can replace some moisture in the oral cavity and lubricate the mouth.[63]
   2. Note that saliva substitutes are formulated to replace natural saliva, but they do not stimulate salivary production.
   3. Examples of commercially available saliva substitutes containing carboxymethyl cellulose and hydroxyethyl cellulose that can be used in dry mouth patients are Salivart, Oralube, and Xerolube.

f. Make the patient aware of simple behaviors to minimize the effects of dry mouth. A variety of suggestions can be made to patients to minimize the effects of dry mouth. **Box 37-9** lists some suggestions for individuals suffering from this disorder.

Box 37-9 Useful Suggestions for Patients with Dry Mouth

- Take frequent sips of water throughout the day or suck on ice chips.
- Use a humidifier in your bedroom at night.
- Chew your food slowly and thoroughly, and sip water with it before swallowing.
- Brush and floss your teeth thoroughly at least twice each day.
- Select and use a fluoride-containing toothpaste daily. (Your dentist may even prescribe a special fluoride containing toothpaste.)
- Clean the surface of your tongue daily.
- Restrict your intake of sugar containing foods and beverages.
- Use time-release xylitol lozenges (xylitol has properties that stimulate saliva). These products last for an hour or longer and some of these products can be used overnight.
- Chew gum that contains no sugar, but that does contain xylitol.
- Avoid your intake of spicy, salty, or very acidic foods.
- Limit your between meal snacks.
- Do not smoke.
- Avoid intake of alcohol-containing products; many mouthrinses contain alcohol.
- Avoid intake of caffeine-containing drinks.
- Avoid products containing the chemical sodium lauryl sulfate.
- Do not wear dentures when sleeping.
- Visit your dentist at least 3 times a year.

g. Consider the use of salivary stimulants. Several pharmacologic stimulants can be prescribed to stimulate salivary glands to increase saliva production.
    1. **Pilocarpine (Salagen™)**
        a. Pilocarpine is a prescription cholinergic parasympathomimetic agent that can stimulate functioning salivary glands.
        b. This drug has been used in some patients with dry mouth caused by Sjögren's syndrome and in patients with hyposalivation caused by radiation treatment for cancer.[64,65]
        c. Research has shown that pilocarpine can significantly decrease dryness of the mouth and eyes in patients with Sjögren's syndrome.
        d. Pilocarpine is available in an ophthalmic solution and gel as well as the oral tablet.
        e. This drug does have side effects; the most common side effects are increased sweating and gastrointestinal intolerance.
        f. Hypotension, rhinitis, diarrhea, and visual disturbances have also been reported with the use of this drug.
    2. **Cevimeline (Evoxac™)**
        a. Cevimeline is a prescription cholinergic agonist that can lead to an increase in exocrine gland secretions, including saliva.
        b. Cevimeline has been used for the treatment of symptoms of dry mouth in patients with Sjögren's syndrome.[66,67]
    3. **Chemical stimulants.** Several products contain citric acid, which can stimulate salivary flow. Mouth-Kote and Optimoist are examples of two such products.

h. **Perform a Comprehensive Oral Exam Frequently**
    1. Because dental caries can develop rapidly in patients with dry mouth, and because these patients tend to develop infections that can affect the oral mucosa, they should be seen on relatively short recall intervals by members of the dental team.
    2. Periodic recalls should include monitoring the salivary flow rate, and a comprehensive oral examination which includes updating radiographs when necessary.

Chapter Summary Statement

The focus of the first half of this the chapter was on oral malodors. An oral malodor usually reflects an underlying oral pathology (such as caries or periodontal disease) that must be treated as part of the management of patients with this condition. The chemical origin of oral malodor is primarily from volatile sulfur compounds, but other non-sulfur compounds have also been implicated in contributing to malodor. Typically, therapy is directed toward eliminating both the bacteria and the volatile, noxious compounds that lead to the unpleasant odors. Equally important is the possibility that some oral malodors may have extraoral origins, such as an underlying systemic disease, which should be comanaged with the patient's physician. Other odors may have psychologic or neurologic origins, such as pseudo-halitosis and halitophobia, which should be comanaged with medical practitioners and mental care experts.

The focus of the second half of this chapter was to distinguish xerostomia from hyposalivation. Recognition of xerostomia and hyposalivation in a dental setting is not difficult, but identifying the underlying cause can be challenging. In many patients, the underlying cause cannot be corrected, so management of many patients suffering from either xerostomia or hyposalivation consists of mitigating the symptoms. Various dental management strategies are available to be implemented, but the precise treatment approach should be tailored to the individual patient's needs.

Section 3

Focus on Patients

Clinical Patient Care

CASE 1

A patient new to your dental team has a chief complaint of bad breath. Your dental team decides to treat this patient for oral malodor and reassess the results following treatment. What steps should be taken by the various members of your dental team to control the patient's oral malodor?

CASE 2

During a routine recall visit, an elderly patient taking several critical medications complains to you of dry mouth. Make a list of suggestions that you can give this patient to help minimize the effects of the patient's xerostomia.

Ethical Dilemma

Ava has been a patient in your practice for the last 15 years. She is a 48-year-old woman who sees you every 6 months for periodontal maintenance. When you escort her from the waiting room to your operatory, you notice that she is quite thin, appears weak and frail and looks to be wearing a wig. As you review her medical history, she tells you that she was diagnosed with breast cancer right after her last appointment with you. She has been undergoing intensive chemotherapy and radiation, and in fact has been using medical marijuana to assist with the painful side effects.

During your intraoral examination, you notice that Ava has extremely bad halitosis and xerostomia. These conditions have not been noted in her chart in the past. You realize that she is unable to thoroughly clean her oral cavity due to her oral mouth sores, most likely from her cancer treatments.

You are aware of research that links the use of cannabis with increased risk of xerostomia. You, however, disagree with the use of marijuana for any purpose. As a result, you are unsure how to best educate Ava.

1. How would you discuss oral malodor/management with Ava?
2. What options could you discuss for management of xerostomia?
3. Are there ethical principles that are in conflict in this dilemma?

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CHAPTER 38

Patient Cases: Radiographic Analysis

- Case 1
- Case 2
- Case 3
- Case 4
- Case 5
- Case 6

LEARNING OBJECTIVE

- Apply concepts from the chapters in this book to complete a radiographic analysis of Cases 1 to 6.

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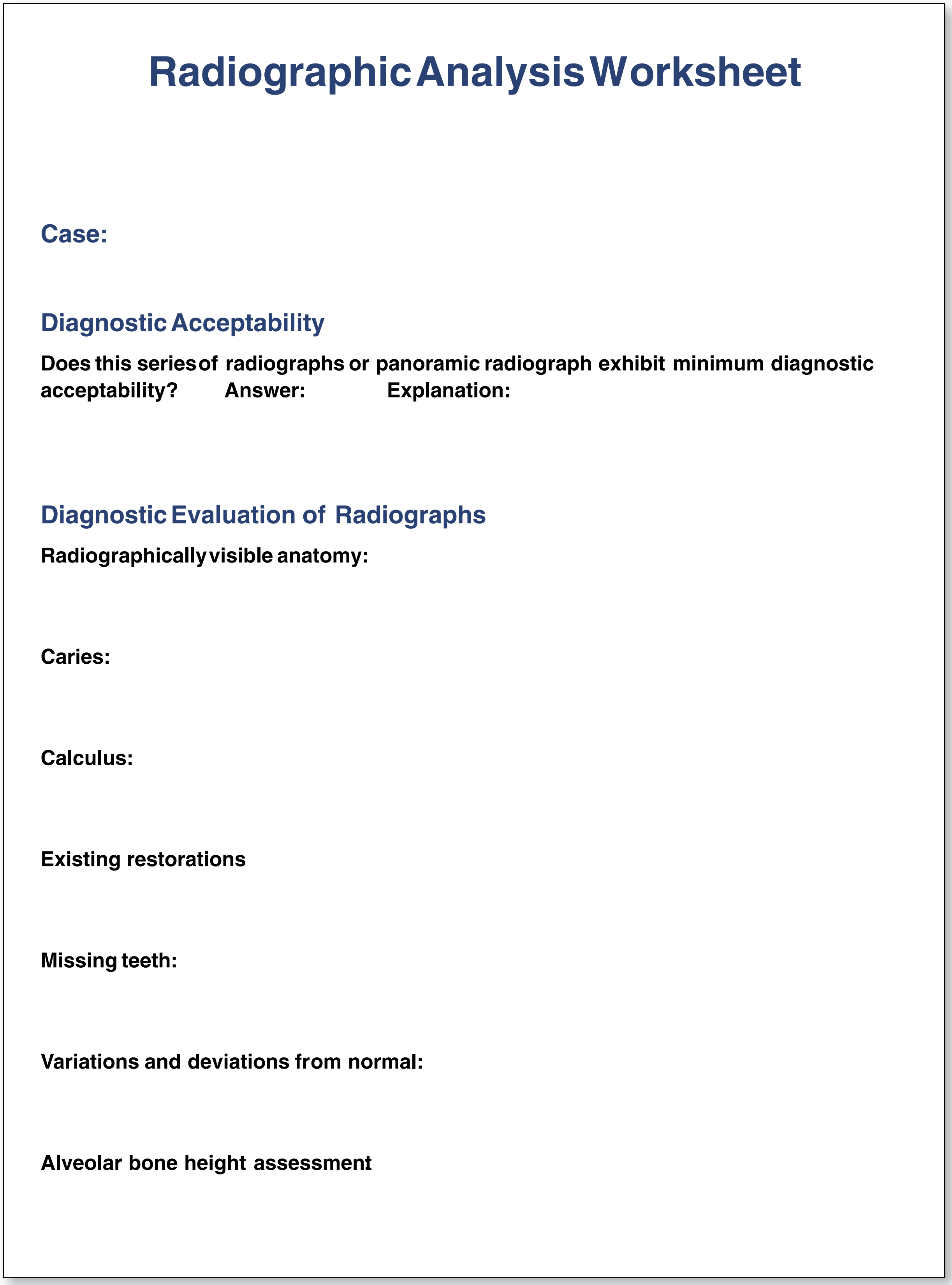
Directions

- Practice your skills in radiographic analysis by examining each patient case.
- Using sheets of notebook paper, recreate the **Radiographic Analysis Worksheet** shown on the facing page.
- Complete a radiographic analysis for each case.



Radiographic Analysis Worksheet

Case:

Diagnostic Acceptability

Does this series of radiographs or panoramic radiograph exhibit minimum diagnostic acceptability? Answer: Explanation:

Diagnostic Evaluation of Radiographs

Radiographically visible anatomy:

Caries:

Calculus:

Existing restorations

Missing teeth:

Variations and deviations from normal:

Alveolar bone height assessment

Figure 38-1 Sample Radiographic Analysis Worksheet.

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Section 1: Case 1

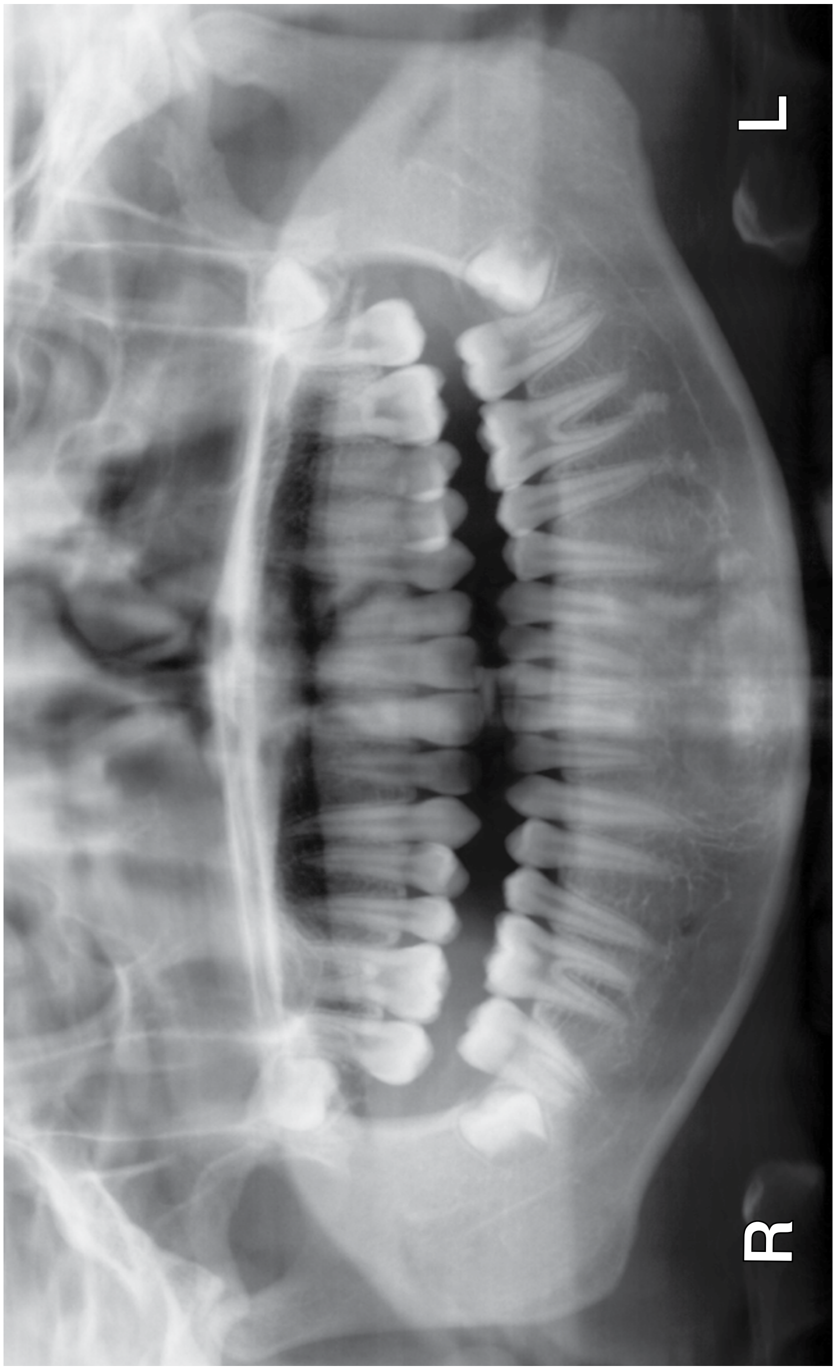

Figure 38-2 Panoramic Radiograph for Case 1.

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Section 2: Case 2

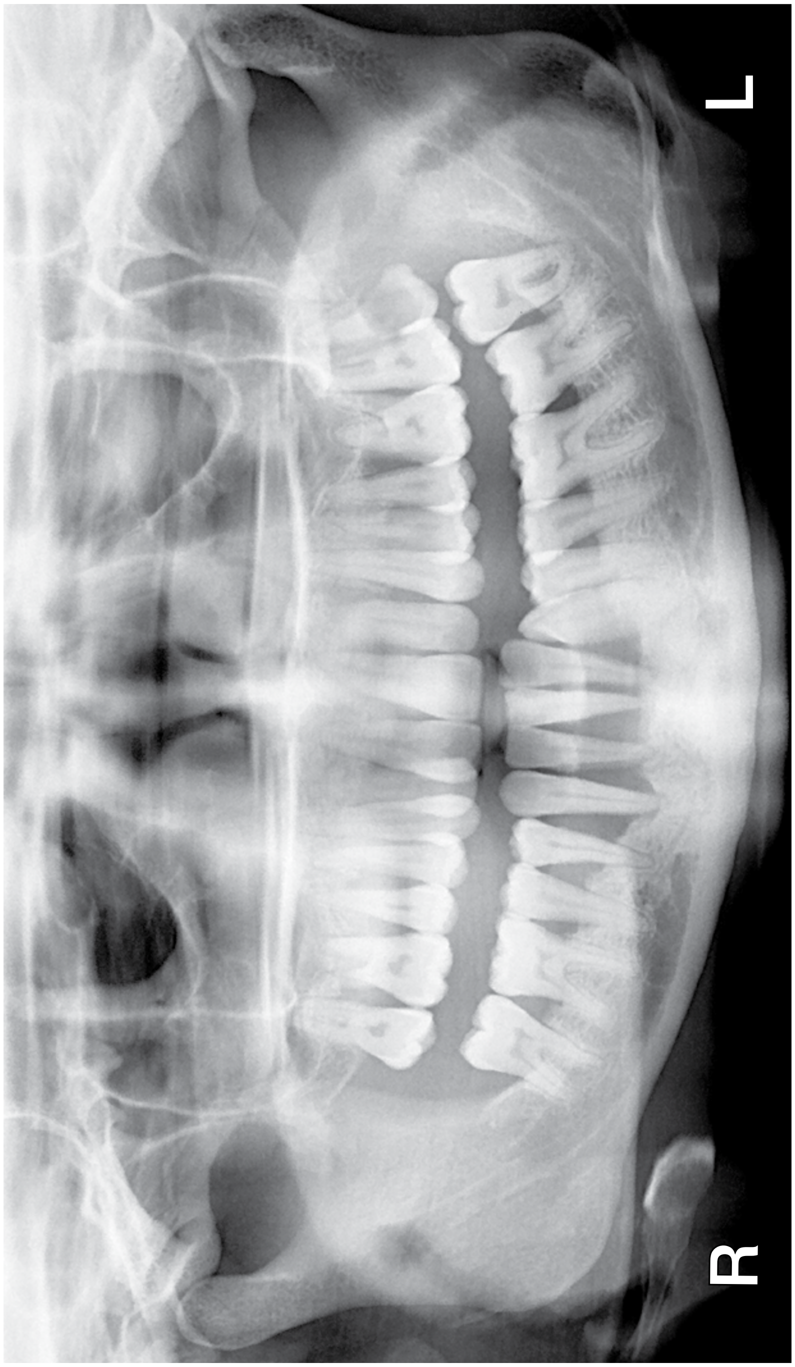

Figure 38-3 Panoramic Radiograph for Case 2.

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Section 3: Case 3

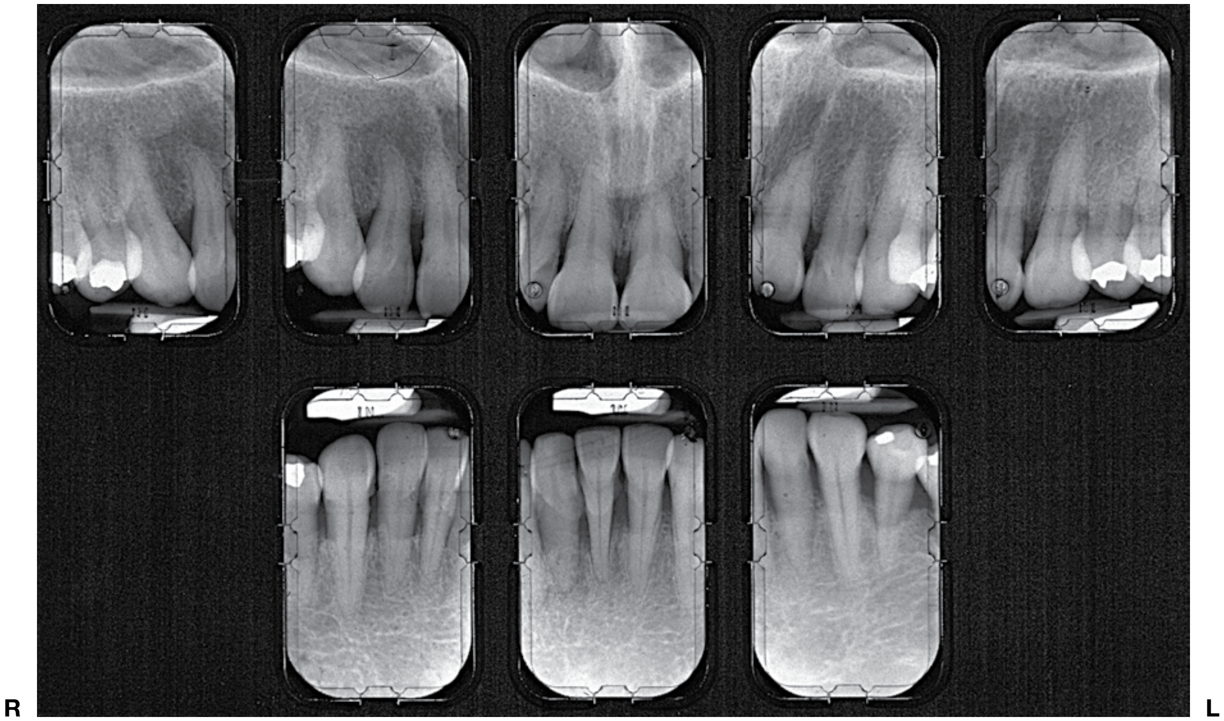

Figure 38-4A Anterior Radiographs for Case 3.

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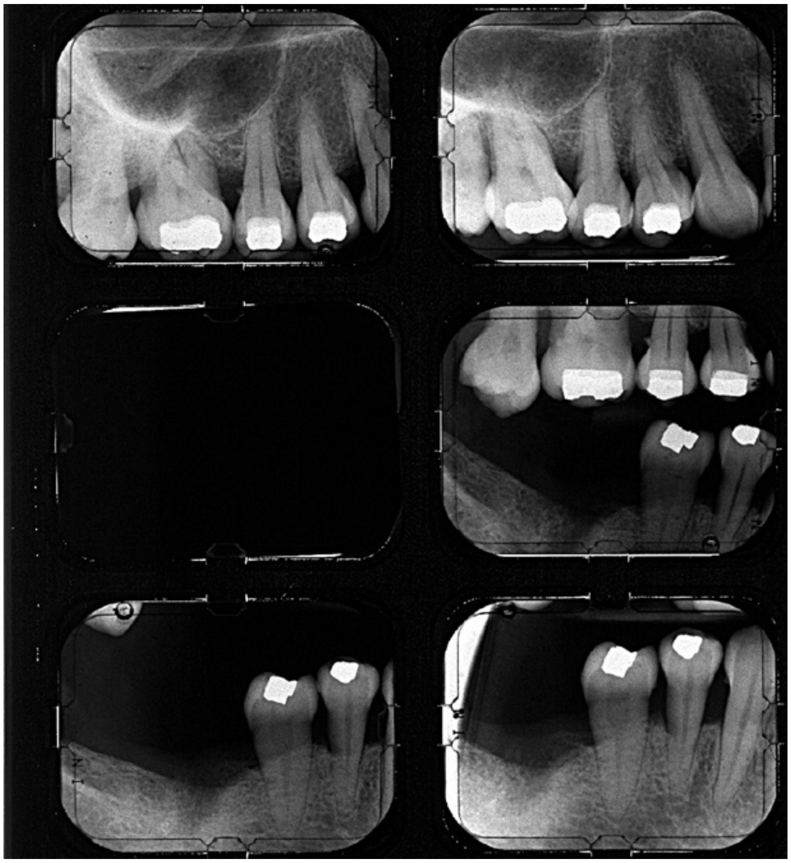

Figure 38-4B Right Posterior Radiographs for Case 3.

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Figure 38-4C Left Posterior Radiographs for Case 3.

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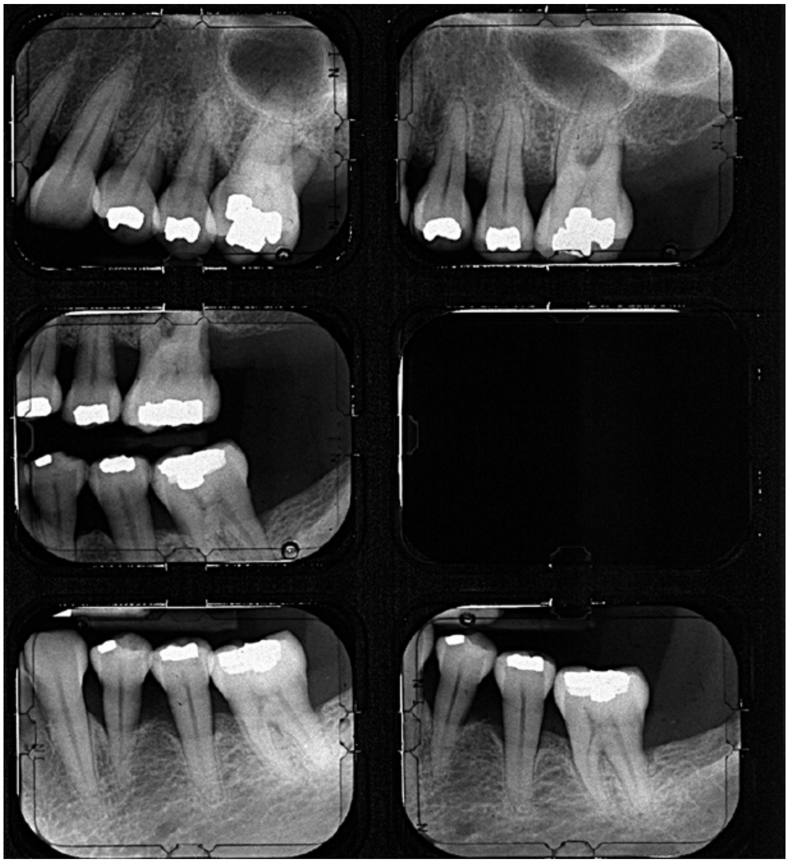

Section 4: Case 4

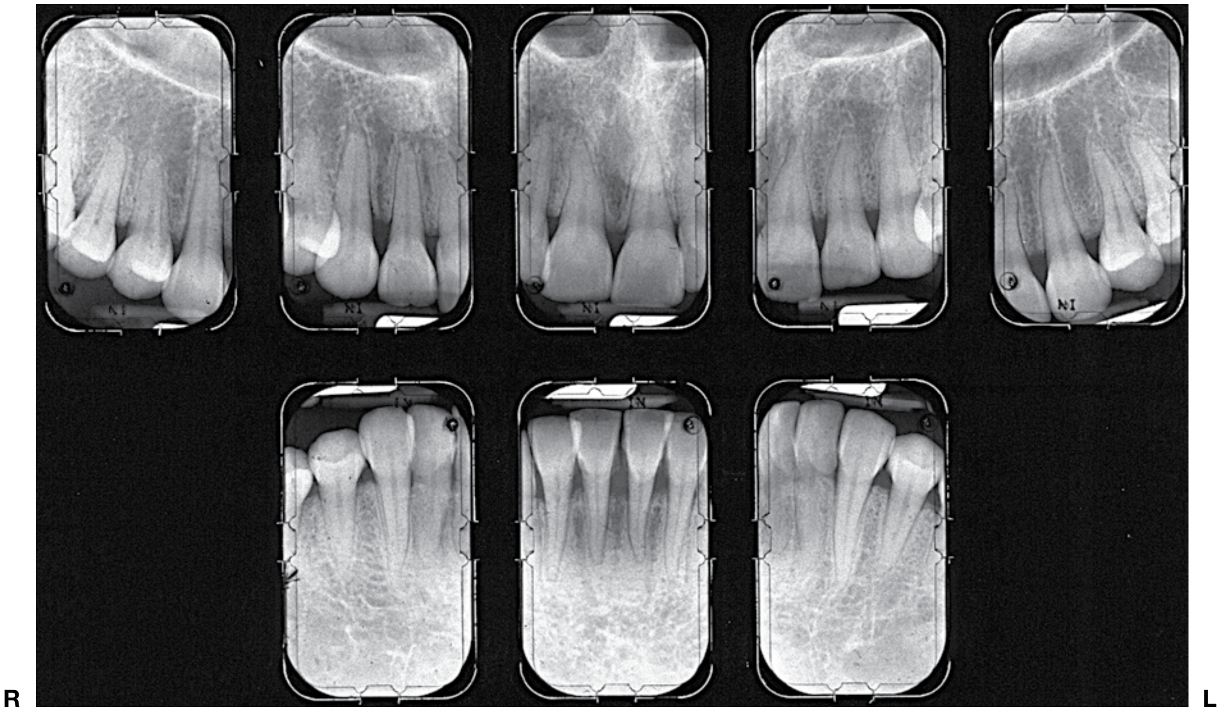

Figure 38-5A Radiographs for Case 4.

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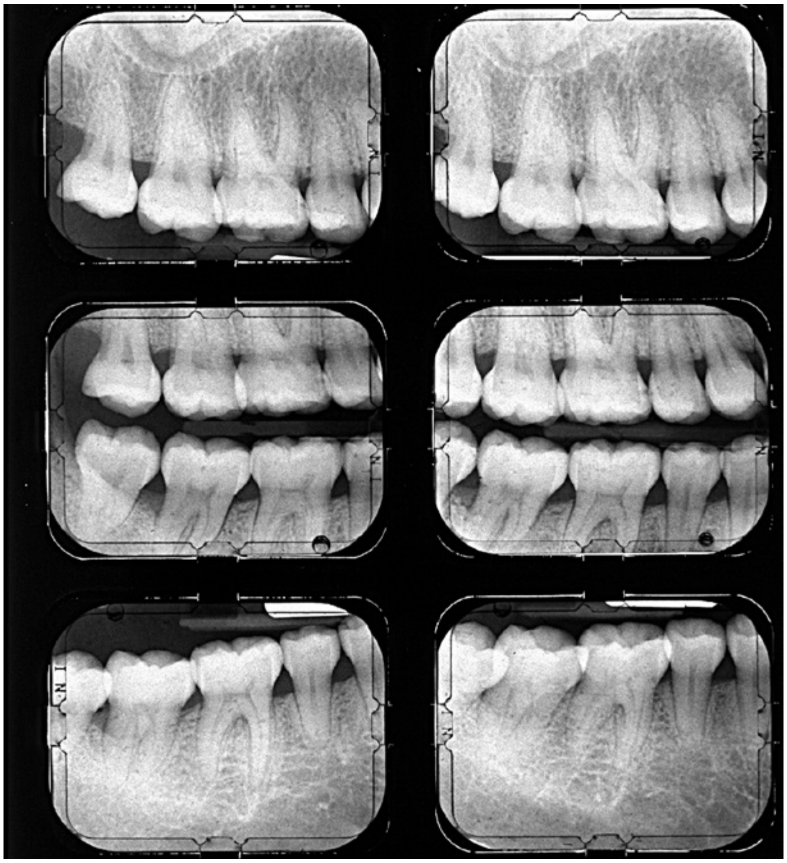

Figure 38-5B Right Posterior Radiographs for Case 4.

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Figure 38-5C Left Posterior Radiographs for Case 4.

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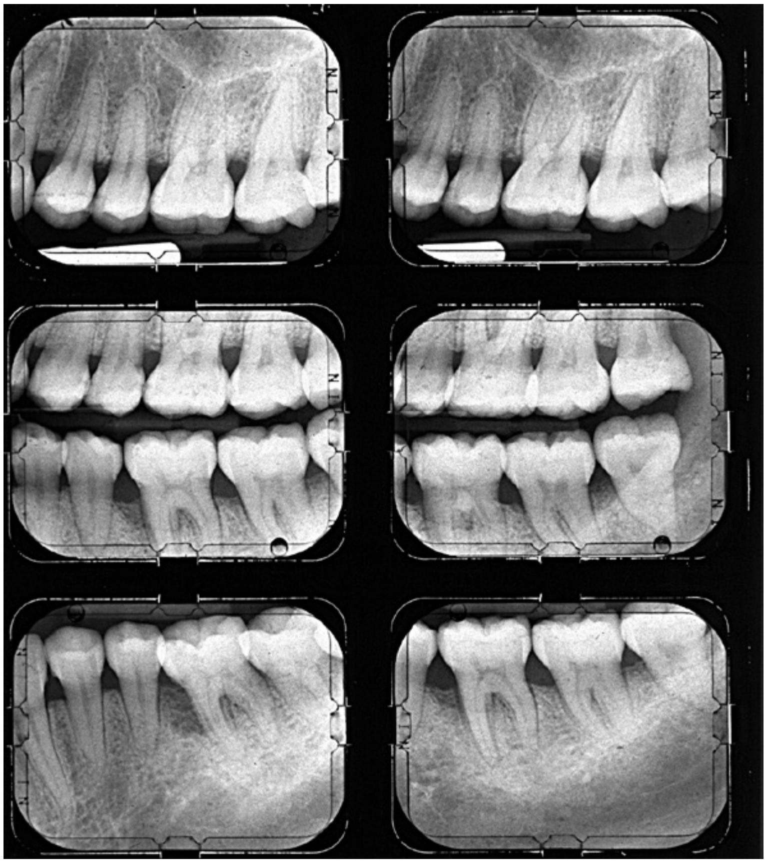

Section 5: Case 5

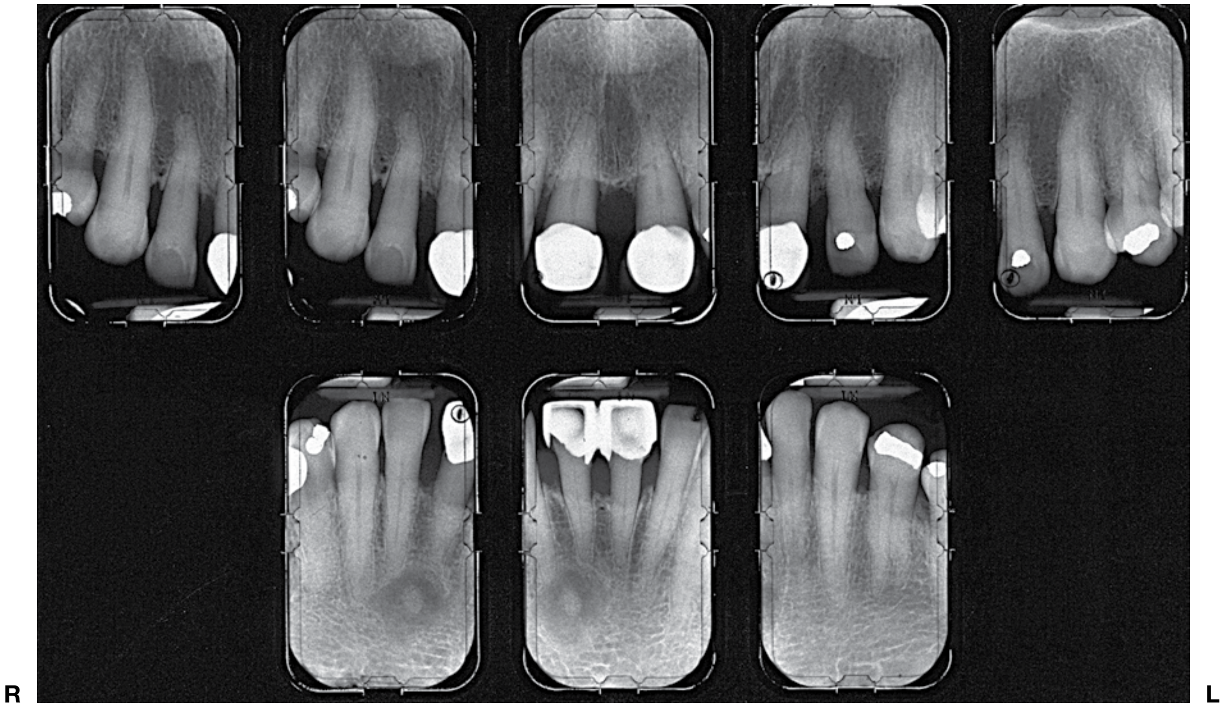

Figure 38-6A Radiographs for Case 5.

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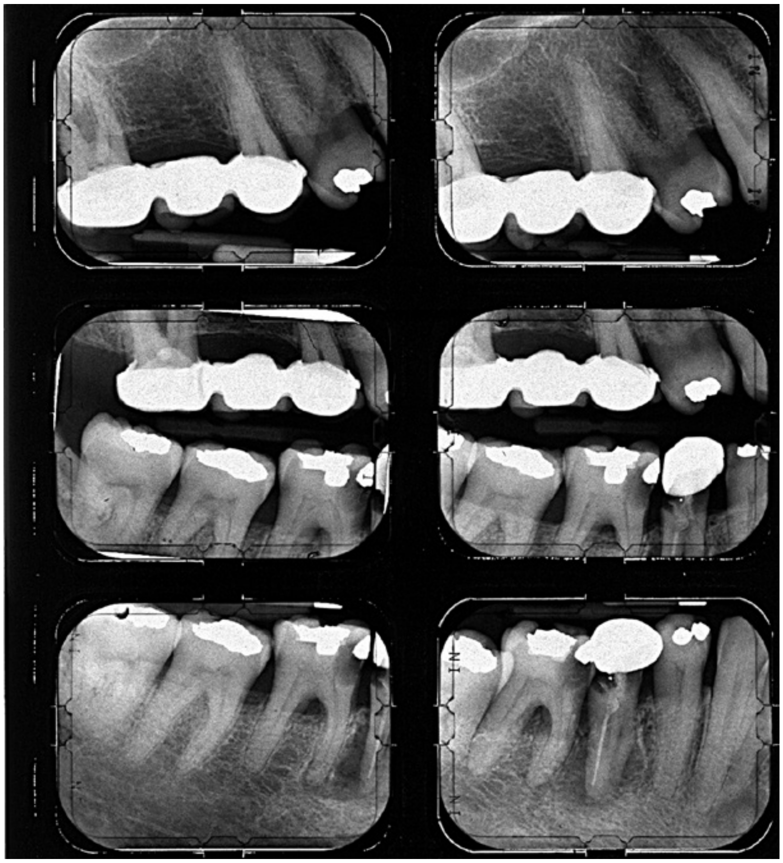

Figure 38-6B Right Posterior Radiographs for Case 5.

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Figure 38-6C Left Posterior Radiographs for Case 5.

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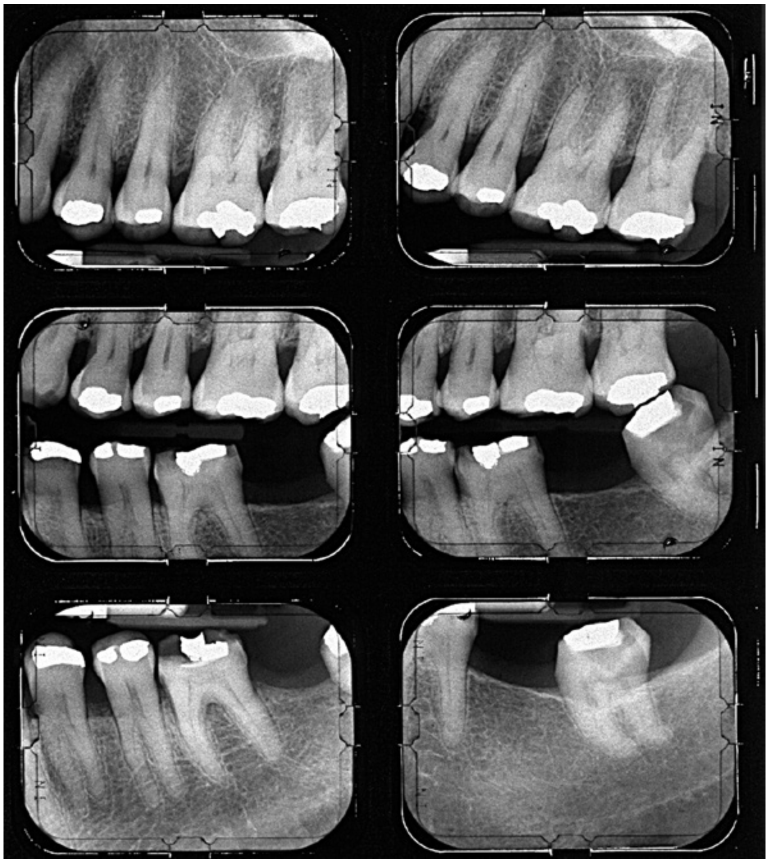

Section 6: Case 6

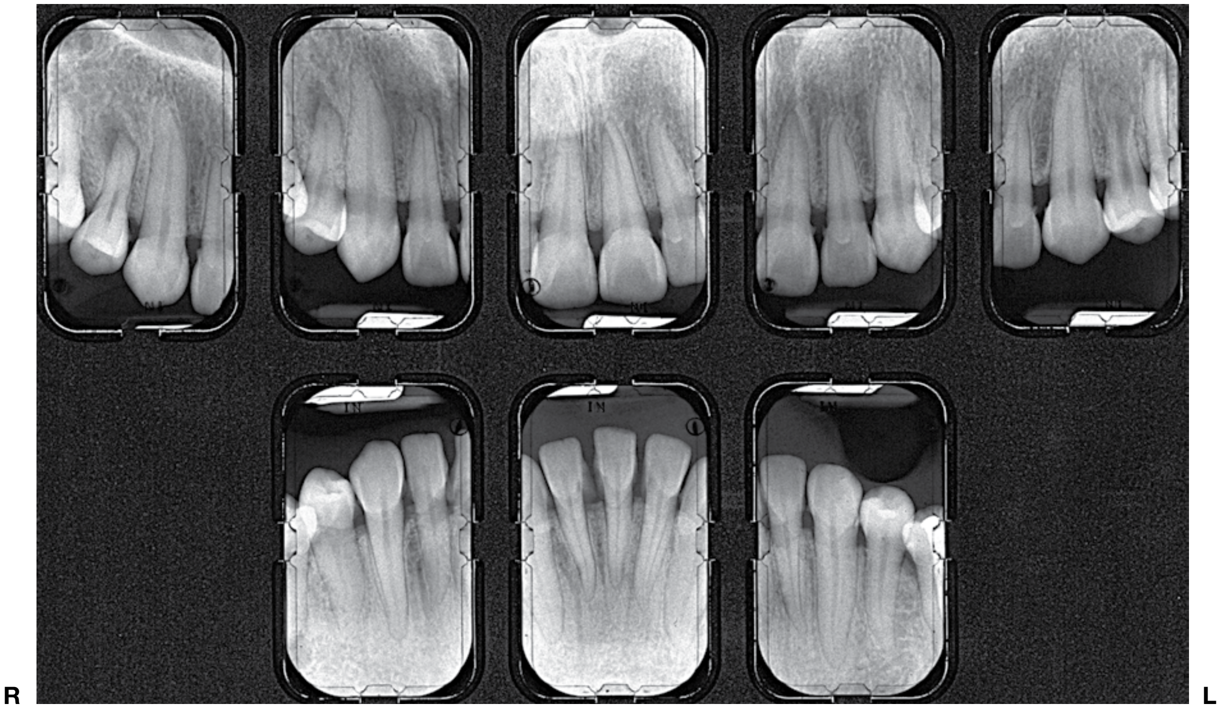

Figure 38-7A Radiographs for Case 6.

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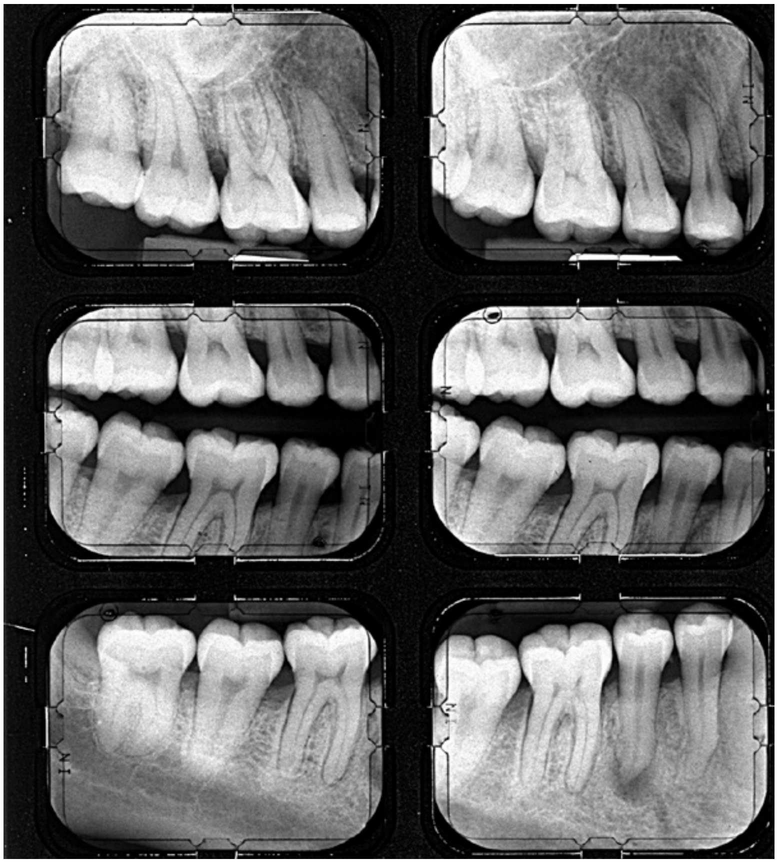

Figure 38-7B Right Posterior Radiographs for Case 6.

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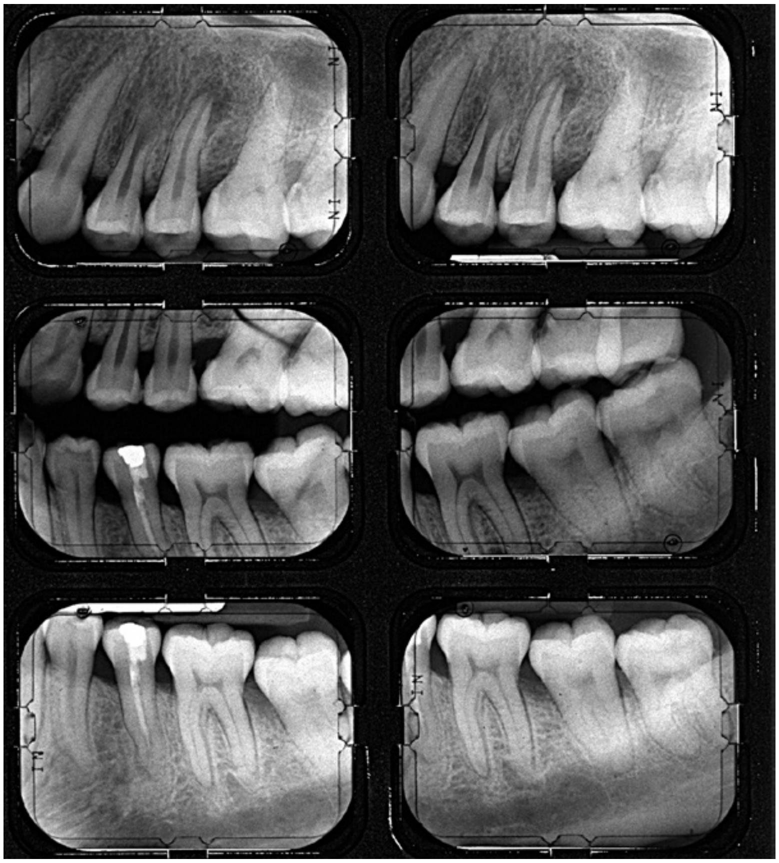

Figure 38-7C Left Posterior Radiographs for Case 6.

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Glossary

2017 AAP/EFP Classification of Periodontal and Peri-Implant Diseases and Conditions a revamped disease classification framework that guides comprehensive treatment planning and allows for a personalized approach to patient care. It includes a multidimensional staging and grading system for periodontitis classification, a recategorization of various forms of periodontitis, and the inaugural classification for peri-implant diseases and conditions.

A

Aberrant frenum an abnormal insertion of the frenal attachment that is located in close proximity to the marginal gingiva. An aberrant frenum may limit movement of the cheek, lip, or tongue, or it may pull the gingival margin away from the tooth, thereby causing gingival recession.

Abscess of the periodontium an acute infection involving a circumscribed collection of pus in the periodontium. Also see gingival abscess, periodontal abscess, and pericoronal abscess.

Absolute contraindication a condition/factor that makes a particular treatment or procedure absolutely inadvisable due to the potential risk/harm outweighing the potential benefit of treatment. Medical and dental treatment must be avoided in such cases because of the severe and potentially life-threatening risks involved. An example of an absolute contraindication for periodontal surgery might be a patient with full-blown AIDS or a patient with advanced chronic obstructive pulmonary disease.

Absorbable suture a suture made from a material designed to dissolve harmlessly in body fluids over time; though absorbable sutures do not normally require removal by the dental team, some absorbable sutures do not dissolve well and thus may require removal by a dental provider 1 to 2 weeks following the surgery. Also see nonabsorbable suture.

Acceptance includes avoiding judgment, supporting the patient's freedom to make their own choices, understanding the patient's perspective, and affirming their strengths and efforts.

Acquired immunodeficiency syndrome (AIDS) a chronic, potentially life-threatening disease caused by human immunodeficiency virus (HIV). People with acquired immunodeficiency syndrome are at an increased risk for developing certain cancers and for infections that usually occur only in individuals with a weak immune system.

Acquired salivary pellicle a salivary film that forms over a clean tooth surface after plaque removal; it is composed of a variety of salivary glycoproteins (mucins) and antibodies; its purpose is to protect the enamel from acidic activity (breakdown of the enamel from the low pH oral environment).

Active disease site an area of tissue destruction that shows continued apical migration of the junctional epithelium over time.

Acute abscess an abscess of the periodontium that has a rapid onset characterized by pain and discomfort; it is primarily caused by an exacerbation of a chronic inflammatory periodontal lesion.

Acute inflammation a short-term, normal inflammatory response that protects and heals the body following physical injury or infection. Also see inflammation and chronic inflammation.

Acute periodontal diseases periodontal conditions that are commonly characterized by a rapid onset and rapid course that are frequently accompanied by pain and discomfort and they may be unrelated to the presence of preexisting gingivitis or periodontitis. One example is an abscess of the periodontium.

Affirm acknowledge and support the patient's willingness to discuss a difficult topic (i.e., a change about which they may feel embarrassment, guilt, or ambivalence).

Agranulocytes a leukocyte that lacks granules. Lymphocytes and monocytes/macrophages are agranulocytes. Compared to granulocytes, agranulocytes have a larger rounded nucleus because of the lack of cytoplasmic granules.

Allografts bone replacement grafts taken from individuals that are genetically dissimilar to the donor (i.e., another human donor); these grafting materials must be modified to eliminate the potential for rejection and minimize the risk of transmitting infection from donor to recipient. Also see autographs, xenografts, and alloplasts.

Alloplasts bone replacement grafts that are synthetic materials or inert foreign materials. Also see autografts, allografts, and xenografts.

Alveolar bone loss the resorption of alveolar bone as a result of periodontitis. Also see horizontal bone loss and vertical bone loss.

Alveolar bone or alveolar process the bone that surrounds the roots of the teeth. It forms the bony sockets that support and protect the roots of the teeth.

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Alveolar bone proper the thin layer of bone that lines the socket to surround the root of the tooth (also called the cribriform plate); the ends of the periodontal ligament fibers are embedded in the alveolar bone proper.

Alveolar crest the coronal-most portion of the alveolar process. In health, the alveolar crest is located 1 to 2 mm apical to (below) the cementoenamel junctions (CEJs) of the teeth.

Alveolar mucosa the apical boundary, or lower edge, of the gingiva; it can be distinguished easily from the gingiva by its dark red color and smooth, shiny surface.

Alveolar process the part of the maxilla and mandible that forms and supports the sockets of the teeth.

Alveolus the bony socket; a cavity in the alveolar bone that houses the root of a tooth.

Ambivalence having mixed feelings and attitudes about something, such as a behavior change.

American Academy of Periodontology an association of dental professionals specializing in the prevention, diagnosis, and treatment of diseases affecting the periodontium.

Anastomose to join together; in the periodontium to create a complex system of blood vessels that supply blood to the periodontal tissues.

Antibiotic resistance the ability of a bacterium to withstand the effects of an antibiotic by developing mechanisms to protect the bacterium from the killing or inhibiting effects of the antibiotic.

Antibodies Y-shaped proteins; one end of the Y binds to the outside of the B cell and the other end binds to a microorganism and helps to neutralize it. Antibodies are known collectively as immunoglobulins.

Anti-cytokine therapy a novel type of therapy used to control systemic inflammatory autoimmune disorders, such as rheumatoid arthritis and inflammatory bowel syndrome (IBS). Anti-cytokine therapy targets inflammation by neutralizing cytokines, blocking cytokine receptors, and/or activating anti-inflammatory pathways such as immunosuppressive pathways. In dentistry, anti-cytokine therapy holds much promise in becoming a host modulating agent to treat and manage periodontal disease, but further research is still needed

Anti-inflammatory mediators biochemical mediators that are protective to the host by limiting the bacterial-induced inflammatory Sresponse from inflicing serious harm to the periodontium.

Antioxidants compounds that are found naturally in the body and in certain foods; antioxidants inhibit oxidation, thereby blocking damage to cells by free radicals.

Apical migration of the junctional epithelium the movement of the junctional epithelium apical to its normal location.

Apically positioned flap with osseous resective surgery a periodontal surgical procedure involving a combination of a displaced flap (displaced in an apical direction) plus resective osseous surgery; this procedure is ideal for minimizing periodontal pocket depths in patients with osseous craters caused by moderate periodontitis. Also see displaced flap.

Papillae small bumps on the upper surface of the tongue that give it its characteristic rough texture. It has taste buds that are sensitive to taste.

Ascorbic acid deficiency gingivitis an inflammatory response of the gingiva caused by dental plaque that is aggravated by chronically low vitamin C (ascorbic acid) levels; manifests clinically as bright red, swollen, ulcerated gingival tissue that bleeds with the slightest provocation.

Assessment and preliminary therapy phase assessment of collected data and urgent care for any acute conditions, such as emergency dental care.

Association a relationship between two or more variables. Association does not imply a causal relationship between two different variables and is therefore not synonymous with causation. See causation.

Atherosclerosis a process characterized by a thickening of artery walls.

Attached gingiva the part of the gingiva that is firm, dense, and tightly connected to the cementum on the cervical-third of the root or to the periosteum (connective tissue cover) of the alveolar bone.

Attachment loss the destruction of the tooth supporting structures destroyed around a tooth; characterized by relocation of the junctional epithelium to the tooth root, destruction of the fibers of the gingiva, destruction of the periodontal ligament fibers, and loss of alveolar bone support from around the tooth. Also see clinical attachment loss.

Autografts bone replacement materials taken from the patient that is receiving the graft; periodontal autografts can be taken from sites in the patient's own jaws or occasionally from other areas of the patient's body. Also see allografts, xenografts, and alloplasts.

B

Bacterium/bacteria the simplest organisms which can be seen only through a microscope.

Basal lamina a thin mat of extracellular matrix secreted by the epithelial cells; this mat supports the overlying epithelium. The basal lamina also functions like a filter that allows only water and small molecules to pass through. Also see external basal lamina and internal basal lamina.

Baseline data clinical data gathered at the beginning of the periodontal treatment that is subsequently used for comparison to clinical information gathered at subsequent appointments.

Basement membrane a thin layer of tissue visible with a light microscope beneath the epithelium. The basement membrane is composed of a basal lamina and a reticular lamina.

Best evidence the highest level of evidence available for a specific clinical question. Also see best practice and levels of evidence.

Best practice clinical practices, treatments, and interventions that result in the best possible outcome for the patient. Also see best evidence and levels of evidence.

Biochemical mediators biologically active compounds secreted by immune cells that activate the body's inflammatory response; inflammatory mediators of importance in periodontitis are the interleukins, prostaglandins, and matrix metalloproteinases.

Biocompatible a nonbiologic material that is not rejected by the body. Titanium is a biocompatible metal that allows tissue healing around an implant abutment.

Biofilm a well-organized community of bacteria that adheres to a surface and is embedded in an extracellular slime layer; forms rapidly on almost any surface that is wet. See extracellular protective matrix and fluid channels.

Biologic equilibrium a state of balance in the internal environment of the body.

Biomechanical forces the biologic and mechanical forces placed on an osseointegrated dental implant; controlling these forces is vital to achieve long-term success with implants.

Biomechanical overload an implant-related complication that occurs when biomechanical force exceeds the physiologic tolerance of the peri-implant bone to withstand loading forces; it can lead to implant failure in the absence of plaque.

Bisphosphonates drugs that can inhibit the resorption of bone by altering osteoclastic activity. One of the possible side effects of these drugs is osteonecrosis of the jaws following their extended use.

Blunt dissection elevation of a flap during periodontal surgery using tools that are not sharpened on the edge (i.e., blunted or slightly rounded); blunt dissection minimizes the chance of accidental damage to the flap. In this type of flap elevation, the flap is lifted or pried up using surgical tools called periosteal elevators, and it is elevated in a manner quite similar to lifting the peeling off an orange. Also see sharp dissection.

B-lymphocytes small leukocytes that help in the defense against bacteria, viruses, and fungi; principal function is to make antibodies. B-lymphocytes can further differentiate into one of the two types of cells plasma B-cells and memory B-cells.

Body mass index (BMI) a simple index that takes account of a person's weight and height to classify the individual as underweight, normal weight, overweight, or obese.

Bone remodeling Alveolar bone constantly undergoes periods of bone formation and resorption (loss) in response to mechanical forces on the tooth and inflammation of the periodontium. This process of bone formation and resorption is known as bone remodeling.

Bone replacement graft a periodontal surgical procedure used to encourage the body to rebuild alveolar bone that has been lost usually as a result of periodontal disease.

Bone resorption occurs when osteoclasts are stimulated by RANKL to resorb alveolar bone.

Bruxism the forceful grinding of the teeth.

C

Cairo classification system a new gingival recession classification system based on the clinical attachment level measurements at both buccal and interproximal sites. Also see Miller classification system.

Cancellous bone (spongy bone) the lattice-like bone that fills the interior portion of the alveolar process between the cortical bone and the alveolar bone proper; cancellous bone is oriented around the tooth to form support for the alveolar bone proper.

Caries Management by Risk Assessment (CAMBRA) a series of protocols that seek to provide practical clinical guidelines for managing dental caries based on risk group assessment. The protocols are based on the best evidence at this time and can be used in planning effective caries management for any patient.

Catabasis a highly regulated process that occurs following removal of microbial challenge and leads to resolution of inflammation and a return to homeostasis.

Causal factor an event or condition that leads to an occurrence of a disease.

Causation one variable directly causes changes in another variable.

Cell envelope a complex, multilayered structure that serves to protect the microorganism from the unpredictable and inhospitable external environment. It is the "skin" of the microorganism.

Cell junctions cellular structures that mechanically "lock" a cell and its cytoskeleton to its neighboring cells or to the basal lamina. Also see desmosome and hemidesmosome.

Cells the smallest structural unit of living matter capable of functioning independently; cells group together to form a tissue.

Cementum proteins a group of proteins present in cementum, in addition to collagens; may have an important role in regulating the mineralization process associated with cementum formation. From the current status of knowledge, it appears that cementum proteins may play an important role in future therapies to achieve regeneration of the periodontal structures.

Cementum a mineralized tissue that covers the roots of the teeth and serves to attach the tooth to alveolar bone via collagen fibers of the periodontal ligament; anatomically, cementum is part of the tooth; however it also part of the periodontium.

Centers for Disease Control and Prevention (CDC) a United States science-based, data-driven federal agency that provides guidance and standardized methods for public health surveillance. It also plays an important role as the lead federal agency in the U.S. in detecting and responding to new and emerging health threats, such as COVID-19.

Cervical enamel projection (CEP) an apical extension of the coronal enamel beyond the CEJ that is projected toward the furcation entrance. A CEP may contribute to an isolated furcation invasion affecting a molar or a birfucated maxillary first premolar.

Change talk encouraging the expression of statements in the direction of change; evoking, facilitating, and strengthening a patient's desire for change. Change talk can be elicited with simple open-ended questions such as "What advantages do you see of cutting back on sugary foods?"

Chemokines a major subgroup of cytokines that cause additional immune cells to be attracted to the site of infection or injury. Also see cytokines.

Chemotaxis the process whereby leukocytes are attracted to an infection site in response to biochemical compounds released by invading microorganisms.

Chemotherapy a broad term that describes any type of therapy that utilizes a chemical agent to treat or control a disease. The term *chemotherapeutic* refers to the actual chemical agent used to treat and manage the disease.

Chronic abscess an abscess of the periodontium that grows slowly and is not typically associated with pain; it forms after the spread of infection has been controlled by spontaneous drainage, host response, or therapy.

Chronic inflammation is a long-lived, out-of-control inflammatory response that continues for more than a few weeks; it is a pathologic condition characterized by active inflammation, tissue destruction, and attempts at repair. Also see inflammation and acute inflammation.

Circumscribed localized or confined to a specific site.

Classification system grouping of similar entities on the basis of certain differing characteristics; classification systems provide a tool for understanding the etiology, pathogenesis, and treatment of periodontal diseases in an orderly manner.

Clenching the continuous or intermittent forceful closure of the maxillary teeth against the mandibular teeth.

Clinical attachment level (CAL) an estimate of the true periodontal support around the tooth as measured with a periodontal probe. This measurement is only an estimation of the actual histologic level of attachment still present. It is a means of estimating the level of the junctional epithelium.

Clinical decision-making the process whereby all members of the dental team use the information gathered during the comprehensive periodontal assessment to arrive at an appropriate diagnosis and identify treatment strategies that meet the individual needs of the patient.

Clinical periodontal assessment a fact-gathering process designed to provide a comprehensive picture of the patient's periodontal health status. Also see comprehensive periodontal assessment and periodontal screening examination.

Coaggregation the cell-to-cell adherence of one oral bacterium to another; the ability to adhere and coaggregate is an important determinant in the development of oral bacterial biofilms.

Cochrane Oral Health database includes systematic reviews of health care interventions that are produced and disseminated by Cochrane Oral Health—a global nonprofit organization. It was established in 1993 by a British epidemiologist who recognized that ready access to systematic reviews of available evidence would facilitate better-informed decisions by health care providers.

Collagen fibers protein fibers that form a dense network of strong, rope-like cables that secure and hold the gingival connective tissues together.

Comanagement is the shared responsibility and accountability for the care of a patient.

Combustible tobacco delivery systems traditional method of nicotine delivery composed of tobacco leaves that are actively burned upon being lit. The burning of the tobacco leaves creates an aerosol that the user can inhale.

Commensal bacteria the natural, indigenous inhabitants of the oral cavity; they play an important role in contributing to host defense by defending against colonization by opportunistic pathogenic bacteria.

Community-acquired pneumonia pneumonia that is contracted outside of the hospital setting. They are commonly caused by aspiration of oropharyngeal organisms such as *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Mycoplasma pneumonia* into the lung.

Compassion the ability to understand and empathize for another individual's sufferings or misfortunes. The hygienist exercises compassion by carefully listening to the patient's concerns and addressing them in a timely and appropriate manner.

Complement system a complex series of proteins circulating in the blood that works to facilitate phagocytosis or kill bacteria directly by puncturing bacterial cell membranes.

Complementary and alternative medicine (CAM) a wide variety of unconventional medical and dental products/remedies/practices that are backed by unproven claims, questionable effectiveness, and dubious safety under the broad umbrella known as complementary and alternative medicine. CAM can be thought of as atypical medicines and health care practices that are not part of standard medical care.

Compliance the extent to which a person's behavior coincides with health advice. Examples of compliant patients would be a patient who faithfully takes antihypertensive medications as prescribed by a physician or a patient who cooperates in meeting regularly scheduled periodontal maintenance appointments as recommended by the dental team. Also see compliant patient and noncompliant patient.

Compliant patient a patient who adheres to the recommendations set forth by the health care provider.

Comprehensive periodontal assessment an thorough and meticulous evaluation of the periodontium; normally includes clinical features such as probing depth measurements, bleeding on probing, presence of exudate, level of the free gingival margin and the mucogingival junction, tooth mobility, furcation involvement, presence of calculus and bacterial plaque, gingival inflammation, radiographic evidence of alveolar bone loss, and presence of local contributing factors.

Compromised periodontal maintenance a type of periodontal treatment that is not intended to attain optimal results; designed to slow down (not eliminate or fully arrest) the progression of periodontitis in patients who may not respond favorably to active conventional periodontal therapy (such as nonsurgical scaling and root planing or periodontal surgery) due to serious health conditions, poor motivation/compliance, advanced age, or severe periodontal disease.

Computed tomography an emerging imaging technique that emits cone-shaped x-ray beams that are recaptured by the scanning machine to reconstruct a three-dimensional image of the patient's anatomy.

Computer-linked periodontal probes probes that use an optical handpiece that electronically reads and records measurements.

Cone beam computed tomography (CBCT) a specialized radiographic imaging method that creates an accurate, three-dimensional (3D) image of hard tissue structures. It is an advanced imaging modality that has growing clinical applications in the field of dentistry.

Confirmation bias a human tendency to look for or interpret information that confirms our beliefs.

Connective tissue papillae finger-like extensions of connective tissue that project up and interlock with the epithelium.

Connective tissue tissue that fills the spaces between the tissues and organs in the body; it consists of sparse cells and collagen fibers separated by abundant extracellular substance.

Continuous disease progression theory periodontal disease progresses throughout the entire mouth in a slow and constant rate over the adult life of the patient.

Controlled-release chemotherapeutic agent an agent that consists of an antibacterial mediator that is embedded within a carrier material. They are designed to be placed directly into the periodontal pocket where the carrier material attaches to the tooth surface and dissolves slowly, producing a steady, sustained release of the antimicrobial agent over a period of several days within the periodontal pocket.

Conventional mechanical periodontal therapy a term that refers to self-care, periodontal instrumentation, and control of local contributing factors.

Coronally positioned flap a periodontal plastic surgical procedure that can be used to repair gingival recession if the recession is not advanced; the coronally positioned flap is a displaced flap (displaced in a coronal direction in this case). Also see laterally positioned flap and semilunar flap.

Cortical bone a layer of compact bone that forms the hard, outside wall of the mandible and maxilla on the facial and lingual aspects; cortical bone surrounds the alveolar bone proper and gives support to the socket.

C-reactive protein (CRP) a special type of plasma protein that is present during episodes of acute inflammation or infection: CRP is an important cardiovascular risk predictor.

Crestal irregularities the appearance on a dental radiographic of breaks or fuzziness instead of a clear, distinct radiopaque line at the crest of the interdental alveolar bone.

Crevicular incision a type of horizontal incision employed during periodontal flap surgery in which the surgical scalpel is simply placed into the gingival crevice or sulcus and the tissues are incised apically to bone. Also see horizontal incision and internal bevel incision.

Cribriform plate the thin layer of bone that lines the socket and surround the root of the tooth (also called the alveolar bone proper); the ends of the periodontal ligament fibers are embedded in the alveolar bone proper.

Crown lengthening surgery a periodontal plastic surgical procedure designed to create a longer clinical crown for a tooth by removing some of the gingiva and usually by removing some alveolar bone from the necks of the teeth. Also see functional crown lengthening and esthetic crown lengthening.

Cyclosporine an immunosuppressive agent used for prevention of transplant rejection as well as for management of a number of autoimmune conditions such as rheumatoid arthritis; associated with drug-influenced gingival enlargement.

Cytokines a general name for powerful regulatory proteins released by immune cells that influence the behavior of other nearby cells: cytokines signal the immune system to send additional phagocytic cells to the site of an infection.

D

Databases online indexes that list all articles published in a given period of time by journals in a particular profession or group of professions, such as PubMed, MEDLINE, or CINAHL (Cumulative Index of Nursing and Allied Health Literature).

Dehiscence loss of alveolar bone on one aspect of the tooth, typically the facial aspect, that leaves the area of the root covered by soft tissue only.

Dental biofilm-induced gingivitis (may also be referred to as dental plaque-induced gingivitis) periodontal disease involving inflammation of the gingiva in response to bacteria located at the gingival margin; the most common form of gingival disease.

Dental calculus mineralized bacterial plaque covered on its external surface by nonmineralized, living bacterial plaque.

Dental implant a nonbiologic (artificial) device surgically inserted into the jawbone to replace a missing tooth or provide support for a prosthetic denture.

Dental stem cells a natural source of undifferentiated stem cells that have the ability to differentiate into a variety of more well-developed specialized cells, such as osteocytes, adipocytes, chondrocytes, and muscle forming cells. Stem cells obtained from the PDL space, for instance, have been shown to potentially have the capability to differentiate into cells that produce pulp tissue and periodontal tissue.

Dentogingival unit a functional unit comprised of the junctional epithelium and the connective tissue attachment; the dentogingival unit acts to provide structural support to the gingival tissue.

Desmosome a specialized cell junction that connects two neighboring epithelial cells and their cytoskeletons together.

Developing discrepancy inconsistencies between a patient's values and goals and current behavior are explored to enhance motivation for change.

Diabetes mellitus a disease in which the body does not produce or properly use insulin. Insulin is a hormone that is needed to convert sugar, starches, and other food into energy that the body uses to sustain life.

Diffuse gingivitis a type of gingivitis, in which the distribution of inflammation may be more extensive to include the gingival margin, papilla, and the attached gingiva.

Disease progression the sequence of events that occur during the development of a disease or abnormal condition; the change or advancement of periodontal destruction. Also see intermittent disease progression theory.

Disease site an area of tissue destruction. A disease site may involve only a single surface of a tooth, for example, the distal surface of a tooth. The disease site may involve several surfaces of the tooth or all four surfaces (mesial, distal, facial, and lingual). Also see inactive disease site and active disease site.

Disengagement behavior behavior characterized by a patient's disinterest, defensiveness, lack of warmth, or negative demeanor. A patient exhibiting disengagement behavior may be a sign of anxiousness or disinterest. See engagement behavior.

Displaced flap a periodontal flap that is sutured with the margin of the flap placed at a position other than its original position in relationship to the CEJ of the tooth; a displaced flap can be positioned apically, coronally, or laterally in relationship to its original position. Also see nondisplaced flap.

Distraction a common iatrosedation technique used to calm an anxious patient. Distraction relies on diverting the patient's attention away from a procedure that the individual may perceive as unpleasant.

Distribution of inflammation describes how spread out the inflammation is within the gingival unit.

Down syndrome a genetic disorder caused by a gene mutation which results in an extra chromosome at position 21 (hence, another name for this disorder is Trisomy 21); it is a lifelong condition, which varies in severity, causing intellectual disability, and developmental delays, and in some people, health problems. There is no cure for this genetic disorder.

Doxycycline an antibiotic drug that is used to treat a variety of infections. Doxycycline at low doses is used as a host-modulating agent to inhibit part of the destruction occurs in periodontitis. See subantimicrobial dose doxycycline.

Drug-influenced gingival enlargement an esthetically disfiguring overgrowth of the gingiva that is a side effect associated with certain medications such as anticonvulsants, calcium channel blockers, and immunosuppressants.

Drug-influenced gingivitis a form of gingivitis that results from any systemic medication that causes an exaggerated inflammatory response to plaque biofilm. Oral contraceptives are an example of medications that triggers an exaggerated inflammatory response to plaque biofilm.

Dysbiosis a microbial imbalance that results in a disruption of the microbial homeostasis inside the biofilm. Dysbiosis is the direct opposite of symbiosis.

E

Ecological plaque hypothesis a novel plaque hypothesis that combines key points from the specific plaque hypothesis and the nonspecific plaque hypothesis. It postulates that the accumulation of nonspecific bacteria triggers the host inflammatory response which, in turn, alters the local environment (ecology) with the gingival sulcus to favor the growth of specific pathogenic bacteria.

Elicit provide elicit The first step (elicit) involves determining what the patient already knows. The second step (provide) involves providing relevant information and advice. The final step (elicit) involves checking back with the patient to see what he or she has taken from the information or advice. This is the three-step process of motivational interviewing.

Embrasure space the space apical to the contact area of two adjacent teeth. Embrasure space is filled by interdental papilla. The ideal shape and size of the embrasure space is one that permits the introduction of interproximal cleaning devices that aid in the removal of plaque. See gingival embrasure space.

Enamel pearl an ectopic, spherical formation of enamel that is located on the root surface. The presence of an enamel pearl can be a local contributing factor to periodontal inflammation.

Endodontic-periodontal lesion a localized bacterial infection affecting both the dental pulp and periodontal tissues of the same tooth. It is a complex disorder that can originate from either the dental pulp or periodontal tissues, or both, making the origin of the infection difficult to determine.

Engagement behaviors behavior characterized by a patient's disinterest, defensiveness, lack of warmth, or negative demeanor. A patient exhibiting disengagement behavior may be a sign of anxiousness or disinterest. See engagement behavior.

Engaging the "connection" process. The first process upon meeting a patient is to engage the patient in wanting to work together. Engaging establishes the connection or rapport that is the foundation for everything that follows.

Environmental tobacco smoke (ETS) refers to being exposed to tobacco smoke from someone else's cigarette, cigar, or pipe. Also known as secondhand smoke.

Epidemiology the study of the health and disease within the total population (rather than an individual) and the risk factors that influence health and disease.

Episodic a term to describe the progression of periodontitis occurring in a succession of acute destructive phases interspersed with intermittent inactive phases.

Epithelial ridges deep extensions of epithelium that reach down into the connective tissue. The epithelial ridges are also known as rete pegs.

Epithelial tissue the tissue that makes up the outer surface of the body (skin or epidermis) and lines the body cavities such as the mouth, stomach, and intestines (mucosa).

Epithelial connective tissue interface the boundary where the epithelial and connective tissues meet.

Eruption gingivitis a transient form of gingivitis that is seen in young children as the teeth erupt out of the gingiva.

Esthetic crown lengthening a type of crown lengthening performed on teeth to improve the appearance of the teeth where there is excessive gingiva or a "gummy smile" as it is sometimes called. Also see functional crown lengthening.

Etiologic factor any attribute, characteristic, or exposure that is known to cause a disease.

European Federation of Periodontology a nonprofit organization dedicated to promoting awareness of periodontal science, the science and clinical practice of periodontics and implant dentistry, and the importance of periodontal health.

Evidence-based health care the conscientious, explicit, and judicious use of current best evidence in making decisions about the care of individual patients; requires the integration of individual clinical expertise and patient preferences with the best available external clinical evidence from systematic research.

Evidence-based practice the clinical application of evidence-based health care to making decisions about the care of individual patients. The practice of evidence-based dental hygiene requires the integration of individual clinical expertise and patient preferences with the best available external clinical evidence from systematic research.

Evocation the concept that the best ideas about change come from the patient. The clinician encourages the patient to do the talking. This is how the clinician learns about the way the patient views the situation and finds out about any internal motivation the patient may have. The clinician does not attempt to persuade the patient.

Evoking a communicative method that encourages the patient to express his or her motivation and confidence to change. The OARS skills are used to encourage patients to talk about and elaborate on their reasons for change and, as necessary, to acknowledge reasons he or she does not want to change.

Extent of inflammation the degree to which the inflammation has spread throughout the mouth. The extent of inflammation is described as localized or generalized in the mouth.

Extent the degree or amount of periodontal destruction and can be characterized based on the number of sites that have experienced tissue destruction.

External basal lamina a thin mat of extracellular matrix between the epithelial cells of the junctional epithelium and the gingival connective tissue. Also see internal basal lamina.

Extracellular matrix a mesh-like network that surrounds the cells; this network helps to hold cells together and provides a biomechanical framework within which cells can migrate and interact with one another.

Extracellular polymeric substance an enveloping protective substance secreted by plaque-forming bacteria.

Extracellular protective matrix an enveloping protective substance secreted by plaque-forming bacteria.

Exudate pus that can be expressed from a periodontal pocket; sometimes called suppuration.

F

Factitious injury an injury wherein an individual deliberately damages his/her own body with premediated, malingering intent. This injury usually happens in individuals suffering from maladjusted cognitive and behavioral processes. A factitious injury is also known as a self-injury.

Familial aggregation clustering of certain traits, behaviors, or disorders within a given family.

Fenestration a "window" of bone loss bordered by alveolar bone on its coronal aspect.

Fiber bundles of the periodontal ligament a specialized connective tissue that surrounds the root of the tooth and connects it to the alveolar bone. These fibers are the largest component of the periodontal ligament space. These fibers are commonly referred to as periodontal ligament fibers.

Filament-type irrigation tips these tips have three soft filaments surrounding a standard jet tip. Around a dental implant, manual toothbrushing paired with the use of an irrigation device with a filament tip is twice as effective as the combined use of manual brushing and flossing and better at reducing bleeding.

Flap elevation a step done in periodontal surgery to separate the soft tissue from the underlying tooth root and alveolar bone.

Flap for access a periodontal surgical technique that involves incising through the gingival tissue, then elevating the flap to facilitate enhanced access to the root for instrumentation. Also see open flap debridement.

Fluid channels a series of channels that penetrate the extracellular slime layer of a biofilm that provide nutrients and oxygen for the bacterial microcolonies and facilitate movement of bacterial metabolites, waste products, and enzymes within the biofilm structure. Also see biofilm and extracellular protective matrix .

Flushing zone the depth of fluid penetration within a subgingival sulcus or pocket as it pertains to the mechanism of action of a power-driven water irrigation device.

Focusing collaborating with the patient to find a clear direction or goal.

Food impaction forceful wedging of food (such as pieces of tough meat) between teeth during chewing, trapping the food in the interdental area.

Frankl Behavioral Rating Scale a system to classify and document behavior of children and adolescents; the scale divides observed behavior into four categories definitely negative, negative, positive, and definitely positive.

Free gingiva the unattached portion of the gingiva that surrounds the tooth in the region of the cementoenamel junction; also known as the unattached gingiva or the marginal gingiva.

Free gingival groove a shallow linear depression that separates the free and attached gingiva; this line is rarely visible to the naked eye.

Free soft tissue autograft (FSTA) a type of periodontal plastic surgery that was one of the first procedures used to augment the width of attached gingiva. The free soft tissue autograft requires harvesting a donor section of tissue, usually from the palate, so there are two intraoral wounds that are created during this surgery (at the donor site and the recipient site).

Fremitus a palpable or visible movement of a tooth when in function.

Frenal pull abnormal frenal attachment located in close proximity to the gingival margin which “pulls” the gingiva away from the tooth, thereby leading to gingival recession.

Frenectomy a type of periodontal plastic surgery that is done to excise the frenum, including the attachment of the frenum to bone.

Full-thickness flap a type of flap elevation that includes elevating the entire thickness of the soft tissue (including epithelium, connective tissue, and periosteum); the full-thickness flap provides complete access to the underlying bone that might be needed when bone replacement grafting or osseous recontouring are anticipated. A full-thickness flap is also known as a mucoperiosteal flap.

Functional crown lengthening a type of crown lengthening surgery performed when the remaining tooth structure is inadequate to support a needed restoration. Functional crown lengthening augments the outcome of a restorative dental procedure (such as a crown) done on a tooth with inadequate axial tooth height.

Functional occlusal forces normal occlusal forces produced during the act of chewing food. Also see parafunctional occlusal forces.

Furcation involvement an osseous defect that results in a loss of alveolar bone between the roots of a multirooted tooth.

Furcation probe used to assess furcation involvement on multirooted teeth. A common type of furcation probe is the Nabers probe.

G

Generalized pattern a form of periodontitis characterized by generalized interproximal attachment loss affecting at least three permanent teeth other than the first molars and incisors; most of the permanent teeth are usually affected.

Generalized periodontitis periodontitis in which 30% or more of the teeth in the mouth exhibit attachment loss and/or bone loss.

Genetic testing a type of medical test that identifies changes in chromosomes, genes, or proteins. The results of a genetic test can confirm or rule out a suspected genetic condition or help determine a person’s chance of developing or passing on a genetic disorder.

Genuine halitosis a form of malodor that can be objectively verified.

Gingiva the tissue that covers the cervical portions of the teeth and the alveolar processes of the jaws.

Gingival abscess an abscess of the periodontium that is primarily limited to the gingival margin or interdental papilla without involvement of the deeper structures of the periodontium. Also see periodontal abscess and pericoronal abscess.

Gingival crevicular fluid a fluid that flows into the sulcus from the adjacent gingival connective tissue; the flow is slight in health and increases in disease.

Gingival epithelium a specialized stratified squamous epithelium that functions well in the wet environment of the oral cavity; the microscopic anatomy of the gingival epithelium is similar to the epithelium of the skin. Also see oral epithelium, sulcular epithelium, and junctional epithelium.

Gingival fibers see supragingival fiber bundles.

Gingival inflammation on a reduced periodontium in a successfully treated stable periodontitis patient the return of biofilm-induced inflammation to the gingival margin on a reduced periodontium with no ongoing evidence of progressive attachment loss on a previously diagnosed periodontitis patient.

Gingival margin the thin, rounded edge of free gingiva that forms the coronal boundary, or upper edge, of the gingiva. In health, the gingival margin follows the scalloped, curved contours of the underlying CEJ.

Gingival pocket a deepening of the gingival sulcus as a result of swelling or enlargement of the gingival tissue. Also see periodontal pocket.

Gingival sulcus the *space* between the free gingiva and the tooth surface.

Gingivectomy a soft tissue resective periodontal surgical procedure designed to excise (cut away) and to remove some of the gingival tissue. Historically, the gingivectomy was used for many years in periodontics as a primary treatment modality, but it plays a greatly reduced role in modern dentistry. Also see gingivoplasty.

Gingivitis on a reduced periodontium in a non-periodontitis patient the presence of dental biofilm-induced inflammation on a periodontium with preexisting loss of connective tissue and/or loss of alveolar bone that can be attributed to non-periodontitis reasons.

Gingivitis on an intact periodontium the presence of dental biofilm-induced inflammation on an intact periodontium.

Gingivitis an inflammation of the periodontium that is confined to the gingiva resulting in damage to the gingival tissue that is reversible.

Gingivoplasty a soft tissue resective periodontal surgical procedure performed to reshape the surface of the gingiva to create a natural form and contour to the gingiva. Unlike a gingivectomy, gingivoplasty implies reshaping the surface of the gingiva without removing any of the gingiva actually attached to the tooth surface. Also see gingivectomy.

Glycemic control a medical term referring to the typical levels of blood sugar (glucose) in a person with diabetes mellitus; optimal management of diabetes involves patients measuring and recording their own blood glucose levels daily.

Grade modifiers risk factors that may impact the rate of disease progression.

Grade an estimate of the future rate of progression of periodontitis, that is, slow, moderate, or rapid; it measures the responsiveness to conventional periodontal therapy and the potential impact on systemic health.

Gram staining a laboratory method that reveals differences in the chemical and physical properties of bacterial cell walls that divides bacteria into Gram-positive (purple color) and Gram-negative (red color) bacterial cell wall types. See Gram-positive bacteria and Gram-negative bacteria.

Gram-negative bacteria bacteria with a thin cell wall that is surrounded by an outer membrane and an inner membrane that shows a red stain under the microscope; it is believed to play an important role in inflammatory periodontitis. See Gram-staining and Gram-positive bacteria.

Gram-positive bacteria bacteria with a single, thick cell wall that show a purple stain under the microscope; most of the bacteria associated with a healthy periodontium are Gram-positive. See Gram-staining and Gram-negative bacteria.

Granulocytes a leukocyte carrying granules within the cytoplasm; the most abundant immune cell type found in peripheral blood.

Guided tissue regeneration (GTR) a periodontal surgical procedure employed to stimulate regeneration of lost periodontal structures (i.e., to regrow lost cementum, lost periodontal ligament, and lost alveolar bone).

Guiding style a form of communication whereby the clinician does not tell the patient what to do, but rather asks the patient what his or her thoughts are toward healthy behavior change, but not in a directive and paternalistic way.

H

Halitophobia an extreme fear and irrational preoccupation of bad malodor that does not actually exist; a variant of pseudo-halitosis; also known as delusional halitosis.

Halitosis a worrisome problem that can stigmatize an individual suffering from oral malodor.

HbA$_1$C level the average blood glucose level over the past 2 to 3 months (which corresponds to the average life span of the red blood cell); the HbA1C test measures the amount of glycosylated hemoglobin in the blood.

Hemidesmosome a specialized cell junction that connects the epithelial cells to the basal lamina. Hemidesmosomes also attach the junctional epithelium to the enamel in teeth without gingival recession.

Hidden signs signs of inflammation in the periodontium that are not readily apparent.

Histology a branch of anatomy concerned with the study of the microscopic features of tissues.

Homeostasis an internal equilibrium/balance, such as an internal equilibrium within the biofilm that ensures the safety and survival of all microorganisms living in the biofilm community without harming the host.

Horizontal bone loss a common pattern of bone loss resulting in a fairly uniform reduction in the height of the alveolar bone with the margin of the alveolar crest remaining perpendicular to the long axis of the tooth. Also see alveolar bone loss and vertical bone loss.

Horizontal incision a type of incision that runs parallel to the gingival margin in a mesiodistal direction.

Horizontal tooth mobility movement of a tooth in a facial-to-lingual direction. Horizontal mobility is assessed using the blunt ends of two dental instrument handles on either side of the tooth.

Horizontal transmission the transfer of bacteria between individuals of the same generation.

Hospital-acquired pneumonia a type of pneumonia contracted during a stay in a hospital or long-term care facility. Hospital-acquired pneumonia is not caused by the same organisms that cause community-acquired pneumonia.

Host modulation therapy a new type of therapy that counters periodontal disease by modulating the host inflammatory response to the bacterial challenge, rather than directly targeting the pathogens.

Host response the way that an individual's body responds to an infection. Also see immune system and inflammation.

Host a human in or on which another organism lives; in the case of periodontal disease, bacterial pathogens infect the host (individual with periodontal disease).

Human immunodeficiency virus (HIV) a virus that damages the immune system and interferes with the body's ability to fight infection and disease. If not controlled by antiviral medication, HIV can potentially manifest as AIDS.

Hydrokinetic activity the energy created by fluids in motion; in dentistry, the pulsating fluid delivered by dental water jet creates two zones of fluid movement: the impact zone and the flushing zone.

Hyperglycemia abnormally high concentration of glucose circulating in blood that occurs especially in individuals with diabetes mellitus.

Hyposalivation the objective reduction in salivary flow rate verified by actual measurements of stimulated or unstimulated salivary flow.

I

Iatrogenic factor dental treatment that results in an inadvertent, adverse outcome. Some examples of iatrogenic factors that increase plaque retention are improperly contoured restorations, overhanging restorative margins, open restorative margins, open contacts, orthodontic appliances, or a poorly designed dental prosthesis.

Iatrosedation the act of making a patient calm based on the doctor's behavior. Also see pharmacosedation..

Immune system a collection of responses that protects the body against infections by bacteria, viruses, fungi, toxins, and parasites; the immune system defends the body against invading microorganisms, as well as toxins in the environment.

Immunoglobulin Y-shaped proteins; the five major classes of immunoglobulin are immunoglobulin M (IgM), immunoglobulin D (IgD), immunoglobulin G (IgG), immunoglobulin A (IgA), and immunoglobulin E (IgE).

Immunoinflammatory response a natural host defense mechanism that is triggered by invading microorganisms. The immunoinflammatory response involves the activation of a wide array of different types of leukocytes and the release of pro- and anti-inflammatory mediators.

Impact zone the area of initial fluid contact to the portion of the tooth near the gingival margin.

Implant abutment a titanium post that connects to the implant body and emerges partially or completely through the gingival tissue into the mouth.

Implant body the portion of the implant system that is surgically placed into the living alveolar bone; sometimes referred to as the implant fixture or implant. The implant body is analogous to the root of a tooth.

Inactive disease site an area of tissue destruction that is stable, with the attachment level of the junctional epithelium remaining unchanged over time.

Incidence the number of new disease cases in a population that occur over a given period of time. Also see prevalence.

Inflammation the body's reaction to injury or invasion by disease-producing organisms that focus host defense components at the site of the infection to eliminate microorganisms and heal damaged tissue. Inflammation is part of the immune response. Also see acute inflammation, chronic inflammation, and immunoinflammatory response.

Inflammatory biochemical mediator biologically active compounds secreted by cells that activate the body's inflammatory response; inflammatory mediators of importance in periodontitis are the interleukins, prostaglandins, and matrix metalloproteinases.

Informed refusal a person's right to refuse all or a portion of the proposed recommended treatment, alternate treatment options, and the likely consequences of declining treatment have been explained in language understood by the patient. Also see informed consent.

Infrabony defect an osseous defect in the alveolar bone resulting in bone resorption that occurs in an uneven, oblique direction.

Infrabony pocket a periodontal pocket in which there is vertical bone loss, and the junctional epithelium, forming the base of the pocket, is located *apical* to the crest of the alveolar bone. The base of the pocket is located within the cratered-out area of the bone alongside of the root surface. Also see periodontal pocket and suprabony pocket.

Innervation nerve supply; innervation to the periodontium occurs via the branches of the trigeminal nerve.

Intact periodontium a periodontium with an absence of detectable attachment and/or bone loss.

Intentional torts actions designed to injure another person or that person's property.

Interdental col a valley-like depression in the portion of the interdental gingiva that lies directly apical to the contact area of two adjacent (touching) teeth and connects the facial and lingual papillae.

Interdental gingiva the portion of the gingiva that fills the interdental embrasure between two adjacent teeth apical to the contact area. The interdental gingiva consists of the facial interdental papilla and the lingual interdental papilla which are connected to each other via the gingival col.

Interdisciplinary care the close collaboration of two or more health care providers from different disciplines to address the needs of the patient and establish an optimal care plan that results in enhanced health outcomes.

Intermittent disease progression theory states that periodontal disease progression is characterized by periods of disease activity and inactivity (remission). See episodic.

Internal basal lamina a thin mat of extracellular matrix between the epithelial cells of the junctional epithelium and the tooth surface. Also see external basal lamina.

Internal bevel incision a type of horizontal incision employed during periodontal flap surgery in which the surgical scalpel enters the marginal gingiva, but is not placed directly into the crevice or sulcus; the scalpel blade enters the gingival margin approximately 0.5 to 1.0 mm away from the margin and follows the general contour of the scalloped marginal gingiva. Also see horizontal incision and crevicular incision.

Interprofessional collaborative practice a process whereby health care providers from different professional backgrounds work together to deliver the highest quality of patient-centered care. For example, interprofessional collaborative practice occurs when dental and medical practitioners work together in tandem to treat an individual with both periodontal disease and diabetes.

Irreversible tissue damage permanent tissue damage of periodontitis, which includes the apical migration of the junctional epithelium, the loss of connective tissue attachment, and the loss of alveolar bone.

J

Junctional epithelium the specialized epithelium that forms the base of the sulcus and joins the gingiva to the tooth surface. Also see gingival epithelium.

K

Keratin a tough, fibrous structural protein that protects the outer layer of the oral epithelium.

Keratinization the process by which epithelial cells on the surface of the skin become stronger and waterproof. Also see keratinized epithelial cells and nonkeratinized epithelial cells.

Keratinized epithelial cells cells that have no nuclei and form a tough, resistant layer on the surface of the skin.

Keystone pathogen–host response a novel plaque hypothesis that expands upon the microbial homeostasis–host response hypothesis by postulating that a specific bacterial species is key to creating the shift from symbiotic microbes to dysbiotic microbes in the biofilm community. In turn, the dysbiotic biofilm community triggers the uncontrolled host response that results in damage to the periodontal tissues.

Knee-to-knee position a common approach to performing a dental examination on a young infant patient. The provider sits knee-to-knee with the caregiver and the infant patient lays on his/her back on the provider's lap.

L

Lamina dura radiographic term that describes the thin layer of dense bone that lines a normal tooth socket.

Laterally positioned flap a type of periodontal plastic surgery technique that can be used to cover root surfaces with gingiva in isolated sites of gingival recession by laterally displacing a flap to cover gingival recession on an adjacent tooth..

Leukemia a type of cancer that begins in blood cells in which the bone marrow produces a large number of abnormal white blood cells that do not function properly.

Leukocytes white blood cells that act much like independent single-cell organisms able to move and capture microorganisms on their own.

Levels of evidence a ranking system used in evidence-based health care to describe the strength of the results measured in a clinical trial or research study.

Liability a health care provider's obligation or responsibility to provide services to another person (the patient). The health care provider's liability entails the possibility of being sued if the person receiving the services feels as if he has been treated improperly or negligently.

Lichen planus a common inflammatory condition that can affect the skin, hair, nails, and mucous membranes. On the skin, lichen planus usually appears as purplish, often itchy, flat-topped bumps, developing over several weeks; in the mouth and other areas covered by a mucous membrane, lichen planus forms lacy white patches, sometimes with painful sores.

Linear gingival erythema (LGE) a gingival manifestation of immunosuppression characterized by a distinct linear erythematous (red) band that is limited to the free gingiva; formerly known as HIV-associated gingivitis.

Local (intraoral) contributing factors Secondary factors that are present in the oral cavity which contribute to disease progression, either elevating the risk of an individual to developing gingivitis and periodontitis or elevating the extent and severity of an already established gingivitis and periodontitis.

Localized periodontitis periodontitis in which less than 30% of the teeth in the mouth exhibit attachment loss and/or bone loss.

Long junctional epithelium a form of periodontal repair that is characterized by the apical downgrowth of the junctional epithelium. This type of healing primarily occurs following periodontal instrumentation of a diseased root surface. Healing by long junctional epithelium is not the same as periodontal regeneration.

Lymph nodes small bean-shaped structures located on either side of the head, neck, armpits, and groin; these nodes filter out and trap bacteria, fungi, viruses, and other unwanted substances to safely eliminate them from the body.

Lymphatic system a network of lymph nodes connected by lymphatic vessels that plays an important role in the body's defense against infection.

Lymphocytes small leukocytes that play an important role in recognizing and controlling foreign invaders; the three types of lymphocytes are B-lymphocytes, T-lymphocytes, and NK-lymphocytes. Also see B-lymphocytes, T-lymphocytes, and NK-lymphocytes.

Lysosome granules found in the cytoplasm of PMNs that are filled with strong bactericidal and digestive enzymes; these granules can kill and digest bacterial cells after phagocytosis. Also see phagocytosis.

M

Macronutrients a type of food required in large amounts in the diet. Fats, proteins, and carbohydrates are classified as macronutrients.

Macrophage large phagocytic leukocytes (located in the tissue) that have one kidney-shaped nucleus and some granules. Also see monocytes.

Malingering the intentional injury to the tissues by the patient to deliberately feign or exaggerate a physical or psychological symptom with the goal of receiving a reward.

Malpractice the improper or negligent treatment by a health care provider that results in injury or damage to the patient.

Marginal gingivitis a type of gingivitis in which the distribution of inflammation may affect the gingival margin and the papilla.

Master treatment plan sequential outline of the steps to be carried out by the dentist, the dental hygienist, and the patient to eliminate disease and restore a healthy periodontal environment.

Matrix metalloproteinases (MMP) a family of at least 12 different enzymes produced by various cells of the body that can act together to break down the connective tissue matrix; the presence of increased MMP levels causes extensive collagen destruction in the periodontal tissues.

Medical lasers a device that produces an intense, narrow beam of light with a single wavelength that can be focused at a precise location. Investigations are still ongoing to determine the effectiveness of using lasers to treat and manage periodontal disease.

Medication-induced xerostomia and hyposalivation dry mouth that occurs when patients take multiple medications simultaneously to treat one or more conditions.

Medication-related osteonecrosis of the jaw (MRONJ) a rare disorder characterized by painful areas of exposed bone in the mouth that fail to heal after an extraction or oral surgery procedure. In dentistry, patients on bisphosphonates, antiresorptive agents, and antiangiogenic medications may be at risk of developing MRONJ following dental surgery.

MEDLINE a free online index that enables quick access to locate relevant clinical evidence in the published medical, dental, and allied health literature; hosted by the National Library of Medicine.

Membrane attack complex a protein unit created by the complement system that is capable of puncturing the cell membranes of certain bacteria. Also see complement system.

Menopausal gingivostomatitis decreased levels of circulating hormones in women who are menopausal or postmenopausal that may result in oral changes, such as thinning of the oral mucosa, dry mouth, burning sensations, altered taste, gingival recession, and alveolar bone loss.

Metabolic syndrome a group of risk factors that place an individual at increased risk of developing several major chronic disorders, such as heart disease, diabetes, and stroke.

Metastatic infection the spread of a pathogen from its initial site to a different secondary site inside the host's body. Periodontal pathogens have the potential to travel to distant sites in the body and cause infections, such as a lung infection or endocarditis.

Microbe all types of microscopic organisms—namely bacteria, fungi, protozoa, and viruses.

Microbial blooms periods that occur when specific species or groups of species grow at rapidly accelerated rates.

Microbial homeostasis–host response hypothesis a novel plaque hypothesis that proposes that although plaque biofilms are the cause of the initial inflammatory response leading to gingivitis, the pathogenic bacteria are not the direct cause of the destruction of tissues seen in periodontitis.

Microbial reservoir a niche or secure place in the oral cavity that can allow periodontal pathogens to live undisturbed during routine therapy and subsequently repopulate periodontal pockets quickly.

Micronutrients nutrients that are needed in trace quantities for normal growth and development of living organisms. Vitamins and calcium are examples of micronutrients.

Miller classification system another type of gingival recession classification system based on the level of gingival margin with respect to the mucogingival junction and the underlying alveolar bone; the system consists of four recessions categories Also see Cario classification system

Modeling an iatrosedation technique used to calm an anxious child. This approach relies on the caregiver or another patient functioning as a model for the child by exhibiting the specific behavior that needs to be imitated.

Modeling is an effective iatrosedation technique to use for adults with behavioral issues or cognitive disabilities.

Molar/incisor pattern a form of periodontitis characterized by localized attachment loss affecting the first molars and/or incisors and involving no more than two teeth other than first molars and incisors; previously called localized juvenile periodontitis and localized aggressive periodontitis.

Monocytes phagocytic leukocytes located in the bloodstream. Also see macrophage.

Morphology the study of the anatomic surface features of the teeth.

Motivational interviewing (MI) defined as a patient-centered method for enhancing a patient's motivation for behavior change by exploring the patient's mixed feelings about change.

Mouth breathing the process of inhaling and exhaling air primarily through the mouth, rather than the nose. It often occurs while the patient is sleeping. Mouth breathing tends to dry out the oral mucosa, especially in the anterior region of the mouth.

Mucogingival defects deviations from the normal anatomic relationship between the gingival margin and the mucogingival junction.

Mucogingival deformity and condition encompasses any type of defect that is associated with a deviation from the normal anatomic/morphologic relationship between the gingiva and the alveolar mucosa.

Mucogingival junction the clinically visible boundary where the pink attached gingiva meets the red, shiny alveolar mucosa.

Mucogingival surgery terminology used in the past to describe periodontal surgical procedures that alter the relationship between gingiva and mucosa. Some of the periodontal plastic surgical procedures utilized in modern dentistry were previously described as mucogingival surgical procedures and this older terminology can still be encountered. Also see periodontal plastic surgery.

Mucogingival the portion of the oral mucosa that covers the alveolar process including the gingiva (keratinized tissue) and the adjacent alveolar mucosa.

Mushroom-shaped microcolonies a cluster of microbes that are attached to the tooth surface at a narrow base.

N

National Health and Nutrition Examination Survey (NHANES) a large-scale program of studies to assess the health and nutritional status of adults and children in the United States. This survey protocol combines a survey with a physical examination.

Natural killer (NK)-lymphocytes an unusual form of lymphocytes that share the characteristics of both a T-lymphocyte and an NK-lymphocyte. They are noticeably larger than B- and T-lymphocytes; their cytoplasm contains granules (containing proteases and perforin), whereas B- and T-lymphocytes do not have granules; they have the attributes of both innate immunity and adaptive immunity.

Necrosis cell death. For example, in the case of necrotizing gingivitis, necrosis refers to the death of the cells comprising the gingival epithelium.

Necrotizing gingivitis tissue necrosis that is limited to the gingival tissues.

Necrotizing periodontal disease (NPD) a unique type of periodontal disease that involves tissue necrosis (localized tissue death); characterized by painful infection with ulceration, swelling, and sloughing off of dead epithelial tissue from the gingiva. Also see necrotizing gingivitis and necrotizing periodontitis.

Necrotizing periodontitis tissue necrosis of the gingival tissues combined with loss of attachment and alveolar bone loss.

Necrotizing stomatitis an extension of either necrotizing gingivitis or necrotizing periodontitis where the necrosis progresses to deeper tissues beyond the mucogingival line, such as the lip or cheek mucosa.

Negligence a failure to exercise reasonable care to avoid injuring others. It is the failure to do something that a reasonable person would do under the same circumstances, or the doing of something a reasonable person would not do. Negligence is characterized by carelessness, inattentiveness, and neglectfulness rather than by a deliberate intent to cause injury.

Neutropenia a polymorphonuclear leukocyte (PMN) count of less than 1,500 cells/μL of blood; indicates an increased risk of infection.

Neutrophil phagocytic immun cells that actively engulf and destroy microorganisms; PMNs play a vital role in combating the pathogenic bacteria responsible for periodontal disease; also known as polymorphonuclear leukocytes.

Neutrophilia a polymorphonuclear leukocyte (PMN) count above 6,000 cells/mL of blood; may be an indication of a chronic inflammation, prolonged infection, or neoplastic condition

New attachment a term used to describe the union of a *pathologically exposed root* with connective tissue or epithelium. Also see reattachment.

Nifedipine a calcium channel blocker used as a coronary vasodilator in the treatment of hypertension, angina, and cardiac arrhythmias; associated with drug-influenced gingival enlargement.

Nonabsorbable suture a suture made from a material that does not dissolve in body fluids; a clinician must remove the nonabsorbable sutures after healing of the wound has occurred. Also see absorbable suture.

Noncombustible nicotine products a novel nicotine delivery system that neither contains tobacco nor undergoes an active burning process. These are battery-powered devices composed of a heating element and a container filled with liquid nicotine derived naturally from tobacco or synthetic liquid nicotine.

Noncombustible tobacco delivery system contains tobacco that is heated using a battery-operated electronic device; instead of being ignited and burned, as with a traditional cigarette.

Noncompliant patient patients who do not follow recommendations for health care advice. Examples of noncompliant patients would be a patient that does not take medications daily to control diabetes as prescribed by a physician or a patient that does not follow instructions for performing daily self-care. Also see compliance.

Non-controlled-release chemotherapeutic agent a locally delivered therapeutic chemical agent directly applied into the periodontal pocket and used as an adjunct to mechanical periodontal therapy.

Nondental biofilm-induced gingival diseases and conditions (may also be referred to as nondental plaque-induced gingival diseases and conditions) a less common, but equally important, form of periodontal disease that is not caused by dental biofilm. They may be manifestations of systemic disorders or may represent pathologic changes limited to the gingival tissues.

Nondisplaced flap a periodontal flap that is sutured with the margin of the flap at its original position in relationship to the CEJ on the tooth. Also see displaced flap.

Nonkeratinized epithelial cells cells that have nuclei and act as a cushion against mechanical stress and wear. Nonkeratinized epithelial cells are softer and more flexible.

Nonpharmacological therapy any type of therapy that calms the patient without the use of medication. Iatrosedation is an example of nonpharmacological therapy.

Nonresponsive disease sites areas in the periodontium that show deeper probing depths, continuing loss of attachment, or continuing clinical signs of inflammation in spite of thorough and meticulous periodontal therapy.

Nonspecific plaque hypothesis an outdated hypothesis that postulated the accumulation of bacterial biofilms leads to periodontal disease.

Nonsteroidal anti-inflammatory drugs (NSAIDs) medications used for many years in medical and dental care to treat pain, acute inflammation, and chronic inflammatory conditions. NSAIDs have been evaluated for their effect on periodontitis (a disease intimately associated with inflammation). NSAIDs can reduce tissue inflammation by inhibiting prostaglandins including PGE2.

Nonsurgical periodontal therapy phase a phase of nonsurgical periodontal therapy that includes self-care measures, periodontal instrumentation, and use of chemical agents to prevent or control plaque-induced gingivitis or periodontitis.

Normal mucogingival condition the absence of a disease state—for example, the absence of gingival recession, gingivitis, and periodontitis.

O

OARS the four core communication skills: Open-ended questions, Affirmations, Reflections, and Summaries.

Obesity an excess amount of body fat in proportion to lean body mass, to the extent that health is impaired.

Objective fear a fear resulting from a direct experience. It is created when an individual has a negative experience in the dental office, such as feeling pain or not understanding precisely what is happening.

OMG (overlap, meet, gap) three possible relationships of cementum and enamel at the cementoenamel junction: 1) Overlap—the cementum overlaps the enamel for a short distance, 2) Meet—the cementum meets the enamel in edge-to-edge contact, 3) Gap—there is a small gap between the cementum and enamel, exposing the dentin in that area.

Open flap debridement a periodontal surgical procedure that is quite similar in concept and execution to flap for access surgery; usually includes more extensive flap elevation than flap for access—providing access not only to the tooth roots but also to all of the alveolar bone defects. Also see flap for access.

Open margin a space or gap between the edge of a restoration and the tooth structure. An open margin is considered to be a local contributing factor since it is an area that can harbor biofilm.

Open-ended questions questions that cannot be answered with a simple yes or no response.

Operculum a flap of gingival tissue covering part of the occlusal surface of a partially erupted tooth. This tissue flap can become infected, and this type of infection under the flap of tissue is referred to as a pericoronal abscess.

Opsonization coating of the surface of a microorganism by complement components to facilitate the engulfment and destruction by phagocytes.

Oral biofilm a polymicrobial, three-dimensional community embedded in a protective matrix that consists of microbial metabolic products and/or host components, such as salivary glycoproteins.

Oral epithelium portion of the gingival epithelium that covers the outer surface of the free gingiva and attached gingiva; it extends from the crest of the gingival margin to the mucogingival junction. The oral epithelium is the only part of the periodontium that is visible to the unaided eye. Also see gingival epithelium.

Oral malodor an unpleasant or offensive oral odor which arises from either oral or systemic sources.

Oral mucositis an inflammation of the oral mucous membranes caused when chemotherapy attacks and kills the rapidly dividing cells of the mucous membranes.

Organoleptic test (OLT) a sensory test that involves having a trained judge smell a patient's expired air to make an assessment whether the breath is unpleasant. The assessment depends on the olfactory organs of the judge.

Orthodontic irrigation tips these tips have a soft tapered brush that enhances biofilm removal and provides for simultaneous irrigation. Orthodontic tips can be used for full-mouth irrigation and can be recommended for people with or without orthodontic appliance implants, or who need additional help with biofilm removal.

Osseointegration the direct contact of living alveolar bone with the surface of the dental implant body (with no intervening periodontal ligament). Osseointegration is a major requirement for implant success.

Osseous crater a bowl-shaped osseous defect in the interdental alveolar bone with bone loss nearly equal on the roots of two adjacent teeth. Also see infrabony defect.

Osseous defect a deformity in the tooth supporting alveolar bone usually resulting from periodontitis. Also see infrabony defect, osseous crater, and furcation involvement.

Ostectomy removal of alveolar bone that is attached to the tooth via the PDL fibers (i.e., that is still providing some support for the tooth). The removal of small amounts of supporting bone is justified by the attainment of alveolar bone contours that are compatible with the natural contours of the gingiva. Also see osteoplasty.

Osteoblasts bone-forming cells.

Osteoclasts bone resorbing cells.

Osteoconduction a physical, passive process whereby the graft material itself acts as a scaffold for host bone-forming cells (existing outside the graft) to migrate, attach, and grow on.

Osteogenesis the production of new bone; in periodontal surgery, osteogenesis is the potential for new bone cells and new bone to form following bone grafting.

Osteoinduction refers to a chemical process whereby complex, biologically active signaling molecules contained in the graft material—known as bone morphogenetic proteins (BMPs)—actively recruit and attract host bone-forming cells (osteoblasts) to the site of implantation to form new bone.

Osteoplasty reshaping the surface of alveolar bone without actually removing any of the supporting bone. Also see ostectomy.

Osteoporosis a reduction in bone mass that causes an increased susceptibility to fractures; occurs most frequently in postmenopausal women, in sedentary or bedridden individuals, and in patients receiving long-term steroid therapy.

Osteoprotegerin (OPG) protein signaling molecule produced by osteoblasts that plays a key role in bone remodeling.

Overhanging restoration a dental restoration that is not smoothly contoured with the tooth surfaces (also called an overhang).

Overt signs signs of inflammation in the periodontium that are readily apparent.

P

Papillary gingivitis a type of gingivitis in which the distribution of inflammation may be limited to affect only the interdental papilla.

Parafunctional occlusal forces occlusal forces that result from tooth-to-tooth contact made when not in the act of eating. Also see functional occlusal forces.

Partial-thickness flap or split-thickness flap as it is also called, includes elevation of only the epithelium and a thin layer of the underlying connective tissue rather than the entire thickness of the underlying soft tissues.

Partnering with the patient an active collaborative process involving the health care practitioner and the patient to reinforce and monitor the patient's efforts at self-care. Partnering with the patient can help enhance compliance and motivation.

Partnership The first key element of motivational interviewing, which is between the patient and clinician. The health care practitioner avoids the "expert" role; instead, the patient and practitioner work collaboratively.

Pathogenesis the sequence of events that occur during the development of a disease or abnormal condition.

Pathologic tooth migration a complication that occurs when severe periodontal inflammation disrupts the continuity of the transseptal fibers, thereby leading to tooth displacement. Pathologic tooth migration may be a foreboding sign of severe periodontitis and may be a motivating factor for patients to seek treatment.

Pathophysiology the study of the pathological manifestations of periodontitis; it does not deal directly with the treatment of disease, rather, it explains the processes within the body that result in the signs and symptoms of periodontitis.

Patient decision aids information resources such as written materials, videos, or web-based tools that are designed to facilitate shared decision-making and patient participation in health care decisions. These are the best known and proven tools to support shared decision-making.

Patient-centered a philosophy of health care that recognizes the patient's dignity, and right of choice in all matters, without exception, related to health care; for example, consideration of behavior changes and treatment planning is viewed from the patient's perspective rather than the clinician's perspective.

Pediatric dentistry triad a collaborative teamwork effort that involves close interaction between the dental clinician, child patient, and parent (or caregiver). Establishing this relationship builds trust between all participants of the triad and serves to facilitate a positive outcome for the child.

Peer-reviewed journals a journal that uses a panel of experts to review research articles for study design, statistics, and conclusions prior to publication.

Pellicle a thin, bacteria-free membrane that forms on the surface of the tooth during the late stages of eruption.

Pericoronal abscess an abscess of the periodontium that involves tissues around the crown of a partially erupted tooth. The pericoronal abscess is also referred to as pericoronitis.

Pericoronitis see pericoronal abscess.

Peri-implant health the healthy soft tissue surrounding an implant with no visual signs of inflammation, absence of profuse bleeding on probing, and stable pocket depths that do not increase over time.

Peri-implant mucositis plaque-induced gingivitis that is localized in the gingival tissues surrounding a dental implant. Also see peri-implantitis.

Peri-implant tissues the periodontal tissues that surround the dental implant.

Peri-implantitis periodontitis in the tissues surrounding an osseointegrated dental implant, resulting in loss of alveolar bone. Also see peri-implant mucositis.

Perimucosal seal a cuff-like biological seal the epithelium adapts to the abutment post or to the implant itself; the seal functions as a barrier between the implant and the oral cavity.

Periodontal abscess a localized collection of pus that forms in a circumscribed area of the periodontal tissues that affects the deeper structures of the periodontium as well as the gingival tissues. Also see gingival abscess and pericoronal abscess.

Periodontal biotype describes features of the periodontium, such as gingival thickness, gingival width, and the shape of the dental crown, that are influenced by both genetic and environmental factors; it refers to features of the soft tissues and the teeth they surround. Also referred to as periodontal phenotype.

Periodontal disease recurrence the return of the disease in a patient that has been previously, successfully treated for periodontitis.

Periodontal dressing a protective material applied over a periodontal surgical wound and used somewhat like using a bandage to cover a finger wound. Periodontal dressings can be placed to facilitate flap adaptation and are frequently indicated when the surgical procedures have created varying tissue levels or when displaced flaps are used.

Periodontal flap a surgical procedure in which incisions are made in the gingiva or mucosa to separate the surface tissues (epithelium and connective tissue) from the underlying tooth roots and underlying alveolar bone. Also see full-thickness flap, partial-thickness flap, nondisplaced flap, and displaced flap.

Periodontal health on a reduced periodontium in a nonperiodontitis patient a periodontium with a presence of detectable attachment and/or bone loss. Note that a reduced periodontium can be stable (periodontitis in remission) or it can exhibit signs of active disease.

Periodontal health on a reduced periodontium in a successfully treated stable periodontitis patient clinical signs of periodontal health on a periodontium with a preexisting loss of connective tissue and alveolar bone that is attributed to periodontitis but that has been successfully treated and is currently stable.

Periodontal health on an intact periodontium clinical signs characteristic of periodontal health coupled with an intact periodontium (a periodontium with an absence of detectable attachment and/or bone loss).

Periodontal health a state free from inflammatory periodontal disease that allows an individual to function normally and avoid consequences (mental or physical) due to current or past disease.

Periodontal instrumentation an essential component of nonsurgical periodontal therapy. The thoroughness of periodontal instrumentation determines the overall success of nonsurgical periodontal therapy in most patients.

Periodontal ligament a thin sheet of fibrous connective tissue that surrounds the roots of the teeth and joins the root cementum to the socket wall; These fibers attach to the bone of the socket on one side and to the cementum of the root on the other side.

Periodontal maintenance phase continuing patient care provided by the dental team to help the periodontitis patient maintain periodontal health following complete nonsurgical or surgical periodontal therapy. The term periodontal maintenance applies specifically to treated periodontitis patients. It is normally not appropriate to use the term periodontal maintenance for patients treated for other conditions such as those treated for the various types of gingivitis.

Periodontal microsurgery a periodontal surgical procedure performed with the aid of a surgical microscope.

Periodontal osseous resective surgery a type of periodontal surgery employed to correct many of the irregular deformities of the alveolar bone that often result from periodontitis. This is done by using a combination of rotary and hand instrumentation to physically reshape or recontour the osseous defects in order to recreate physiologic bony architecture.

Periodontal plastic surgery periodontal surgery that is directed toward correcting problems with attached gingiva, aberrant frenum, or vestibular depth; includes an array of periodontal surgical procedures that can be used to improve esthetics of the dentition and to enhance prosthetic dentistry as well as to deal with damage resulting from periodontitis. Also see free soft tissue autograft, subepithelial connective tissue graft, coronally positioned flap, frenectomy, and crown lengthening surgery.

Periodontal pocket a pathologic deepening of the gingival sulcus as the result of the apical migration of the junctional epithelium, destruction of the periodontal ligament fibers, and destruction of alveolar bone. Also see apical migration of the junctional epithelium.

Periodontal Screening and Recording (PSR) an efficient, easy-to-use screening system for the detection of periodontal disease. Also see periodontal screening examination.

Periodontal screening examination a periodontal assessment used to determine the periodontal health status of the patient and identify patients needing a more comprehensive periodontal assessment. Also see World Health Organization Probe.

Periodontitis case a patient is a periodontitis case if the interdental clinical attachment loss is detectible at two or more nonadjacent teeth or the facial or lingual clinical attachment loss of 3 mm or more with pocketing greater than 3 mm is detectible at two or more teeth.

Periodontitis a bacterial infection of the periodontium resulting in destruction of all parts of the periodontium including the gingiva, periodontal ligament, bone, and cementum; results in irreversible destruction to the tissues of the periodontium.

Periodontium the functional system of tissues that surrounds the teeth and attaches them to the jawbone. The periodontium is also called the "supporting tissues of the teeth" and "the attachment apparatus."

Periosteum a dense membrane composed of fibrous connective tissue that closely wraps the outer surface of the alveolar bone; it consists of an outer layer of collagenous tissue and an inner layer of fine elastic fibers.

PGE prostaglandins of the E series; play an important role in the bone destruction seen in periodontitis. The major source of PGE in inflamed periodontal tissues is the macrophage, although PMNs and gingival fibroblasts also produce them.

Phagocytosis the process by which leukocytes engulf and digest microorganisms.

Phagolysosome fusion of lysosome granules to the phagosome.

Phagosome a vesicle that surrounds the ingested bacterium; it is found within the cytoplasm of a phagocytic cell.

Pharmacosedation the act of alleviating a patient's distress and anxiety by administering anxiety reduction medications, such as a barbiturate or benzodiazepine.

Phenytoin one of the most commonly used anticonvulsant medications used to control convulsions or seizures in the treatment of epilepsy; associated with drug-influenced gingival enlargement.

PICO process a structure for formulating questions that the PICO entails four critical components **P**atient, **I**ntervention, **C**omparison, and **O**utcome.

Planning one of the four processes of motivational interviewing. To encourage commitment to change and to develop a specific plan of action for change. The OARS skills are used in this process to ensure the plan for change is developed collaboratively rather than falling into the trap of telling the patient what to do.

Polymicrobial a microbial community that is composed of many different species of microorganisms; plaque biofilm is a polymicrobial community.

Polymorphonuclear leukocytes phagocytic cells that actively engulf and destroy microorganisms; PMNs play a vital role in combating the pathogenic bacteria responsible for periodontal disease; also known as neutrophils.

Polypharmacy a condition in which patients take multiple medications simultaneously to treat one or more conditions.

Positive reinforcement a method of behavioral modification that reinforces positive behavior and nurtures such desired behaviors.

Power-driven air floss a new type of power-driven irrigation device that uses a mix of high-speed liquid droplets interspersed with compressed air to mechanically remove interproximal plaque.

Power-driven irrigation device a device that cleans teeth by gently delivering a pulsating stream of water or other solution (like an antimicrobial solution) around and between teeth above the gingival margin and into the gingival sulcus or periodontal pocket, or a stream of compressed air interspersed with microdroplets of water (or an antimicrobial solution) around and between teeth above the gingival margin and into the gingival sulcus or periodontal pocket. A power-driven air floss and a power-driven water irrigation device are two types of power-driven irrigation devices.

Power-driven water irrigation device a device that emits a pulsating stream of water or antimicrobial agent (e.g., Listerine or chlorhexidine) to flush out plaque biofilm and other extraneous debris (like food) from the tooth surface and the gingival sulcus. It is also known as an oral irrigator, water flosser, or power-driven water floss.

Preeclampsia a pathologic multi-system complication of pregnancy. Preeclampsia is characterized by a sudden spike in blood pressure, swelling in the lower extremities, and high levels of protein in the urine (known medically as proteinurea). If not managed appropriately, preeclampsia can lead to eclampsia which is characterized by convulsive seizures, coma, and end-organ dysfunction which may result in substantial maternal and fetal morbidity and mortality.

Pregnancy gingivitis gingival inflammation initiated by plaque and exacerbated by hormonal changes in the second and third trimesters of pregnancy.

Pregnancy-associated pyogenic granuloma a localized, mushroom-shaped gingival mass projecting from the gingival margin or more commonly from a gingival papilla during pregnancy.

Prevalence the number of all cases (both old and new) of a disease that can be identified within a specified population at a given point in time. Also see incidence.

Primary herpetic gingivostomatitis a painful oral condition that results from the initial infection with the herpes simplex virus.

Primary intention healing that occurs when wound margins or edges are closely adapted to each other; an example of primary intention healing would be seen in a small wound in a finger that required stitches. Also see secondary intension and tertiary intention.

Primary trauma from occlusion excessive occlusal forces acting upon a tooth with a sound (healthy) periodontium and no previous history of periodontal breakdown.

Probiotic a dietary substance or preparation that contains the "good" microorganisms that keep the body healthy and in homeostatic balance when consumed or applied to the body. *Lactobacillus*, *Streptococcus*, *Bifidobacterium*, and *Enterococcus* are the beneficial bacteria found in many probiotic products, such as yogurt, fermented foods, and probiotic supplements.

Professional subgingival irrigation the in-office flushing of pockets performed by the dental hygienist or dentist using one of three systems: a blunt-tipped irrigating cannula, an ultrasonic unit equipped with a fluid reservoir, or a specialized air-driven hand piece.

Proinflammatory mediators protein molecules that are synthesized and released by host cells as part of the immunoinflammatory process. Release of pro-inflammatory mediators activate the host immune system which leads to subsequent production of more cytokines which damages host tissues, such as periodontal tissue. These proinflammatory biochemical mediators include chemicals such as matrix metalloproteinases, certain cytokines, prostanoids, and other less well-understood mediators.

Pro-resolving lipid mediators specialized chemical mediators that regulate the resolution of inflammation.

Prostaglandins a series of powerful biochemical mediators, of which prostaglandins D, E, F, G, H, and I are the most important biologically; prostaglandins of the E series (PGE) play an important role in the bone destruction seen in periodontitis.

Prosthesis an appliance used to replace missing teeth. A prosthesis can be removable, fixed (like a crown or bridge on a natural tooth), or implant-supported.

Pseudo-halitosis a form of malodor where the affected individual simply imagines that he or she has malodor but there is no objective evidence to verify it.

Pseudomembrane a yellowish white or grayish tissue slough that covers the necrotic areas of the gingiva in necrotizing periodontal diseases. Also see necrotizing periodontal diseases.

Puberty gingivitis a common form of gingivitis seen in children between 9 to 14 years of age. Most likely associated with hormone changes that intensify the inflammatory response to plaque biofilm.

PubMed a gateway hosted by the National Library of Medicine. Anyone can access MEDLINE for free using PubMed. One feature of the MEDLINE database is the PubMed Clinical Query, which provides specialized searches using an evidence-based filter.

Pulpal abscess an abscess that results from an infection of the tooth pulp. A pulpal abscess can be caused by death of the tooth pulp from trauma to the tooth or from deep dental decay.

Punched-out papillae the destruction of the interdental papillae due to tissue necrosis, which is a characteristic feature observed in necrotizing periodontal diseases.

Pus collections of dead white blood cells that can result when body defense mechanisms are involved in attempting to control an infection. The formation of pus is known as purulence.

Q

Quorum sensing the ability of one microbe to communicate with other microorganisms living within the plaque biofilm by sending signaling molecules.

R

Radiolucent radiographic term that refers to materials and structures that are easily penetrated by x-rays and appear as dark gray to black on the radiograph; examples of radiolucent structures are the tooth pulp, periodontal ligament space, a periapical abscess, marrow spaces in the bone, and bone loss defects.

Radiopaque radiographic term that refers to materials and structures that absorb or resist the passage of x-rays and appear light gray to white on the radiograph; examples of radiopaque structures and materials are metallic silver (amalgam restorations) and newer composite restorations, enamel, dentin, pulp stones, and compact or cortical bone.

Reactive oxygen species (ROS) highly unstable molecules that are contributors to oxidative stress, which can lead to various disorders, such as cardiovascular disorders, cancer, and various neurodegenerative diseases.

Reasonable patient standard a standard that is based on the premise that the health care practitioner must disclose all information that may help a reasonable patient decide to proceed or decline treatment.

Reattachment healing of a periodontal wound by the reunion of the connective tissue and roots where these two tissues have been separated by incision or injury but *not by disease*. Also see new attachment.

Receptor activator of nuclear factor kappa-B ligand (RANKL) a protein catalyst expressed (produced and released) on the membrane of an osteoblast; it is known as an osteoclastic differentiation factor because it plays a role in the differentiation of osteoclastic precursors into mature activated osteoclasts. Along with OPG, RANKL plays a critical role in regulating the bone remodeling process.

Recession of the gingival margin the movement of the gingival margin to a point apical to the cementoenamel junction (CEJ); also known as gingival recession.

Recurrent aphthous ulcers recurrent, painful ulcers of the oral mucosa that occur in school-aged children and adults; also referred to as recurrent aphthous stomatitis.

Recurrent form a return of destructive periodontitis that had been previously arrested by conventional periodontal therapy.

Recurrent herpes simplex labialis a recurrent form of Herpes Simplex Virus Type-I (HSV-1) affecting the lip; commonly referred to as a fever blister or cold sore.

Reduced periodontium a periodontium with a presence of detectable attachment and/or bone loss. Note that a reduced periodontium can be stable (periodontitis in remission) or it can exhibit signs of active disease.

Reevaluation a formal step after the completion of nonsurgical therapy. During the reevaluation appointment, the members of the dental team perform another periodontal assessment to gather information about the patient's periodontal status and determine how well the periodontium has responded to initial therapy.

Reflective listening the process in which the health care provider listens to the patient's remarks, then paraphrases what the clinician heard the patient say. This allows the clinician to check with the patient that he or she is "getting the patient's message right," ensuring that the clinician is developing a good picture of the patient's perspective.

Refractory form a form of periodontitis seen in a patient who has been previously treated and monitored over time, but exhibits continuing attachment loss despite the following conditions (a) the patient receives appropriate and continuous professional periodontal therapy, (b) the patient practices satisfactory self-care, and (c) the patient follows the recommended program of periodontal maintenance visits.

Refractory periodontitis periodontitis that is resistant to treatment from the outset of therapy even with what appears to be appropriate periodontal therapy.

Regeneration the biologic process by which the architecture and function of lost tissue is *completely* restored. Also see repair.

Relative contraindication conditions that may make periodontal surgery inadvisable for some patients when the conditions or situations are severe or extreme; these same conditions may not be contraindications when the conditions are mild.

Repair the healing of a wound by formation of tissues that do not precisely restore the original architecture or original function of the body part. Also see regeneration.

Resective surgery any type of periodontal surgery which involve excising and removing periodontal tissues. See ostectomy, osteoplasty, gingivectomy, and gingivoplasty.

Resolution process a host-protective response that follows the initiation phase of inflammation. The resolution process blunts the production of inflammatory mediators, clears dead cells, regulates leukocyte trafficking, and stimulates tissue repair. The complete and timely resolution of inflammation is critical to restoring tissue homeostasis.

Restorative therapy phase treatment include placement of dental restorations and/or replacement of missing teeth by fixed or removable prostheses. Also called as phase III therapy.

Reversible tissue damage the tissue damage that the body can repair itself with good self-care, as in gingivitis.

Risk assessment the process of identifying risk factors that increase an individual's probability of disease.

Risk factor factors that modify or amplify the likelihood of developing periodontal disease; major established risk factors for periodontitis are specific bacterial pathogens, cigarette smoking, and diabetes mellitus.

Root caries tooth decay that occurs on the root surfaces.

Root concavity a trench-like depression on the root surface that can harbor plaque biofilm.

S

Secondary intention healing that takes place when the margins or edges of the wound are not closely adapted (i.e., the two wound edges are not in close contact with each other). When healing by secondary intention takes place, granulation tissue must form to close the space between the wound margins prior to growth of epithelial cells over the surface of the wound. Also see primary intention and tertiary intention.

Secondary trauma from occlusion defined as normal occlusal forces acting upon a tooth with an unhealthy periodontium previously weakened by periodontitis.

Semilunar coronally repositioned flap a periodontal plastic surgical procedure employed to cover gingival recession by coronally advancing a flap. It is indicated for recession sites that are not far advanced and where the keratinized tissues have an adequate thickness.

Sequestrum a fragment of necrotic (dead) alveolar bone. Necrotizing periodontitis can be accompanied by the formation of bone sequestra.

SHARE Approach a five-step process—Seek, Help, Assess, Reach, Evaluate—for shared decision-making that includes exploring the benefits and risks of each care/treatment option through meaningful discussion about what matters most to the patient.

Shared decision-making a collaborative process that recognizes a patient's right to make decisions about his/her care once fully informed about the treatment options.

Sharp dissection elevation of a flap by incising the underlying connective tissue in such a manner as to separate the epithelial surface plus a small portion of the connective tissue from the periosteum; use of this technique would leave the periosteal tissues covering the bone. Also see partial-thickness flap.

Sharpey's fibers the ends of the periodontal ligament fibers that are embedded in the cementum and alveolar bone.

Sialometry an objective method of measuring salivary flow.

Signs of periodontal disease features of a disease that can be observed or are measurable by the dental clinician. Include gingival erythema (redness), gingival edema (swelling), bleeding on gentle probing, loss of attachment, tooth mobility, or loss of alveolar bone support.

Silent disease lack of, or "silence," of early symptoms that are not obvious to the patient until the disease has progressed to cause considerable damage to the periodontium.

Site-specific periodontal inflammation that only affects a single site of a tooth or localized to a specific region of the mouth. A patient with site-specific periodontitis does not exhibit attachment loss and loss bone throughout the dentition.

Sjögren syndrome an inflammatory autoimmune disease that results in a general dryness caused by damage to the salivary and other similar glands, such as tear glands.

Smoking cessation counselling includes information on smoking cessation and prevention of tobacco use, as well as referrals to other health professionals for tobacco cessation programs.

Specialized proresolution mediators (SPMs) naturally occurring compounds that are derivatives of omega-3 fatty acids; they promote the resolution phase of inflammation by limiting further neutrophilic infiltration to the target site, thereby allowing the body to return to homeostasis.

Specific plaque hypothesis an outdated hypothesis which postulated that specific pathogenic bacteria and their products in the biofilm lead to periodontal disease.

Stage one of two diagnostic parameters (along with periodontitis grading) that is used to define an individual patient's periodontitis. It provides a way of classifying the extent, severity, and complexity of managing the periodontitis case.

Standard irrigation tips this type of tip is recommended for generalized, full-mouth irrigation. It is the most evaluated tip and has been shown to improve oral health better than dental floss.

Standard of care the degree of prudence, caution, and attention that a reasonable health care practitioner is expected to exercise when caring for a patient.

Statins specialized medications widely prescribed to prevent cardiac and cerebrovascular events and to manage hypercholesterolemia. The primary mechanism of action is mediated by its ability to inhibit the key enzyme in endogenous cholesterol production, known as HmG-CoA reductase inhibitor.

Stem cells a class of undifferentiated (precursor) cells that have the capability to differentiate into specialized cell types.

Stippling the dimpled appearance, similar to the skin of an orange peel, that may be visible on the surface of the attached gingiva.

Stratified squamous epithelium a type of epithelium that is comprised of flat cells arranged in several layers that makes up the skin and the mucosa of the oral cavity.

Subantimicrobial dose doxycycline (SDD) A 20-mg dose of orally-administered doxycycline which is used as an adjunct to scaling and root planing to blunt the host inflammatory response. As of the publication of this textbook, SDD is the only FDA-approved host modulating agent approved for periodontal therapy.

Subepithelial connective tissue graft (SCTG) a periodontal plastic surgical procedure that can also be used to augment the width of attached gingiva and to cover areas of gingival recession. In addition to gingival augmentation, the subepithelial connective tissue graft is used to alter the contour of alveolar ridges to improve the esthetics of some types of dental prostheses.

Subgingival irrigation tips these tips usually have a soft rubber-tipped end. Subgingival irrigation tips are not cleansing tips. They are to be used to deliver a medicament in areas such as deep pockets, furcation areas, dental implants, or areas that are difficult to access with a standard tip.

Subjective fear a fear that does not spring from a prior experience. It may result from images of terrifying dentists on television, playground horror stories from classmates, or by sensing mom's anxiety before her own dental appointment.

Substantivity the property of a chemical to adhere to a target and be released slowly over time.

Sulcular epithelium the epithelial lining of the gingival sulcus; it is continuous with the oral epithelium and extends from the crest of the gingival margin to the coronal border of the junctional epithelium. Also see gingival epithelium.

Summaries one of the four core communication skills of OARS. To link and reinforce what has been discussed and provide an opportunity for the clinician to demonstrate empathy. Summaries can be used to move the conversation in a new direction; are also used to tie different elements of the conversation together.

Suppuration The process whereby tissues and inflammatory exudates are liquefied by the action of the offending microorganisms. See exudate.

Suprabony defect a type of bone defect in which the base of the periodontal pocket is located coronal to the alveolar crest.

Suprabony pocket a periodontal pocket in which there is horizontal bone loss, and the junctional epithelium, forming the base of the pocket, is located *coronal* to the crest of the alveolar bone. Also see periodontal pocket and infrabony pocket.

Supracrestal tissue attachment the zone of periodontal tissue composed of the junctional epithelium and the connective tissue attachment located coronal to the alveolar crest. Previously referred to as biologic width.

Supragingival fiber bundles a network of rope-like collagen fiber bundles in the gingival connective tissue located coronal to (above) the crest of the alveolar bone.

Surgical therapy phase treatment includes any necessary periodontal surgery to control or eliminate periodontal disease that cannot be managed by nonsurgical therapy alone. The surgical phase also includes the placement of dental implants to replace missing teeth and reestablish form and function of the oral dentition.

Sustain talk statements that are against change (or in favor of continuing with their current behavior).

Symbiosis a beneficial interaction between microorganisms living in close association within the biofilm or between the microorganisms and the host organism, typically to the advantage of each other. The direct opposite is dysbiosis.

Symptoms of periodontal disease features of a disease that are noticeable only to the patient.

Systematic desensitization a common iatrosedation technique used to calm an anxious child. This approach relies on initially presenting a situation that evokes a little fear, and then progressively presenting stimuli that are more fear-provoking. Systematic desensitization may be an effective iatrosedation technique for anxious adult patients.

Systematic review a concise summary of individual research studies on a treatment or device to determine the overall validity and clinical applicability of that treatment or device.

Systemic antibiotics antibiotics that are taken orally or injected intramuscularly or intravenously.

Systemic delivery in dentistry, usually refers to administering chemical agents in the form of a tablet or capsule. When a tablet is taken by the patient, the chemical agent contained is released as the tablet dissolves, and the agent subsequently enters the blood stream—thus the chemical agent is circulated "systemically" throughout the body.

Systemic risk factors conditions or diseases that increase an individual's susceptibility to periodontal infection by modifying or amplifying the host response to the bacterial infection; proven systemic risk factors include diabetes mellitus, osteoporosis, hormone alteration, medications, tobacco use, and genetic influences.

T

Tell–Show–Do an iatrosedation technique that works to alleviate the anxiety of apprehensive children receiving dental care. Tell-show-do involves: "Tell": the health care provider explains what they are planning to do to the child patient; "Show": the health care provider shows the instruments to the child patient and demonstrates it on a finger; and "Do": the health care provider performs the procedure. Tell-show-do is an effective iatrosedation technique that can be used in anxious adult patients as well or in adult patients who have intellectual disabilities.

Tertiary intention healing of a wound that is temporarily left open with the specific intent of surgically closing that wound at a later date; healing by tertiary intention is not normally a type of healing that applies to healing of periodontal surgical procedures. Also see primary intention and secondary intention.

Therapeutic mouth rinses a mouth rinse that has some actual benefit (provides some therapeutic action) to the patient in addition to the simple goal of making the breath smell a bit better.

Tissue inhibitors of matrix metalloproteinases (TIMP) an inhibitory enzyme that is released by the host to regulate the activity of MMPs. TIMPs and MMPs work in concert to maintain tissue function and form, extracellular matrix integrity, and wound healing. Also see matrix metalloproteinases.

Tissue a group of interconnected cells that perform a similar function within an organism. For example, muscle cells group together to form muscle tissue that functions to move parts of the body. Also see connective tissue and epithelial tissue.

Tissue-associated plaque biofilms bacteria that adhere to the epithelium.

T-lymphocytes small leukocytes whose main function is to intensify the response of other immune cells—such as B-lymphocytes and macrophages—to the bacterial invasion.

Tongue coating an accumulation of bacteria, food debris, and desquamated epithelial cells on the dorsal surface of the tongue and is a source of oral malodor.

Tongue thrusting the application of forceful pressure against the anterior teeth with the tongue an example of a parafunctional habit.

Tooth-associated plaque biofilms bacteria that are attached to the tooth surface.

Topical delivery in dentistry, usually refers to placing a chemical agent into the mouth or even into a periodontal pocket where the chemical agent then comes into contact with plaque forming either on the teeth or in the periodontal pocket.

Tort an act that injures someone in some way, and for which the injured individual may sue the wrongdoer for damages.

Transendothelial migration the process of leukocytes squeezing their way through the thin endothelial wall of a blood vessel to enter the surrounding connective tissue.

Trauma from occlusion excessive occlusal forces that cause damage to the periodontium. Also see primary trauma from occlusion and secondary trauma from occlusion.

Traumatic tooth brushing the aggressive or overzealous use of a toothbrush that injures the gingiva or loss of tooth structure. The trauma may be caused by improper toothbrushing habits that traumatize the gingiva or the use of a hard or medium-bristle toothbrush.

Triangulation the appearance on a dental radiographic of widening of the periodontal ligament space; triangulation is caused by the resorption of bone along either the mesial or distal aspect of the interdental (interseptal) crestal bone.

Trigeminal nerve Cranial nerve V responsible for the sensory innervation of most of the skin, of the front part of the face and head, the teeth, oral cavity, maxillary sinus, and nasal cavity.

Trismus a painful condition that restricts normal mandibular movement and function as a result of a low-grade infection; limited mouth opening.

U

Ultrasonic periodontal probes incorporation of ultrasonic technology within a small intraoral handpiece as a noninvasive means to assess periodontal health; also known as ultrasonographic periodontal probes or ultrasound periodontal probes. An ultrasonic periodontal probe promises reduced intra- and inter-examiner variability in probing measurements, improved ease of use, and enhanced patient comfort because the tip is not inserted in the sulcus.

Unattached bacteria the free-floating bacteria in the periodontal pocket that are not part of the biofilm.

Undifferentiated (cells) stem cells that are unspecialized.

Upcoding an illegal practice where the health care practitioner reports a higher level of service or procedure than was actually performed.

V

Ventilator-associated pneumonia a type of hospital-acquired pneumonia that develops after intubation for mechanical ventilation.

Vertical bone loss a less common pattern of bone loss resulting in an uneven reduction in the height of the alveolar bone. In vertical bone loss, the resorption progresses *more rapidly* in the bone next to the root surface leaving a trench-like area of missing bone alongside the root. Also known as vertical defect or angular defect. Also see alveolar bone loss and horizontal bone loss.

Vertical incisions an incision that runs perpendicular to the gingival margin in an apico-occlusal direction, primarily used to advance the flap without overstretching or damaging the soft tissues during flap elevation.

Vertical tooth mobility the ability to depress the tooth in its socket. This is assessed by placing the blunt end of an instrument handle on the occlusal or incisal surface of the tooth and exerting a downward (apically directed) pressure to determine if the tooth is depressible.

Vertical transmission the transfer of bacteria between members of different generations. For example, pathogenic bacteria can be transmitted from a mother to a child by the sharing of utensils.

Vestibular depth the distance between the coronal border of the attached gingiva to the greatest concavity of the mucobuccal fold; an adequate vestibular depth is desired to enable placement of toothbrush and interdental cleaning aids, thus facilitating proper oral hygiene.

Virulence factor the mechanisms that enable biofilm bacteria to colonize, invade, and damage the tissues of the periodontium.

Volatile sulfur compounds (VSCs) a family of gases that is responsible for oral malodor.

W

Well-controlled (diabetes) a condition of a diabetic person whose A1C (used to monitor how well diabetes treatment is working overtime) value is between 6.5 to 7% or lower. This also means that an individual's average blood sugars are between 140 to 154 mg/dL. Targets are typically geared toward fasting blood sugars less than 130 mg/dL and two hours after meals less than 180 mg/dL.

World Health Organization (WHO) probe used when performing the periodontal screening examination. This probe is easily identifiable by a 0.5-mm diameter ball at the tip, wide black band located 3.5 to 5.5 mm from the ball tip, and markings further up the probe which represent 8.5 mm and 11.5 mm from the ball tip. Another name for the WHO probe is the CPITN (Community Periodontal Index of Treatment Needs) probe. Also see periodontal screening examination.

X

Xenografts bone replacement grafts taken from another species, such as bovine bone replacement graft material; these materials must be modified to eliminate the potential for rejection. Also see autografts, allografts, and alloplasts.

Xerostomia the subjective sensation of dry mouth, as expressed by the patient.

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